Sjøfartsdirektoratet
Norwegian Maritime Authority

GUIDANCE TO REGULATIONS ON THE MEDICAL EXAMINATION OF EMPLOYEES ON NORWEGIAN SHIPS AND MOBILE OFFSHORE UNITS

Ver 2.3. – 8th June 2018

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1. INTRODUCTION

1.1 ADMINISTRATIVE AND LEGAL

The **Regulations of 5th June 2014 No 805 on the medical examination of employees on Norwegian ships and mobile offshore units** (the Health Regulations) are based on various sources. They have their legal basis in section 17 of the Ship Safety and Security Act, whereas the main part of the medical requirements are taken from the *ILO/IMO Guidelines on the medical examinations of seafarers* (henceforth called the "Guidelines"). The Guidelines are in turn a result of a cooperation between the International Labour Organization (ILO)and the International Maritime Organization (IMO). The ILO are responsible for the Maritime Labour Convention (MLC), whereas the IMO are responsible for the International Convention on Standards of Training, Certification and Watchkeeping for Seafarers (STCW). The purpose of the Guidelines was to develop an instrument reflecting the contents of both Conventions, and which could be helpful for the flag State when implementing these two Conventions.

Amendments have been made to these new Regulations, by the Norwegian Maritime Authority (NMA) and the industry, based on experiences related to the previous Regulations. We hope that the new Regulations will be an improvement compared to the old, and that they will be easier to use for persons who work on board Norwegian vessels, companies and seafarer's doctors.

1.2 MEDICAL GUIDANCE

The guide to the medical conditions is meant to be dynamic. That is, it will be gradually developed and revised as cases are processed in the appellate body. The selection of diagnoses currently described is built on case processing in the appellate body in the period September 2009 to January 2015.

Feedback from users regarding the need for corrections or requests for additions or elaborations of special topics will be important in the continuous revision process.

An updated guidance will always be available online. Downloads are only valid until a new version is published, and are made at the user's own responsibility.

The responsible party for the medical section is the Norwegian Centre for Maritime and Diving Medicine at Haukeland University Hospital, Bergen, Norway.

1.3 WHY WE HAVE HEALTH REQUIREMENTS

When the Ship Safety and Security Act was adopted, a provision regarding health requirements was included in the Act. Section 17 in the Act stipulates that any person who is working on board must be physically and mentally fit for the role and not pose a danger to other persons on board. Each person who work on board (herinafter referred to as person) shall present a medical certificate that confirms that these requirements are met.

«Physically and mentally fit» is a very wide term, and in order to enable persons to document this, there is a need for more concrete health requirements for examination purposes. In addition to being bound by the Ship Safety and Security Act, Norway and the Norwegian Maritime Authority have committed to fulfilling the requirements of the Maritime Labour Convention, 2006 (MLC, 2006) and the International Convention on Standards of Training, Verification and Watchkeeping for Seafarers (STCW Convention).

In 2013 The ILO and IMO published the Guidelines on the medical examinations of seafarers, which are guidelines for the member States of the ILO and IMO when developing national legislation in this area.

In order to implement this international set of rules, a more detailed regulation than we currently have in section 17 of the Ship Safety and Security Act is required.

The Norwegian Maritime Authority therefore have developed the Regulations on the medical examination of persons on Norwegian ships and mobile offshore units.

1.4 STCW CONVENTION

The **STCW** Convention (The International Convention on Standards of Training, Certification and Watchkeeping for Seafarers) prescribes minimum standards relating to training, certification and watchkeeping for seafarers which countries are obliged to meet or exceed. The STCW Convention was originally drafted in 1978, entered into force in 1984, was amended in 1995 and then again in 2010 (The Manila Amendments). The Manila Amendments were adopted on 1 January 2012 and had to be implemented by 2017.

The STCW Convention from 1978 was the first international convention prescribing minimum requirements for training, certification and watchkeeping. Before the STCW Convention entered into force, this varied a lot between different maritime administrations, despite the fact that shipping is an international industry.

The most significant changes in The Manila Amendments are

- New rest hours for seafarers
- New grades of certificates of competence for Able seaman in both deck and engine
- New and updated training, refreshing requirements
- Mandatory security training
- Additional medical standards
- Specific Alcohol limits in blood or breath

Information regarding the STCW Convention at IMO Website:

http://www.imo.org/OurWork/HumanElement/TrainingCertification/Pages/STCW-Convention.aspx

1.5 THE PURPOSE OF THIS GUIDANCE

The purpose of this guidance is to make the Regulations more accessible for the users and to standardise the way in which persons and seafarer's doctors use and interpret the Regulations.

Even with a guide, it is important to remember that the Regulations are the official governing instrument to use in case processing. Seafarer's doctors shall have a solid knowledge of both the Regulations, section 17 of the Ship Safety and Security Act, the Public Administration Act and the guidance.

The terms absolute and relative contra indications have been removed from the new Regulations. Stricter demands are made on the medical judgement of the seafarer's doctor. The guidance is meant to work as a support for the exercise of discretion in relation to, for example, epidemiological data.

1.6 THE HEALTH REGULATIONS APPLY TO

The Regulations apply to persons working on board Norwegian ships, (cf. section 2 of the Regulations) with the exception of those who only:

a) work on board while the vessel is in port;

b) carry out inspections on board.

Furthermore, the Regulations do not apply to persons who have turned 18 years old and who are working on board the following vessels, when the vessel is at sea for continuous periods of no more than three days:

a) fishing vessels of up to 15 metres in overall length or of less than 100 gross tonnage when the vessels is less than 24 metres in length (L);

b) fishing vessels of less than 24 metres in length (L) certified for Bank fishing I or lesser trade areas;

c) cargo ships of less than 15 metres in length (L) engaged on domestic voyages.

Additional the Regulations also apply to persons working on board a mobile offshore unit, but in this case an exception has been made for persons not serving in positions for which a maritime certificate is required (called "Certificate of Competency" in the Regulations of 22 December 2011 No. 1523 concerning qualifications and certificates for seafarers). These persons may choose whether they want to use a seafarer's doctor and be issued a medical certificate in accordance with the maritime legislation, or whether they want to see a petroleum doctor and be issued a medical certificate in accordance with Petroleum Safety Authority Norway's Regulations. In other words, a person working with, for example, seismology or in the canteen may choose which type of medical certificate to obtain when working on a Norwegian mobile offshore unit. Persons in positions that require a maritime certificate, such as the captain, an engineer and a mate must still have a medical certificate pursuant to the maritime Health Regulations discussed in this guide.

1.6.1 THE HEALTH REGULATIONS CONCERNS SEAFARER'S DOCTORS

Definision *"Seafarer's doctor":* A medical practitioner approved for the purpose of conducting medical examinations and making decisions in accordance with the provisions of the Health Regulations.

1.7 PUBLIC BODIES INVOLVED IN THE REGULATIONS

The Ministry of Trade, Industry and Fisheries is the Ministry responsible for the Ship Safety and Security Act and the derived Regulations.

The Norwegian Maritime Authority is the government agency which has been given the authority to develope legislation and to carry out the supervision of Norwegian ships, persons working on board and seafarer's doctors.

See www.sdir.no for more information.

The Norwegian Centre of Maritime and Diving Medicine (NCMDM) at Haukeland University Hospital is Norway's national centre of excellence in maritime and diving medicine. It is authorised by the Parliament, and acts as adviser to the Norwegian Maritime Authority. See www.ncmm.no for more information.

1.8 RESPONSIBLE PARTY AT THE NMA

The Section for Seafarers is the responsible party in the Norwegian Maritime Authority.

1.9 DEVELOPMENT OF THE GUIDANCE

Responsible legal adviser has been the main editor in the development of the Regulations. Medical advisors at the NCMDM have developed the medical part of the Guidance.

2 INCLUDED IN THE HEALTH REGULATIONS

2.1 THIS IS REQUIRED IN ORDER FOR THE PERSON TO GET A MEDICAL CERTIFICATE

The person must be examined by a seafarer's doctor in accordance with the requirements of the Health Regulations. Lists of approved seafarer's doctors can be found at the NMA web site, and the person may freely choose the seafarer's doctor he/she prefers.

The seafarer's doctor shall, after the examination, assess the person's health against the requirements of section 1 of the Regulations, as elaborated in the Appendix to the Regulations.

If the doctor finds that the person fulfils the requirements laid down by the Regulations, the doctor shall issue a medical certificate.

2.2 THIS IS HOW A MEDICAL CERTIFICATE SHALL BE ISSUED

When the medical examination is over, the seafarer's doctor register the applicable Medical certificate/Declaration of unfitness on his computer in the NMA's electronical submission system (herinafter referred to as the database/system). The seafarer's doctor shall search for the person in the NMA's database, via www.altinn.no. If the person is not found in the database the seafarer's doctor shall register a new person via the system. When the registration is done the Medical certificate/Declaration of unfitness is to be printed, stamped, signed and given to the person. The person shall sign it too. The seafarer's doctor shall keep a copy for the file.

All profiles include the person's personal data as well as information of Medical certificates or Declarations of unfitness issued to the person.

See "Guidance for electronic submission on medical certificate and declaration of unfitness" for further information.

Paper forms of Medical certificate (KS 0499-1 B/E) and Declaration of unfitness (KS 0415 B/E) are to be used as a back up when the electronic system is unavailable. The Norwegian Royal Embassy/Consulate General in the relevant country supplies the approved seafarer's doctor with forms in paper.

2.3 A MEDICAL CERTIFICATE SHALL BE ISSUED BY

Only approved seafarer's doctors may issue medical certificates pursuant to the Health Regulations.

The seafarer's doctor's approval certificate shall be visibly placed in the office of the seafarer's doctor. This certificate shall state when the approval expires.

A medical certificate will not be valid if the seafarer's doctor's approval is expired. It is therefore important that the person makes sure that the seafarer's doctor's approval is not expired when obtaining a medical certificate.

2.4 DUTIES IMPOSED ON THE COMPANY, MASTER AND THE PERSON AS REGARDS THE MEDICAL CERTIFICATE

The company has a duty to ensure that all persons working on their (Norwegian) ships have a valid medical certificate, cf. section 6 of the Ship Safety and Security Act.

The master shall participate in ensuring that the person working on board have a valid medical certificate, cf. section 19 second paragraph (d) of the Ship Safety and Security Act. The medical certificate shall be kept by the master on board in its original form, cf. section 20 of the Regulations of 22 December 2011 No. 1523 concerning qualifications and certificates for seafarers.

The person have a duty to present a valid medical certificate in original form, cf. section 20 first paragraph (d) of the Ship Safety and Security Act, ref. section 20 of the Regulations of 22 December 2011 No. 1523 concerning qualifications and certificates for seafarers.

Any person that have reason to believe that the requirements for a medical certificate are no longer satisfied shall inform the master or the company and consult a seafarer's doctor, cf. section 6 of the Health Regulations. Furthermore, the person has to submit to medical examination if the company or master considers that the health requirements may no longer be met, cf. section 17 of the Ship Safety and Security Act, ref. section 6 of the Regulations.

2.5 A PERSON IS REQUIRED TO HAVE A VALID MEDICAL CERTIFICATE

Any person working on board a Norwegian ship shall have a valid medical certificate. See https://www.sdir.no/en/shipping/seafarers/helse/sjofolks-helse/medical-certificatedeclaration-of-unfitness/ for detailed information of accepted medical certificates on Norwegian ships.

Persons who work on board a mobile offshore unit in a capacity for which a certificate of competency is not required pursuant to the Regulations of 22 December 2011 No. 1523 concerning qualifications and certificates for seafarers, may as an alternative hold a medical certificate issued in accordance with the Petroleum Safety Authority Norway's Regulations.

Persons holding medical certificates that expired up to a month earlier, may commence service on board when a new medical certificate cannot reasonably be obtained without delaying the vessel.

This is a specific exemption, and it will only be applicable in cases where conditions have arisen that company, master and person could not have foreseen. Poor planning and oversights will not be considered as grounds for using this provision. Illness or injury to a crew member scheduled to work and whom it was not possible to replace by other means than by a person without a valid medical certificate is an example of a case where this provision may be applied.

2.6 APPROVAL AS A SEAFARER'S DOCTOR

In order to become an approved seafarer's doctor you must meet the requirements in section 7 in the Health Regulations. Medical practitioners with practice in Norway shall be approved as seafarer's doctor by the Norwegian Maritime Authority.

Medical practitioners with practice outside of Norway shall be approved as seafarer's doctor by a foreign service mission on behalf of the Norwegian Maritime Authority.

See the NMA website for detailed information and application form.

It is not possible to issue valid medical certificates pursuant to the Health Regulations without first being approved by the NMA or a Norwegian foreign service mission.

Medical practitioners already approved must apply for renewal of their approval no later than one month before the expiry of the current approval. This is important in order to avoid the situation where the doctor is without approval for a period while the application for renewal is being processed.

In exceptional cases, the Norwegian Maritime Authority may grant exemptions from the requirements for approved seafarer's doctors.

2.7 A COURSE IN MARITIME MEDICINE HAS TO BE COMPLETED

Doctors already approved as a seafarer's doctor will be included in the transitional arrangement of section 19 of the Health Regulations, and will therefore have 5 years from the date of entry into force of the Regulations – 1st July 2014 – in order to complete a course in maritime medicine.

Doctors not approved as a seafarer's doctor when the Regulations enter into force will have to complete a course in maritime medicine approved by the NMA before being approved as a seafarer's doctor.

Seafarer's doctors must complete a refresher course approved by the NMA during each approval period in order to have their approval renewed.

2.7.1 COURSES IN MARITIME MEDICINE:

Mandatory Basic Course – transitional arrangements for doctors already in service as a seafarer's doctor

- Seafarer's doctors who have not attended the NCMM Basic Course in Maritime Medicine, shall attend a Basic Course for Seafarer's doctors or a Basic Course in Maritime Medicine before 1st July 2019.
- Seafarer's doctors who have attended the NCMM Basic Course in Maritime Medicine No. 1-19 (up to nov 2008), need a Basic Course for Seafarer's doctors or a Basic Course in Maritime Medicine before 1st July 2019.

- Seafarer's doctors who have attended the NCMM Basic Course in Maritime Medicine No 20 (May 2009) – 29 (May 2013), need two refresher courses within 1st July 2019.
- Seafarer's doctors who have attended the NCMM Basic Course in Maritime Medicine No 30 (November 2013) or later, need 1 refresher course before 1st July 2019.

Those who are not approved earlier, will have to attend a Basic Course for seafarer's doctors or a Basic Course in Maritime Medicine before approval.

Refresher training is mandatory for all seafarer's doctors.

2.8 QUALITY SYSTEM FOR A SEAFARER'S DOCTOR

The Health Regulations entered into force on 1 July 2014, and doctors who were approved as seafarer's doctors at this time are covered by the transitional arrangement of section 19 of the Regulations, and must therefore implement a quality system by 1 July 2019. Doctors not approved as seafarer's doctors when the Regulations entered into force will have to implement a quality system before they can be approved as seafarer's doctors.

Pursuant to the Regulations seafarer's doctors are required to have a quality system ensuring that the work is carried out in accordance with the requirements of the Health Regulations. The quality system shall be in accordance with a internationally recognised standard, cf. section 7, first paragraph (h) of the Regulations. The seafarer's doctor is not required to have the quality system certified. It is up to the individual doctor to find the standard best suited for his or her practice, and to decide how to obtain and implement their quality system. The seafarer's doctor shall submit a self-declaration form to the NMA or the foreign service mission when he/she applies for approval or re-approval as a seafarer's doctor. If the quality system is changed/replaced for any reasons, a new self-declaration form shall unsolicited be submitted to the NMA.

The main principle of a quality system is that the company shall develop, deliver and improve products and services in accordance with both specified requirements and expectations. The quality system shall clarify how the company is organised and managed in order to meet both external and internal quality requirements and expectations.

When the NMA carries out supervision, the seafarer's doctor must demonstrate that the quality system is functioning. The seafarer's doctor must be able to account for their administrative procedure, including medical decisions.

2.8.1 MINIMUM REQUIREMENTS FOR A QUALITY SYSTEM

The following minimum requirements shall be included in a seafarer's doctor's quality system:

2.8.1.1 QUALITY MANUAL

The seafarer's doctor must develop and maintain a quality manual for quality management and the documented routines which have been established. The following paragraphs concretise the contents of a quality manual.

2.8.1.2 QUALITY POLICY

The seafarer's doctor must enter the quality goals he or she has for the work as a seafarer's doctor in the quality manual. An example of a quality goal is: "The practice as seafarer's doctor shall be carried out in accordance with the Health Regulations, the Public Administration Act and sound medical judgement."

2.8.1.3 NORMATIVE DOCUMENTATION

The seafarer's doctor must identify the documents, both internal and external, which are normative for how he or she shall operate as a seafarer's doctor. The quality manual is a typical internal normative document, whereas laws and regulations are typical external normative documents.

The quality system shall show the requirements applicable to seafarer's doctors and their products, i.e. medical certificates and declarations of unfitness. The Health Regulations, the guide to the Regulations and the Public Administration Act are examples of external normative documents which concretise such requirements. For doctors in Norway, another such normative document will be the Health Personnel Act.

2.8.2 A DOCUMENTED ROUTINE FOR THE CONTROL OF NORMATIVE DOCUMENTS SHALL BE ESTABLISHED, IN ORDER TO

- approve the adequacy of documents before they are published
- review and, if necessary, update and reapprove documents
- ensure that amendments of current documents are made evident
- ensure that the correct version of relevant documents are available where they are being used
- ensure that documents are readable and easy to identify
- ensure that documents with external origin, which the seafarer's doctor has decided are necessary for the planning and operation of the quality management system, have been identified, and that the documents are being distributed to the relevant recipients (seafarer's doctors and any other persoms forming part of the administrative procedure)
- prevent unintentional use of out-dated documents, and apply the use of appropriate identification (e.g. mark the document with "expired version") for documents which are

to be kept for a specific purpose, so that they are not confused with current applicable documents

2.8.2.1 THE MAIN PROCESSES OF THE PRACTICE

The main processes of a seafarer's doctor's practice are listed below. The following supplementary points are important to take into consideration. (Note that the list is not exhaustive, there may also be other processes carried out by the seafarer's doctor, which must be identified and described.)

MEDICAL EXAMINATION OF THE PERSON

- Is the basis for the decision sufficiently documented in the medical record?
- Is the medical documentation sufficient?
- Have the right supplementary examinations been carried out?
- Has a statement from specialist been requested, if necessary?
- Has purchasing competence been documented in the referral letter to the specialist by requesting assessment of relevant circumstances?
- Is there sufficient epidemiological knowledge of the person's condition, and is there a correct understanding of the likelihood for complications and other medical events associated with the condition?
- Has the likelihood for an event occurring during the certificate period been individualised, based on the person's type and degree of a medical condition, compared to the group of individuals with the same condition?
- Has a "best practice" assessment been made of the likelihood for a medical event occurring?
- Has the seafarer's doctor in the medical record demonstrated that he or she is familiar with the person's duties, tasks and working situation?
- Have the consequences of a medical event occurring in the person's working situation been assessed, and has a risk assessment been carried out thereof?
- Has a risk assessment been carried out with regard to the provisions of the Regulations (likelihood x consequence)?
- Have compensating measured been assessed?

ISSUE OF MEDICAL CERTIFICATE

- Has the person been informed in writing of the grounds for the decision?
- Has the person been informed in accordance with the Public Administration Act of the possibility to appeal the seafarer's doctor's decision or to apply for an exemption?

ISSUE OF LIMITED MEDICAL CERTIFICATE

- Has the person been informed in writing of the grounds for the decision?
- Has the person been informed in accordance with the Public Administration Act of the possibility to appeal the seafarer's doctor's decision or to apply for an exemption?

ISSUE OF PERMANENT, TEMPORARY OR PROVISIONAL DECLARATION OF UNFITNESS

- Has the person been informed in writing of the grounds for the decision?
- Has the person been informed in accordance with the Public Administration Act of the possibility to appeal the seafarer's doctor's decision or to apply for an exemption? (Note: it is not possible to apply for exemption in the event of a provisional declaration of unfitness.)

A description of the main processes and their interrelation shall be included in the quality manual.

2.8.2.2 SYSTEM FOR NON-CONFORMANCE REPORTING AND IMPROVEMENT SUGGESTIONS

The seafarer's doctor must have a system for the treatment of non-conformance. The system shall record non-conformities and corrective actions, and shall contain an overview of preventive measures.

A non-conformity in the practice as seafarer's doctor will typically be feedback given to the seafarer's doctor demonstrating that the decision he or she made had a material (medical) or procedural (non-compliance with the Health Regulations or the Public Administration Act) error. Failure to check the person's ID will for instance be a procedural error, which will result in the issued medical certificate being invalid. If the seafarer's doctor is made aware of this error, he/she will first of all have to implement corrective measures (invite the person to a new examination and issue a valid medical certificate). The seafarer's doctor must then consider which preventive measures to implement in order to avoid such incidences in the future.

Procedures shall be established for non-conformance treatment, corrective measures and preventive measures. If appropriate, these procedures may be gathered in one common procedure.

2.8.2.3 REQUIREMENTS FOR REGISTRATIONS

As part of the quality system, the implementation of the below points must be recorded with traceability. The registrations may e.g. be made in a log or in a report of the activity (in a Word document, on a paper, etc.; there are no formal requirements other than the registration being in writing).

COMPETENCE AND TRAINING

- Competence and training
  The Health Regulations require the seafarer's doctor to complete a course in maritime medicine. The completion of the course and other forms for competence enhancement as seafarer's doctor or for other persons shall be recorded.

- Identification and traceability
  The decisions made by the seafarer's doctor shall be traceable and identifiable. This shall be ensured through the new electronic administrative system for medical certificates and declarations of unfitness. For other documents, such as medical examination forms and medical records, the seafarer's doctor must describe how these are stored and made traceable, e.g. by means of a filing system. This will normally be regulated by national health legislation.

- Calibration and verification of measuring equipment
  It must be stated in the quality manual how the seafarer's doctor ensures the proper functioning of the equipment used.

- Corrective/preventive measures
  When a non-conformity has been registered, the seafarer's doctor must make a memo of how the non-conformity was rectified, and how this non-conformity is being followed up in order to prevent the same non-conformity from reoccurring.

- Internal audit / management review
  The seafarer's doctor or other person in the management must regularly review and evaluate the quality system, in order to ensure its proper functioning.
  A report shall be made of internal audits / management reviews.

  A documented routine shall be established in order to stipulate the control necessary in order to identify, store, protect, retrieve, maintain and delete registrations. Registrations shall be readable and easy to identify and retrieve.

2.8.2.4 INTERNAL AUDIT BY THE MANAGEMENT

The seafarer's doctor shall carry out internal revisions at least once a year, in order to determine whether the quality system is in accordance with the requirements for the practice, and whether the system has been efficiently implemented and maintained.
The management / seafarer's doctor shall in addition review the quality system in order to identify and implement measures to strengthen the system.

A routine for the planning and execution of internal revisions shall be established and documented.

2.9 DEADLINE FOR APPEAL OR APPLICATION FOR EXEMPTION

Appeal must be sent within three weeks from the receipt of a Declaration of unfitness or a limited/restricted medical certificate.

There is no deadline for application for exemption.

2.10 ELECTRONIC REGISTRATION AT THE PORTAL ALTINN

Electronic forms for issuance of medical certificates and declarations of unfitness can be used by all NMA approved seafarer's doctors with a Norwegian ID-/D-number.

NMA approved seafarer's doctors abroad will have to apply for a D-number. Forms for this purpose are to be found on the website of Brønnøysund Register Centre[1], and sent to the NMA for confirmation. When D-number is given, PIN codes should be ordered from the Agency for Public Management and e-Government (Difi): here. When the PIN codes have arrived by ordinary mail the NMA approved seafarer's doctors abroad have to register as a new user on the Norwegian portal of Altinn[2]: here

The NMA approved seafarer's doctor can submit Medical certificates and Declarations of unfitness immediately when the registration is done.

The Form is available directly from the Altinn portal. Choose «all forms», select «Agencies» find Norwegian Maritime Authority and select «Form for health certificates and declaration of unfitness».

2.11 RECORDING INFORMATION AT ALTINN

See "Guidance for electronic submission on medical certificate and declaration of unfitness" for further information.

2.12 CONDUCT OF MEDICAL EXAMINATION IN ACCORDANCE WITH THE REGULATIONS

Medical examination should be carried out with the purpose to assess whether the person is medically fit for work on board Norwegian ships, cf. Section 1 of the Regulations.

The seafarer's doctor should decide which examinations and supplementary tests to carry out, based on knowledge about best medical practice and sound medical assessment in accordance with the attachment to the Regulations and this guidance.

2.13 MEDICAL EXAMINATION WITHOUT ACCESS TO INTERNET

Access to internet is a prerequisite for being a NMA approved seafarer's doctor. This guidance is only applicable for the possibility that the internet connection incidentally is down – making electronic forms unavailable to the doctor. Paper forms should then be used.

[1] The Brønnøysund Register Centre develops and operates many of the nation's most important registers and electronic solutions. Administering Altinn, coordinating data in the public sector and providing advisory services are central tasks that make things easier for business and industry.

[2] Altinn is an internet portal for digital dialogue between businesses, private individuals and public agencies. Altinn is also a technical platform that government bodies can use to develop digital services.

The person should be provided with one of these forms: KS 0499-1 B/E or KS 0415 B/E. As soon as possible these forms should be replaced with an electronic form, and a new medical certificate/declaration of unfitness should be sent to the person.

It is important that this procedure is followed, to ensure that the information in the electronic registry and on the person's medical certificate/declaration of unfitness is identical.

2.14 DOCUMENTS THE PERSON SHALL PRESENT AT THE HEALTH EXAMINATION

- Proof of identification
- Latest issued medical certificate/declaration of unfitness
- Relevant reports from other medical doctors
- Statements from person when relevant

The person shall submit a self-declaration on his/her health on the form prescribed by the NMA. The self-declaration shall be signed in the presence of the seafarer's doctor and be kept by the seafarer's doctor.

3 THE PUBLIC ADMINISTRATIVE ACT

3.1 THE PUBLIC ADMINISTRATIVE ACT IS IMPORTANT TO THE SEAFARER'S DOCTOR

It is the responsibility of the Ministry of Trade, Industry and Fisheries and the Norwegian Maritime Authority as the administrative agency to issue medical forms to persons on Norwegian flagged ships.

The NMA has chosen not to employ doctors directly, rather approve external doctors to issue medical forms on their behalf – and empower them to act as administrative agencies as far as issuance of such medical forms is concerned. The person will have the same priveleges as if the doctor was employed directly by the NMA.

When an NMA approved seafarer's doctor makes a decision with legal authority in the Health Regulations, this is regarded as an administrative decision under the Public Administration Act. The seafarer's doctor therefore will have to comply with the administrative procedures derived from this Act.

3.2 THE APPROVED SEAFARER'S DOCTOR SHALL BE FAMILIAR WITH THE PUBLIC ADMINISTRATIVE PROCEDURES

The NMA approved seafarer's doctor should be familiar with all relevant administrative procedures, and be able to inform the person regarding rights of appeal and applications for exemptions, as well as assisting them in such procedures.

The seafarer's doctor is also expected to substantiate his/her decision stating medical as well as legal grounds for the decision.

3.3 ENGLISH VERSION OF THE PUBLIC ADMINISTRATION ACT

The English version of the Public Administration Act is to be found at the NMA website.

In all cases of dispute, the Norwegian version is the official one, and the one which overrule any discrepancies in translation.

Questions regarding public administration procedures or case handling, as well as questions regarding the Health Regulations should be forwarded to the responsible legal adviser at the NMA.

3.4 ADMINISTRATIVE LAW TOPICS OF SPECIAL IMPORTANCE TO THE SEAFARER'S DOCTOR

The below presentation on the Public Administrative Procedures, does not cover everything the NMA approved seafarer's doctor should be familiar with in his/her practice. The purpose of this presentation is to introduce some of the more important topics for the doctors.

Anyone who miss important topics in this presentation is encouraged to send new suggestions for topics to post@sdir.no. This will be an important contribution to the improvement of the guidance.

3.4.1 DECISIONS REGULATED BY THE PUBLIC ADMINISTRATION ACT

One has to look at Sections 1 and 2 to find the answer. Section 1 is concerned with activities conducted by admininstrative agencies, included private legal persons (NMA approved seafarer's doctors) who make individual decisions. An individual decision is defined in Section 2 as a decision regarding somone's rights (the right to get a medical certificate if requirements of the Health Regulations are met), or duties to one or several defined persons.

A decision by the approved seafarer's doctor to issue a medical certificate fulfils both requirements, and is therefore considered to be regulated by the Public Administration Act.

3.4.2 THE ASSESSMENT OF CONFLICTS OF INTEREST/LEGAL DISQUALIFICATION

Section 6 of the Public Adminsitration Act discusses how the NMA approved seafarer's doctor shall assess his/her own possible conflicts of interests and his/her possible legal disqualification. These requirements are objective – the question is not whether the seafarer's doctor him/herself thinks that he/she will be able to reach an impartial decision, rather whether the relationship between the doctor and the person, a shipping company, an employers' or employees' organization is of a kind apt to create doubt about his/her impartiality.

As a general rule, the NMA approved seafarer's doctor cannot be employed by a shipping company, an employees' or employers' organisation. This has nothing to do with the seafarer's doctor's personal character, but such a relationship could make other people uncertain about the seafarer's doctor's ability to discern between his different roles.

It has been accepted that the NMA approved seafarer's doctor may act as an external resource for a shipping company's occupational health service. The more work he/she does for a shipping company, the nearer he/she comes the point of discqualification regarding medical examinations of seafarers and individual decisions on behalf of the NMA. Should it happen that the amount of work the seaferer's doctor carries out is more or less like what would be done by en employed doctor of the company, he/she should not carry out medical examinations of persons employed by that company.

Furthermore, if the relationship between the seafarer's doctor and the company is so close that it looks like the seafarer's doctor cannot be impartial, he/she is most probably in a position where he/she should resign as an NMA approved seafarer's doctor.

Section 6 of the Public Administration Act also lists other causes of legal disqualification to handle cases under the Act, mostly concerned about family relationship, friendship and business relationship.

The assessment regarding possible conflicts of interest and possible legal disqualification shall be carried out by the seafarer's doctor prior to any medical examination which leads to an individual decision (medical certificate or declaration of unfitness).

3.4.3 DUTY TO PROVIDE GUIDANCE

The NMA approved seafarer's doctor is a representative of the governmental administration of the Health Regulations. As such, he/she has a duty to provide individualized adjusted guidance to persons who see him/her for a medical examination, cf. Section 11 of of the Public Administration Act.

The seafarer's doctor shall give such guidance that the person is capable to take care of his/her own interests and rights – which means that the doctor must inform about:

- The right to appeal the seafarer's doctor's decision
- The right to apply for an exemption from the requirements of the Health Regulations
- When additional information, examination or investigation is needed, the seafarer's doctor shall advise the person on how this can be obtained; see also the point below regarding the duty to clarify a case and the duty to inform, cf. section 17 of the Public Administration Act
- Inform about the content of the Health Regulations
- Inform about the content of the Appendices to the Health Regulations
- Assist in writing an appeal or application for exemption. It is important, though, that the persons name and signature is on the document, even if it is written by the seafarer's doctor
- Assist in sampling all necessary documents for the appeal or application for exemption
- Inform about the right to be represented/assisted by a lawyer or another deputized person in the case handling, cf. section 12 of the Public Administration Act

3.4.4 DUTY OF SECRECY

Following Section 13 of the Public Administration Act, the seafarer's doctor has duty to keep secret all information he/she has got during the handling of the case. This duty comes in addition to the general duty of secrecy derived from the Health Personnel Act.

3.4.5 DUTY TO CLARIFY THE CASE AND TO PROVIDE INFORMATION

The seafarer's doctor shall ensure that the case is clarified as thoroughly as possible before any administrative decision is made, which means that the facts of the case shall be clarified and that the seafarer's doctor has a duty to investigate circumstances which may be advantageous or disadvantageous to the person's case. Information provided by the person shall be verified where this is possible.

That the case shall be clarified as much as possible, should not be read literally. The seafarer's doctor should assess what is practical (e.g. time and cost consumption) in each case.

The main point is that the case is clarified sufficiently for the seafarer's doctor to be able to reach a decision whether a medical certificate should be issued or not in a sound way.

3.4.6 THE PERSON'S RIGHT TO ACQUAINT HIM-/HERSELF WITH THE DOCUMENTS OF THE CASE

The person has the right to see all documents of a case – i.e. the letters and the statements which have been written or have been used as evidence in the case.

The right to acquaint oneself with the documents does not include documents which have been written solely for internal case preparation. This would first and foremost be documents written by the seafarer's doctor him-/herself, i.e. the document is not shared with external parties or other administrative agencies.

Documents shared with the NMA or the Appelate Body will not be exempted from this rule.

3.4.7 THE DECISION OF THE SEAFARER'S DOCTOR SHALL ALWAYS BE JUSTIFIED

On making an individual decision (issuing a medical certificate or a declaration of unfitness), the seafarer's doctor shall always inform about the grounds for the decision. This information shall always be in writing.

This information is of special importance in cases where a declaration of unfitness is issued. In such cases the person needs the substantiation from the seafarer's doctor to be able to make up his/her mind regarding a possible filing of an appeal or application for an exemption.

The information shall be given together with the decision.

On discussing the grounds for the decision, the relevant parts of the Health Regulations or their appendices shall be pointed out as well as the medical reasoning in accordance with the facts of the case, sound clinical judgement, best clinical practice and the risk assessment carried out.

In cases where Declarations of unfitness, the content of the rules must be explained for the person.

3.4.8 DUTY TO INFORM THE PERSON ABOUT THE DECISION

The information about the decision usually should be given together with the appropriate medical certificate/declaration of unfitness.

If the decision is not made during the medical examination, notification about the decision should be sent to the person as soon as possible.

3.4.9 THE PERSON HAS THE RIGHT TO APPEAL AND APPLY FOR EXEMPTIONS FROM THE HEALTH REQUIREMENTS

A person who is not satisfied with the decision of the sefararer's doctor, has the right in accordance with Section 15 of the Health Regulations, cf. Section 28 of the Public Administration Act, to appeal the seafarer's doctor's decision. The appeal does not need to be justified or sensible in the eyes of others – if the person wants to appeal, he has the right to do so.

A person who regards him-/herself as medically fit for duty on board ships in accordance with the main objective of the Health Regulations, even though the formal requirements of the appendices are not met, has the right to apply for an exemption from the formal requirements in accordance with Section 16 of the Health Regulations, cf. Section 28 of the Public Administration Act.

3.4.10 THE DUTIES OF THE SEAFARER'S DOCTOR IN CASES OF APPEAL OR APPLICATIONS FOR EXEMPTIONS

In such cases, the seafarer's doctor's duties consist of the 6 steps mentioned below:

1. Assess the case once again, and decide whether there is new information which should bring about a change of the original decision.
2. Inform the person of the right to appeal (or apply for exemption), and how such cases are handled.
3. Inform about the Health regulations in a way that enables the person to formulate his/her appeal or application properly.
4. If the person so wants, assist in the writing of the appeal/application. It is important that the document contains information regarding which decision the appeal is about, what change in the decision that is asked for, and the grounds claimed as reason for the appeal.
5. Ensure that the person has signed the appeal/application. It is the person who has the right to appeal/apply for exemption, not the seafarer's doctor, and it is the person who

shall sign the document, even if it is written by the seafarer's doctor. It should however appear from the document which assistance the doctor has provided.

6. Post the appeal/application with all necessary documents (see duty to clarify the case) to the NMA.

The deadline for appeal is three weeks from the time the person received information about the decision. There is no deadline for an application for exemption.

In this Regulations there are no "relative" and "absolute contraindications" – which was the case with the expired Regulations. In the present Regulations the medical appendix is based on the requirements of the STCW Convention and the ILO/IMO Guidelines on the medical examinations of seafarers. This implies that the Appelate Body has no authority to grant exemption on minimum requirements lower than the requirements of the STCW convention. As the Health Regulations include the same minimum requirements as the STCW Convention, the freedom to grant exemptions is strongly limited. The NMA approved seafarer's doctors will need to carry out sound clinical judgement to a greater extent than they did under the previous Regulations.

The possibility of applying for an exemption is continued for possible special cases where this might be appropriate.

3.4.11 POSSIBILITY TO CHANGE THE DECISION

If the seafarer's doctor should receive information which indicates that his/her decision about issuing a declaration of unfitness or a limited/restricted medical certificate is not correct, he/she has the power to change this decision of his/her own motion.

The possibility to change a decision in a way that would be disadvantageous to the person (from medical certificate to a declaration of unfitness or a limited/restricted medical certificate) is strongly limited – which means that the seafarer's doctor should discuss this case with the NMA prior to making such decisions.

The possibility for change of decision is not any longer there, when the case has been considered by the Appelate Body. The decision by the Appelate Body cannot be changed by the seafarer's doctor, due to the fact that the case is decided by a superior body (Appelate Body). Change of the decision shall in such cases be done by the Appelate Body, in the same way as the seafarer's doctor can change his/her own decision in accordance with section 35 of the Public Administration Act.

3.4.12 LEGAL COSTS

A person may have appreciable costs related to a change of decision covered, if the change is advantageous to the person, cf. the Public Administration Act section 36. For further information, please contact the NMA.

3.4.13 ADMINISTRATIVE CHECKLIST FOR THE MEDICAL EXAMINATION

- Has the identity of the person been confirmed by passport or other proof of identity?
- Has the person been informed that the seafarer's doctor acts in the capacity as a NMA approved seafarer's doctor, and not in the capacity of a general practitioner, an occupational physician or other role?
- Is the declaration signed, dated and stamped?
- If relevant, has the person signed personally on the documents regarding appeal or application for exemption?
- If relevant, are all necessary documentation filed together with the appeal/application for exemption?

4 WORKING PLACES AND JOB POSITIONS ON BOARD SHIPS

4.1 DECK DEPARTMENT

- Captain/master
  The captain has the senior authority and the responsibility for the safety and operation of the vessel and for prevention of pollution. The captain is furthermore responsible for fulfilling obligations laid down by law (the Norwegian Maritime Code, the Ship Labour Act, the Ship Safety and Security Act), for ensuring that the watchkeeping arrangements on board do not at any time compromise safety, that the navigation is carried out by a competent person, and that the crew is well rested. The captain is also responsible for i.a. safe loading/unloading, that the certificates are valid at all times, and that the ship's safety management system is being followed up
- Chief officer/Chief mate
  The chief officer is the captain's substitute and right hand. As laid down by law, he is the second in command on board, and reports directly to the captain. Should the captain become incapacitated, the chief officer will step into the role of the captain.
  The chief officer is in charge of the deck department, and is responsible for planning the loading/unloading and the ship's voyage in cooperation with the captain. The chief officer is often also the designated security and medical officer. Is listed in the muster list and emergency instructions
- 2nd officer
  Deck officer, undertakes bridge watches, participates in route planning and maintains/upgrades charts and nautical publications. Furthermore responsible for the maintenance of all life-saving and fire-fighting equipment as directed by the chief officer. Is listed in the muster list and emergency instructions
- 3rd officer
  Deck officer, undertakes bridge watches. Odd jobs as directed by chief officer/2nd officer. Is listed in the muster list and emergency instructions

- Cadet
  Training position. Period of practical training on board following school. Practical/theoretical training together with the person responsible for the training on board, in accordance with the applicable requirements. Otherwise same as able seafarer deck or able seafarer engine. Not listed in the muster list and emergency instructions
- Bosun/Boatswain
  In charge of all work on deck. Delegates tasks and participates to a certain extent. Normally not a part of the navigational watch. Is listed in the muster list and emergency instructions
- Able seafarer deck/Able-bodied seafarer (AB)
  General maintenance and cleaning tasks on deck. Cleans and prepares cargo spaces and cargo tanks. Forms part of the navigational watch. Periodically physically strenuous work. Exposed to the elements (cold/heat depending on trade area). Is listed in the muster list and emergency instructions
- Ordinary seafarer
  Training position, otherwise same as able seafarer deck. Is listed in the muster list and emergency instructions
- Trainee rating deck
  Training position, otherwise same as able seafarer deck. Not listed in the muster list and emergency instructions

4.2 ENGINE DEPARTMENT

- Chief engineer officer
  The chief engineer officer is the senior engineer officer who is responsible for the mechanical propulsion and the operation and maintenance of the mechanical and electrical installations on board the ship. The chief engineer ensures that the ship's operational and safety management system is maintained by competent technical personnel in accordance with laws and regulations. Within his/her department, the chief engineer officer is also responsible for i.a. HSE, delivery and storage of goods, stays in shipyards and finances. Usually works in the daytime with tasks that are not physically demanding, but is in charge of operations in the event of damage or fire. May be a smoke diver, and has a leading role during evacuation of the ship
- 2nd engineer officer
  The 2nd engineer officer is next in rank to the chief engineer officer and will take over the responsibilities of the chief engineer officer should become incapacitated. The 2nd engineer officer is a working supervisor, often works nights and has physically demanding tasks. Often has smoke diving tasks
- 3rd engineer officer
  The 3rd engineer officer is on the same operational level as the officer in charge of an engineering watch, and is thus the person responsible for a watch in the engine-room during a given time period. Often works nights and has physically demanding tasks. May have demanding tasks in the event of damage or fire, such as smoke diving

- 4th engineer officer
  Rarely used in Norway, but assists the 3rd engineer officer in his/her tasks. Is listed in the muster list and emergency instructions
- Fitter/Repairman deck/engine
  The fitter is the "janitor" of the ship. Performs tasks like a mechanic, but will in addition carry out a lot of steel work and welding. Physically demanding tasks with some heavy lifting
- Pumpman
  The pumpman oversees, operates and maintains cargo and ballast pumps. Participates at all times during loading/unloading, and participates in tank cleaning. The tasks require accuracy, good understanding/knowledge and safety awareness. May work both day and night. Is exposed to environmental factors such as chemicals, noise, etc.
- Motorman/Able seafarer engine
  Operates machinery, carries out engine repairs, maintenance and cleaning tasks in the ship's engine room as directed, forms part of an engineering watch together with an engineer officer, works with turning, welding and plumbing tasks, often undertakes mooring tasks during arrival and departure. Physically demanding labour, exposed to environmental factors such as chemicals, dust, noise, etc. Is listed in the muster list and emergency instructions
- Greaser/Wiper
  Assists the motorman. Is listed in the muster list and emergency instructions
- Refrigeration engineer officer
  Supervises the operation, maintenance and repair of the ship's HVAC equipment/system, cold service system, ventilation, refrigeration system and air conditioning system. Position on large freezer ships and cruise ships. Is listed in the muster list and emergency instructions
- Engine-room attendant
  Same as motorman. Position on smaller ships in coastal waters. Is normally alone in the machinery space, and is then additionally responsible for the engine department. Is listed in the muster list and emergency instructions
- Environmental engineer officer
  Position on large passenger ships. Takes care of waste and is responsible for incinerators. Is listed in the muster list and emergency instructions
- Stoker
  Oversees and maintains large marine boilers. Forms part of watches, and has physically demanding tasks. Is listed in the muster list and emergency instructions

4.3 GALLEY

- Steward
  Administrative position. Head of cook and catering staff. Responsible for purchasing provisions and planning the menu. The position is mostly gone in cargo ships, but is found on larger passenger ships. Is listed in the muster list and emergency instructions

- Cook
  Prepares all food. Cleans galley and provision rooms. Some office work. Is listed in the muster list and emergency instructions

4.4 OTHER JOB POSITIONS ON BOARD

- Electrician/Electro-technical officer/rating
  Supervises and maintains all the electrical equipment with appurtenant components. Mostly works in the field, no heavy lifting. Is listed in the muster list and emergency instructions if this position forms part of the safe manning personnel
- Catering personnel
  On passenger ships: General cleaning of the hotel department. Waiter.
  On cargo ships: Cleaning of all accommodation spaces. Assists the cook during food service, etc. May be listed in the muster list and emergency instructions
- Mechanic
  Inspects, repairs and maintains equipment and machinery. Generally non-physically demanding work, some heavy lifting. Not listed in the muster list and emergency instructions
- ROV pilot
  Only task is to remote control mini submarines on the ocean floor. Requires high attentiveness and alertness. Not listed in the muster list and emergency instructions
- Fisherman
  Work on deck in connection with various fishing equipment. Cleaning and processing of catch. Physically demanding labour. Exposed to the weather elements. Normally not part of the navigational watch. May be listed in the muster list and emergency instructions

5 JOB POSITIONS ON MOBILE OFFSHORE UNIT

- Offshore installation manager/Platform manager
  The senior administrative manager on board. Overall responsibility for operations, safety and emergency preparedness. Responsible for compliance with relevant rules and regulations
- Stability section leader
  Responsible for navigation, positioning and stability. Has responsibilities and tasks related to i.a. maintenance and cargo handling
- Control room operator
  Works closely with the stability section leader, and is responsible for the operation and coordination of the systems in the control room. Forms part of shift schedule. Equivalent to Deck/Engineer officer Class 4
- Technical section leader
  Equivalent to Engineer officer Class 1
- Technical assistant
  Equivalent to Engineer officer Class 2

- Engine room operator
  Equivalent to Engineer officer Class 4

6 TRADE AREAS

The Regulations of 4th November 1981 No 3793 concerning trade areas defines the different trade areas, divided in three groups: 1) Domestic voyages, 2) Foreign voyages and 3) Trade areas for fishing vessels of less than 15 m in overall length.

The following trade areas are defined. The below list is not a complete description of the different trade areas. For the complete description, please visit the Regulations, which can be found here.

Trade Areas - Domestic Voyages		
Section 6	Trade on lakes and rivers	Trade on navigable Norwegian lakes and river
Section 7	Trade Area 1 –	Trade on Norwegian lakes and rivers, and inner parts of fjords and in other Norwegian waters where smooth waters can generally be expected. Annex I.
Section 8	Trade Area 2 –	Voyage in Norwegian waters which are protected against waves and wind from the open sea, including more restricted waters. Annex II.
Section 9	Trade Area 3 -	Voyage on the Norwegian coast where the stretches without protection against waves and wind from the open sea do not exceed 5 nautical miles, including more restricted waters. Annex III.
Section 10	Trade Area 4 -	Voyage in sheltered waters where the unsheltered stretches do not exceed 25 nautical miles. Annex IV
Section 11	Small coasting	Voyage on the Norwegian coast where the unsheltered stretches exceed 25 nautical miles, including all more restricted waters, but never farther off the coast than 20 nautical miles from the Base Line (ref. Regulation of 14 June 2002 No. 625 issued by the King).
Trade Areas – foreign voyages		
Section 13	Great Coasting	Voyages in small coasting as well as voyages in Swedish, Danish and German waters east of a line Lindesnes – the western entrance of Limfjord to a line Karlskrona – Swinoujscie.
Section 14	North Sea and Baltic trade	Voyages in small coasting as well as voyages in Skagerrak, Kattegat, the Baltic Sea including the Gulf of Bothnia and the Gulf of Finland, the North Sea south of latitude 61°N, and trade to Great Britain, Ireland east of longitude 8° W, and the English Channel limited by a line Brest – Cork.

Section 15	Euorpean trade	All trade within the following outer boundaries: the White Sea, Svalbard, Jan Mayen, Iceland, Madeira, the Azores, the Canary Islands, the west coast of Africa north of latitude 30° N, the Mediterranean and the Black Sea.
Section 16	Short international voyage	An international voyage (ref. section 17) where the ship does not proceed more than 200 nautical miles from a port or place where passengers and crew can be brought to safety, and where the distance between the last port of call in the country of embarkation and the final port of destination does not exceed 600 nautical miles.
Section 17	International voyage	A voyage from a country to which the International Convention for the Safety of Life at Sea, 1974 (SOLAS Convention) applies to a port outside such a country, or the converse of this; and in this connection any territory for whose international relations a Contracting Government is responsible, or for which the United Nations Organization is the administering authority, shall be regarded as a separate country.
Section 18	Overseas voyage	A voyage from one continent to another across one of the oceans
Section 19	Unrestricted voyages	Voyages with unrestricted trade areas.
Trade Areas – Fishing vessels of less than 15 m in overall length		
Section 21	Fjord fishing	Fishing and sealing/whaling in waters on the Norwegian coast where unsheltered stretches do not exceed 5 nautical miles (ref. Annex III), or unsheltered waters up to 3 nautical miles from harbours or other protected waters.
Section 22	In-shore fishing	Fishing and sealing/whaling within 12 nautical miles from the Base Line.
Section 23	Bank fishing I (Ground fishing I)	Fishing and sealing/whaling within the area bounded by the coordinates described in Section 23 of the Regulations – mainly along the Norwegian coast, and parts of Skagerrak and Kattegat.
Section 23	Bank fishing II (Ground fishing II)	Fishing and sealing/whaling within 200 nautical miles from the Base Line, and shelter and rest near Bear Island in the period from 1 May to 31 August within the area bounded by the co-ordinates described in Section 23 of the Regulations.
Section 24	Deepsea fishing I	Fishing and sealing/whaling within the area defined as bank fishing II, and also the North Sea, the Skagerrak, the Cattegat with adjacent waters, limited between 50° N – 62° N and 10° W.
Section 24	Deepsea fishing II	Fishing and sealing/whaling in all waters except waters with open/scattered drift ice concentration

		(4/10-6/10) or higher beyond 200 nautical miles from the Base Line.
Section 25	Fishing in ice-covered waters I	Fishing and sealing/whaling in all waters except waters with a heavy/very heavy drift ice concentration (8/10-9/10) or higher beyond 200 nautical miles from the Base Line.
Section 25	Isfarvann II	Fishing and sealing/whaling in all waters.

7 SAFE MANNING AND SAFETY FUNCTION

7.1 SAFE MANNING LEVELS

Safe manning levels for a specific vessel or type of vessels are laid down by the Norwegian Maritime Authority in accordance with Regulations of 18 June 2009 No. 666 concerning the manning of Norwegian ships (Manning Regulations 09).

"Safe manning levels" are defined in section 7 of the Regulations:

"For each ship the Norwegian Maritime Authority shall determine the minimum safe manning including job specifications and qualification requirements etc. which are necessary to maintain the safety of the ship and those on board and prevent pollution of the marine environment."

According to section 8 of the Regulations, the company shall propose a minimum safe manning level which is necessary to maintain the safety of the ship and those on board and prevent pollution of the marine environment. This proposal shall be based on

a) Safety Management System
b) risk analysis
c) evacuation analysis, for ships for which such analysis is required
d) organization plan
e) job instructions for each post in the organization
f) the technical standard of the ship
g) propulsion machinery output
h) alternations
i) job combinations or/and overlapping competence
j) working hours arrangements to be applied in each case
k) number of passengers

The proposed safe manning shall cover all relevant operations, tasks and functions for the safe operation of the ship, including

a) watchkeeping both at sea and in port, as well as safety and emergency response drills
b) operation and maintenance of vital operating systems, including propulsion machinery and rescue and emergency response systems
c) operation and maintenance of technical equipment on the bridge and in machinery spaces, as well as in other control rooms

d) operation and maintenance of internal and external communication equipment
e) maintenance of critical components
f) catering requirements of the crew, as well as required cleaning
g) anchoring and mooring, as well as making the ship ready for the voyage
h) maritime operations such as navigation, manoeuvring, stability, etc.
i) monitor the loading and unloading, securing and placement of cargo (dangerous cargo, etc.)
j) first aid, treatment of injuries, and medical assistance
k) safety training and other safety work, including the tasks specified in the Regulations of 22 June 2004 No. 972 on security anti-terrorism and anti-piracy measures and the use of force on board ships and mobile offshore drilling units
l) familiarization of new crew members
m) inspection of the intake of bunkers, supplies and provisions
n) other operations essential to safe manning

7.1.1 CAN A PERSON ON SICK-LEAVE BE A PART OF THE SAFETY MANNING?

The Regulations do not prohibit that a person on partial sick leave can be a part of the minimum safety manning, although this will only be relevant in very special cases, where mitigating measures are established as appropriate.

Being a part of the minimum safety manning also implies being on emergency preparedness during rest hours. Following Section 11 of Regulations of 18th June 2009 No 666 concerning the manning of Norwegian ships (Manning regulations 09) it is not allowed for a ship to leave port if the minimum safety manning has shortcomings. A person on partial sick leave will not be able to form a part of the emergency preparedness while resting between working hours – as he/she will not have enough working capacity to work regular hours, and therefore cannot be assumed to have any additional working capacity for use after working hours. If the individual is not forming a part of the emergency preparedness, this is regarded a shortcoming in accordance with section 11 of the Regulations mentioned above, and the ship will not be allowed to leave port.

Conclusion:

On this background, the NMA concludes that an individual on partial sick leave as a general rule cannot form a part of the minimum safety manning.

7.2 ADDITIONAL MANNING

In order to ensure safe manning, the company and master shall assess whether additional manning is necessary. Additional manning is the extra manning the company in agreement with the master considers necessary to have on board to carry out operations that cannot be handled by the minimum safe manning alone without compromising the safety of the ship and those on board.

Even if persons in the additional manning do not form a part of the minimum safe manning, they can have safety functions as a part of their duties.

7.3 SAFETY FUNCTION

Safety functions are duties covered by Chapter VI in the STCW Code, Part A: "Standards regarding emergency, occupational safety, security, medical care and survival funtions".

If the job description/task description contains duties which are covered in one of the following Sections of Chapter VI of the STCW Code A, the person is regarded to "have a safety function".

Individuals who do not belong to the minimum safe manning, still can have a safety function. All persons who belong to the minimum safety manning have safety functions.

A-VI/1-1	Survive at sea in the event of ship abandonment
A-VI/1-2	Minimize the risk of fire and maintain a state of readiness to respond to emergency situations involving fire Fight and extinguish fire
A-VI/1-3	Take immediate action upon encountering an accident or other medical emergency
A-VI/1-4	Comply with emergency procedures Take precautions to prevent pollution of the marine environment Observe safe working practices Contribute to effective communications on board ship Contribute to effective human relationships on board ship Understand and take necessary actions to control fatigue
A-VI/2-1	Take charge of a survival craft or rescue boat during and after launch Operate a survival craft engine Manage survivors and survival craft after abandoning ship Use locating devices, including communication and signalling apparatus and pyrotechnics Apply first aid to survivers
A-VI-2-2	Understand the construction, maintenance, repair and outfitting of fast rescue boats Take charge of the launching equipment and appliance as commonly fitted, during launching and recovery Take charge of a fast rescue boat as commonly fitted, during launching and recovery Take charge of a fast rescue boat after launching Operate a fast rescue boat engine
A-VI/3	Control fire-fighting operations aboard ships Organize and train fire parties Inspect and service fire-detection and fire-extinguishing systems and equipment Investigate and compile reports on incidents involving fire
A-VI/4-1	Apply immediate first aid in the event of accident or illness on board
A-VI/4-2	Provide medical care to the sick and injured while they remain on board Participate in coordinated schemes for medical assistance to ships
A-VI/5	Maintain and supervise the implementation of a ship security plan Assess security risk, threat and vulnerability Undertake regular inspection of the ship to ensure that appropriate security measures are implemented and maintained Ensure that security equipment and systems, if any, are properly operated, tested and calibrated Encourage security awareness and vigilance
A-VI/6-1	Contribute to the enhancement of maritime security through heightened awareness Recognition of security threats Understanding of the need for and methods of maintaining security awareness and vigilance
A-VI/6-2	Maintain the conditions set out in a ship security plan Recognition of security risks and threats Undertake regular security inspections of the ship Proper usage of security equipment and systems, if any

8 TEMPLATE FOR ISSUING THE INFORMED DECISION IN WRITING TO THE EMPLOYER IN CONNECTION WITH DECISIONS PURSUANT TO THE REGULATIONS

8.1 DECISION

Pursuant to Regulations of 5 June 2014 No. 805 on medical examination of persons on Norwegian ships and mobile offshore units

I have today made a decision concerning the issue of:

☐ **MEDICAL CERTIFICATE** pursuant to section 10 of the Regulations

☐ **MEDICAL CERTIFICATE WITH LIMITATION** pursuant to section 11 of the Regulations

Position: Limitation: ……..
Trade area: Limitation: ……..
Validity: Limitation: ……..

☐ **PERMANENT UNFITNESS** pursuant to section 12 of the Regulations

☐ **PROVISIONAL UNFITNESS** pursuant to section 12 of the Regulations

☐ **TEMPORARY UNFITNESS** pursuant to section 12 of the Regulations

☐ **POSTPONED EXECUTION** of unfitness pursuant to section 17 of the Regulations

Postponed execution is decided after consent from

☐ Company ……

☐ Master on (ship): ……

The postponement is valid until: ……

The postponement presupposes that you send an application for exemption or appeal my decision to the Appellate Body.

Grounds

Your condition fails the following section of the Appendix to Regulations of 5 June 2014 No. 805 on medical examination of persons on Norwegian ships and mobile offshore units:

☐ Letter A, point……

☐ Letter B, point……

☐ Letter C, point……

☐ Letter D, point……

☐ Letter E, point……

Medical justification

[Free text]

Appeal against decision made by a seafarer's doctor

You can appeal against this decision. The appeal should be addressed to the Norwegian Maritime Authority and sent to the undersigned, who will then reconsider the decision. If I decide to maintain the decision, the case will be forwarded to the Norwegian Maritime Authority for processing by the Appellate Body for seafarers.

Any new information which might be significant to the case should be provided, and attached to the appeal or forwarded separately.

The deadline for appeal is within three weeks of the date when you received the original decision.

Application for exemption

You can also apply for an exemption from the requirements laid down in the Regulations, pursuant to section16. This will be applicable only in special circumstances, as the Norwegian regulatory requirements on certain aspects (eg. vision standards) are identical to the international minimum requirements from which exemptions cannot be granted.

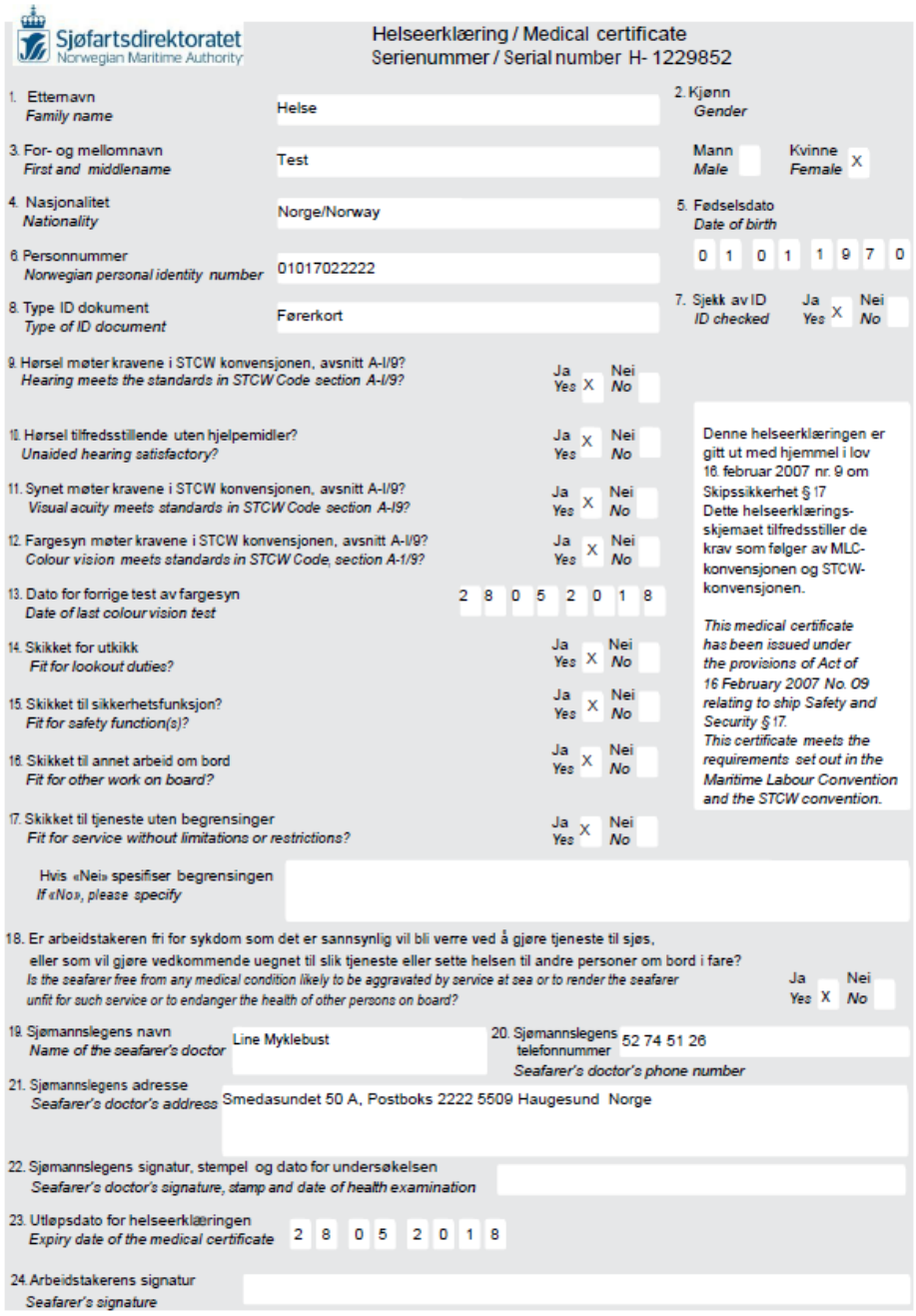
There is no deadline for application for exemption.

Guidance

If you need guidance in the drafting of an appeal or application for exemption, or if you have questions regarding the process of appeal and exemption cases, you are welcome to contact me for assistance.

Best regards,
N.N.

9 EXAMPLE OF PROPERLY COMPLETED MEDICAL CERTIFICATE



Sjøfartsdirektoratet
Norwegian Maritime Authority

Helseerklæring / Medical certificate
Serienummer / Serial number H- 1229852

1. Etternavn / *Family name*	Helse
3. For- og mellomnavn / *First and middlename*	Test
4. Nasjonalitet / *Nationality*	Norge/Norway
6. Personnummer / *Norwegian personal identity number*	01017022222
8. Type ID dokument / *Type of ID document*	Førerkort

2. Kjønn / *Gender*: Mann / *Male* ☐ Kvinne / *Female* X

5. Fødselsdato / *Date of birth*: 0 1 0 1 1 9 7 0

7. Sjekk av ID / *ID checked*: Ja / *Yes* X Nei / *No* ☐

	Ja / Yes	Nei / No
9. Hørsel møter kravene i STCW konvensjonen, avsnitt A-I/9? / *Hearing meets the standards in STCW Code section A-I/9?*	X	☐
10. Hørsel tilfredsstillende uten hjelpemidler? / *Unaided hearing satisfactory?*	X	☐
11. Synet møter kravene i STCW konvensjonen, avsnitt A-I/9? / *Visual acuity meets standards in STCW Code section A-I/9?*	X	☐
12. Fargesyn møter kravene i STCW konvensjonen, avsnitt A-I/9? / *Colour vision meets standards in STCW Code, section A-1/9?*	X	☐
13. Dato for forrige test av fargesyn / *Date of last colour vision test*: 2 8 0 5 2 0 1 8		
14. Skikket for utkikk / *Fit for lookout duties?*	X	☐
15. Skikket til sikkerhetsfunksjon? / *Fit for safety function(s)?*	X	☐
16. Skikket til annet arbeid om bord / *Fit for other work on board?*	X	☐
17. Skikket til tjeneste uten begrensinger / *Fit for service without limitations or restrictions?*	X	☐

Hvis «Nei» spesifiser begrensingen / *If «No», please specify*

Denne helseerklæringen er gitt ut med hjemmel i lov 16. februar 2007 nr. 9 om Skipssikkerhet § 17
Dette helseerklærings-skjemaet tilfredsstiller de krav som følger av MLC-konvensjonen og STCW-konvensjonen.

This medical certificate has been issued under the provisions of Act of 16 February 2007 No. 09 relating to ship Safety and Security § 17.
This certificate meets the requirements set out in the Maritime Labour Convention and the STCW convention.

18. Er arbeidstakeren fri for sykdom som det er sannsynlig vil bli verre ved å gjøre tjeneste til sjøs, eller som vil gjøre vedkommende uegnet til slik tjeneste eller sette helsen til andre personer om bord i fare? / *Is the seafarer free from any medical condition likely to be aggravated by service at sea or to render the seafarer unfit for such service or to endanger the health of other persons on board?* Ja / *Yes* X Nei / *No* ☐

19. Sjømannslegens navn / *Name of the seafarer's doctor*: Line Myklebust

20. Sjømannslegens telefonnummer / *Seafarer's doctor's phone number*: 52 74 51 26

21. Sjømannslegens adresse / *Seafarer's doctor's address*: Smedasundet 50 A, Postboks 2222 5509 Haugesund Norge

22. Sjømannslegens signatur, stempel og dato for undersøkelsen / *Seafarer's doctor's signature, stamp and date of health examination*

23. Utløpsdato for helseerklæringen / *Expiry date of the medical certificate*: 2 8 0 5 2 0 1 8

24. Arbeidstakerens signatur / *Seafarer's signature*

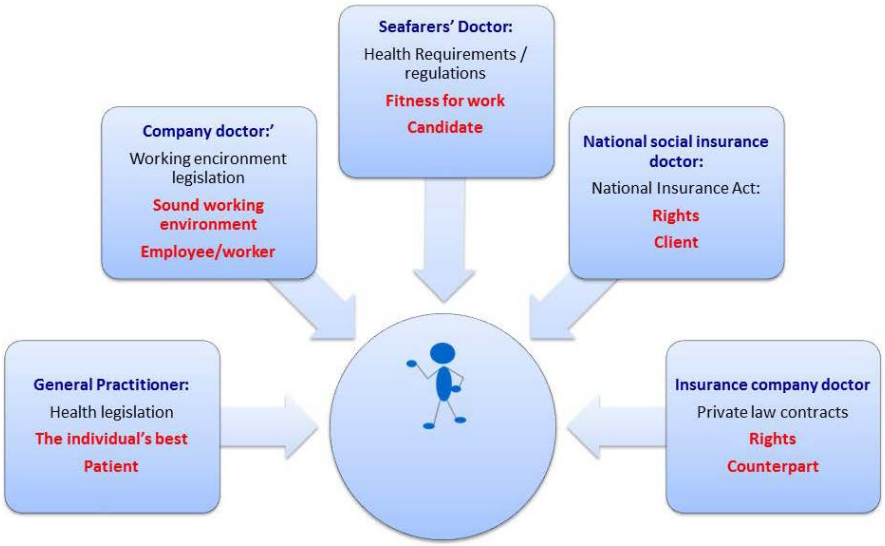
10 THE WORK OF THE SEAFARER'S DOCTOR – SELECTION MEDICINE

10.1 DIFFERENT ROLES

Being a seafarer's doctor involves ensuring that the person fulfils the health requirements pursuant to the Regulations on medical examination of persons on board Norwegian ships and mobile offshore units.

This is a role which is substantially different from being a treating physician, doing preventive work as a municipal medical officer, having a working environment focus as a company doctor or

assessing rights with regards to the Norwegian Labour and Welfare Administration (NAV) or an insurance company. The figure below clearly demonstrates this.



An individual who is meeting with a doctor may be considered as a patient by the general practitioner, as a person by the company doctor, as a candidate for passing a selection based on criteria by the seafarer's doctor, as a client entitled to his or her rights by NAV or as an opposing party by an insurance company.

In many cases the doctor will have several roles simultaneously - especially in sparsely populated areas. In such cases it is extremely important that the doctor is conscious of his/her role, and informs the individual person seeing the doctor in such a way that the person understands the doctor's current medical role.

10.2 BOUNDARIES BETWEEN MEDICAL ROLES

Different laws apply to different medical roles. How the seafarer's doctor practises his/her profession as a doctor is regulated by the Health Personnel Act. The work doctors perform however, may be regulated by other Acts. The practise of a seafarer's doctor is regulated by the Ship Safety and Security Act, the Ship Labour Act and the Public Administration Act (PAA).

Individual decisions are made in accordance with the PAA, and are made on behalf of the Authority. The seafarer's doctor impinges on an individual's rights when they limit the trade area, capacity on board or the validity of the medical certificate. The Norwegian Maritime Authority manages the Regulations and carries out supervision of the seafarer's doctors to ensure that they perform their work in accordance with these.

The Working Environment Act does not apply on ships, however, company doctors onshore practise pursuant to this Act. The Norwegian Labour Inspection Authority is the superior authority for the company doctor.

The regular general practitioner practises pursuant to the Health Care Act, which is managed by the Norwegian Directorate of Health, and the Norwegian Board of Health Supervision carries out the supervision of the practise of the regular general practitioner.

	Seafarer's Doctor	Company Doctor	General Practitioner
Role	Verification	Prevention	Treatment
Focus	(Fitness for work) / Absence of disease	Working environment / occupational injuries and diseases	Individual health
Prespective	Maximum two years ahead	Past, present and future	(past) present and future
Tools	Medical selection in relation to requirements	Environmental factors affecting the worker	Medical treatment of illness and injuries
Aim	Contribution to the safety of the working environment	Prevention of occupational injuries and diseases	Prevention and treatment of disease regardless of cause
Acts	Ship Safety Act	Working Environment Act	Health and Care Act
The individual	A Candidate (for getting a medical certificate)	An employee/worker	A Patient

10.3 POSITIVE VS. NEGATIVE SELECTION

Selection medicine is the part of medicine involved with the selection of persons based on medical criteria.

In principle, there are two different ways of selecting individuals, namely so-called positive and negative selection.

	Positive selection	Negative selection
Method	Inclusion of people considered fit based on established performance	Exclusion of people based on established dysfunction
Starting point	All people are considered unfit, and you select the fit people based on the criteria	All people are considered fit, and you select the unfit people based on the criteria
Criteria	Performance requirements	Medical criteria
Keywords	«Unfit until proven fit»	«Fit if not proven unfit»

The seafarer's doctor's examination is mostly considered with negative selection. In some cases positive selection will also be used, i.e. if there is a reason to believe that physical performance requirements are not satisfied, for instance when the person is overweight.

10.4 THE AIM OF THE EXAMINATION

The aim of a medical examination by a regular general practitioner is to treat the individual for the individual's own benefit.

The aim of a medical examination by a company doctor is to register possible health risks in the work place, so that suitable actions may be implemented in order to prevent work-related illness and injury both for the individual doctor and for other persons.

The aim of a medical examination by a seafarer's doctor is to ensure that the person is medically fit to safely perform his or her routine and emergency duties, is not suffering from any medical condition likely to be aggravated by service at sea and that he/she does not constitute a danger to the health and safety of other persons on board or to the safe operation of the ship.

It is important that the seafarer's doctor is aware of the angle he is expected to take when examining the person's health.

10.5 LIST OF DISEASES AND CONDITIONS - APPENDIX E

This list is not exhaustive. It is estimated that there could currently be close to 30,000 known medical conditions (diagnoses), and it is not possible to make a complete list.

This means that when the seafarer's doctor is facing a condition not mentioned in the list of diseases and conditions, the list should be used in conjunction with review of the concerned medical condition with regards to the requirements of the objects clause in the Regulations (section 1).

In other words, the fact that a condition is not mentioned in the list should not automatically lead to issuance of a medical certificate. If the conditions are not found to be compatible with the safe performance of duties, or if there is a danger to the safe operation of the ship or to the safety of others on board, a limitation in the medical certificate or a declaration of unfitness should be considered. The condition is classified according to ICD 10, in accordance with the final point in Appendix E.

In addition to the guidelines found directly in the list of conditions, it might be useful to read "Handbook for Medical Examiners" - a guide to the IMO/ILO's Guidelines on Medical Examination of Seafarers, published by the Norwegian Centgre for Maritime and Diving Medicine in cooperation with the International Maritime Health Association, see http://handbook.ncmm.no.

11 RISK ASSESSMENT

Risk assessment in this regard is a step-by-step process. The method is described below. This is the principle for how you should think, but it does not mean that this list should be followed mechanically in all cases. Some cases are simple, and easy to make a decision on. Others are more complicated and require a more thorough approach.

11.1 FIRST STEP: IDENTIFICATION OF THE TYPE OF INCIDENT WHICH MAY OCCUR WITH REGARD TO THE UNDERLYING CONDITION(S)

When the underlying medical condition is known, you should identify the incidents to take into consideration with regard to safety. There may be an increased risk of syncope, infarct, spasms, thrombosis, bleeding, paralysis, psychotic reactions, depression, anxiety, etc.

The essential point of this identification is whether the condition can lead to loss of either physical or mental/cognitive function.

Loss of physical function may present itself as e.g. reduced muscle strength, tempo, coordination, balance, etc.

Loss of mental function may present itself as e.g. loss of memory, vigilance, alertness, ability to react, ability to concentrate, prioritisation, perception of reality, situation awareness, depression, mania, hallucinations, simultaneous processing, overview, etc.

Either may present a risk to the seafarer, other crew members or the ship.

11.2 SECOND STEP: GENERAL ASSESSMENT OF LIKELIHOOD OF SUCH AN INCIDENT OCCURRING

Based on the knowledge of the underlying conditions the likelihood of an unwanted incident occurring within the relevant time frame – namely a maximum of 2 years – can be assessed for the diagnostic group to which the person concerned belongs.

Evidence on this point is in short supply. It is possible to find bits and pieces in certain areas of the specialist literature, particularly in general overview articles, and you then have to search in headlines such as course, prognosis, complications, treatment, adverse effects, follow-up.

Once this likelihood is found, it will not necessarily be applicable to the person concerned. He may belong to those with an exceptionally good prognosis or those with an exceptionally bad prognosis within the group, and may not necessarily represent the average.

In the list of medical conditions in this guide we provide some background information where this is known. This part is likely to build up, and the seafarer's doctor should always use the last edition of this guidance.

11.3 THIRD STEP: INDIVIDUAL ASSESSMENT OF LIKELIHOOD

With the general assessment of likelihood as a basis and knowledge about the individual candidate, the likelihood may be modified based on factors such as age, gender, general condition, co-morbidity, degree and stage of illness, duration of illness, duration of observation, effect of treatment, follow-up and surrounding framework, etc.

This is a challenging step in the process, and requires experience and clinical exercise of discretion in line with best practice.

11.4 FOURTH STEP: CONSEQUENCES FOR THE WORK SITUATION

This is the next step. This is not only related to medical consequences of an underlying condition, but also any safety-related consequences this may have for the work situation. The work situation and duties must therefore be known and understood.

Likelihood	Consequence		
	Insignificant	Significant	Serious
Very low (< 2% per year) = 1	1 Acceptable risk	2 Acceptable risk	3 Acceptable risk
Low (2-5% per year) = 2	2 Acceptable risk	4 Acceptable risk if mitigated	6 Acceptable risk if mitigated
Moderate (5-10% per year) = 3	3 Acceptable risk	6 Acceptable risk if mitigated	9 Unacceptable risk
High (<10% per year) = 4	4 Acceptable risk if mitigated	8 Unacceptable risk	12 Unacceptable risk

Designation of position shall not be entered on the new medical certificates. If the seafarer's doctor finds that the seafarer may only serve in one specific capacity, or if there are capacities within the categories e.g. navigational watch, safety functions or others in which he cannot serve, this must be entered on the medical certificate as a limitation.

- Which position/capacity does the seafarerhave?
- Which duties does he or she have?
- How is the condition of the workplace?
- Are there several with similar jobs, who could take over if anything happens?
- How important is the time factor? Will the incident have immediate consequences or not?

Next you should find out what will happen in the event of an incident, both with regard to the person himself, to others or to the ship.

This may for instance be:

- fall from mast/in stairs/over guard rails/over board
- injury from dangerous machinery/tools
- self-harm in the event of grave psychiatry
- illness not treated in time
- death
- injury of persons in the event of misactions and misjudgements
- overexertion of others in the event of loss of function
- damage to material goods as a result of misaction and misjudgement
- operation damage as a result of misaction and misjudgement

Situations related to both ordinary duties and emergency duties should be considered. The consequences may be different in two scenarios that are so different.

11.5 FIFTH STEP: CALCULATION OF RISK

We have then reached the point where we can start calculating risk. Risk is the product of likelihood and consequence. Consequence in this regard shall mean consequences for the work situation, not the immediate medical consequence of an underlying medical conditions (e.g. syncope in the event of heart flicker).

Maximum limits for acceptable risks in percentage terms have not been established. This will depend on the type of role and the relevant duties.

For instance, it is more dangerous to be the only navigator on a passenger high speed craft along the Norwegian coast than being a third mate on a supertanker on autopilot in the Pacific Ocean. It is more dangerous to take a ferry in and out of port calls twice an hour than to call at a port once every 14 days. If one is the only engineer on board, that is worse than if there were several, and if you are a module handling operator or crane operator, that is worse than serving in many other capacities.

11.6 SIXTH STEP: MITIGATING MEASURES

In some cases it might be relevant to set conditions, either in the form of limitations or orders, that can mitigate some of the safety risk.

The limitations may be in the form of trade area, capacity or validity. The orders may be related to the use of glasses, hearing aid or medication, order to go to the seafarer's doctor, specialist or general practitioner for checkups at stated times, duty to inform the captain and the company of any changes in the medical condition, requirement for new examination within a certain time, duty to report back to the seafarer's doctor in the case of aggravation of condition, etc.

Sometimes compensating measures may make it justifiable for a person to sail, who without these measures had represented too great of a safety risk.

11.7 SEVENTH STEP: RISK EVALUATION AND CONCLUSION WITH GROUNDS

When you have calculated the risk, if applicable assessed whether compensating measures may reduce the safety risk connected to the seafarer, it is time to evaluate your result with regard to applicable legislation.

Is the seafarer within the safety standards laid down by the Regulations? Is the seafarerwithin the standards if we implement compensating measures, or not?

When the seafarer's doctor has thought through the points above, he/she is ready to conclude and to make a decision.

Conclusions and decisions to issue a medical certificate, limited medical certificate, permanent, temporary or provisional declaration of unfitness are individual decisions pursuant to the Public Administration Act, therefore it is essential that the seafarer's doctor states the basis on which the decision is made.

Both medical and legal grounds shall be given – i.e. the safety risk assessment and referral to the relevant part of the regulations or their appendices.

12 THE APPELLATE BODY

The appellate body is composed of a leader who is a medical practitioner, a representative for the Norwegian Maritime Authority and a representative from one of the three trade unions, the Norwegian Maritime Officers' Association, the Norwegian Union of Marine Engineers and the

Norwegian Seafarers' Union, depending on the job category to which the applicant/complainant concerned belongs.

The competence of the appellate body is set out in sections 13-14 of the Regulations.

12.1 PREPARATION OF CASES FOR THE APPELLATE BODY

The appellate body processes cases in writing based on submitted documentation. The seafarer's doctor has a duty to prepare the case, and to ensure that it is clarified as thoroughly as possible before it is submitted to the appellate body for further consideration.

Cases which are not prepared adequately will be returned requesting complete or partial revision. Practically this will always result in large delays in the case processing, and it is therefore very important that the seafarer's doctor is aware of this responsibility and prepares the case adequately.

During the preparation of a case for the appellate body it is important to remember that the appellate body shall consider the case on an independent basis.

The seafarer's doctor must therefore prepare the basis for the decision in such a way that the appellate body gets an overview of the case which is sufficient for consideration. The seafarer's doctor's conclusion and grounds must be verifiable.

The appellate body has competence as the appealinstance pursuant to the Public Administration Act, and shall try all aspects of the case. This applies to both the administrative and medical aspects of the case.

In exemption cases the appellate body will never grant exemptions from the objects clause in the Regulations or the medical requirements according to international minimum standard (the STCW Convention).

In exemption cases the appellate body shall on an independent basis consider whether it is established that the person – despite not fulfilling the terms according to the list of conditions – may upon individual assessment still be found to satisfy the objects clause of the Regulations. If this clause or the medical requirements according to the international minimum standard as laid down in the STCW convention are not met an exemption will not be made by the appellate body.

12.2 OBTAINING SPECIALIST STATEMENTS – THE SEAFARER'S DOCTOR'S REQUESTING COMPETENCE

Obtaining specialist statements requires a few, simple actions in order to ensure a good response.

Obtaining such information is a part of the duty of the seafarer's doctor as subordinate authority pursuant to section 17 of the Public Administration Act in order to ensure that the case is clarified as thoroughly as possible before any decision is rendered.

The seafarer's doctor cannot expect that hospital specialists within different fields of expertise are familiar with the Regulations concerning the medical examination of persons on board Norwegian ships and mobile offshore units, the health requirements contained therein, what it entails to work at sea, have watch-keeping duties, safety functions, etc. However this knowledge is necessary in order to assess whether the person may be issued a medical certificate.

Furthermore, the specialist may not necessarily be familiar with the selective medical approach and may not be aware that the purpose of the Regulations is the safety of the ship and crew along with ensuring that the individual can perform their duties – not rehabilitation, treatment or facilitation of what the person primarily wants.

Medical commentaries are often of little value. They concern what happened at the hospital when the person was admitted, the status upon discharge, how follow-up, if any, shall take place, when he should be referred back, and similar. It is very rare to find anything regarding the likelihood of becoming acutely ill within a 2 year period, the consequences for himself, for the ship and for the crew if something happens, and one will practically never find any risk analysis.

Therefore if the seafarer's doctor is to obtain answers to the questions that need to be clarified when preparing a case for the appellate body, concrete questions must be asked.

The questions must be adapted to the individual person's situation, diagnostic condition, work on board, and if appropriate; type of vessel, trade area, need for check-ups, consequences if something happens, etc.

Some general advice can nevertheless be given:

- Is the person sufficiently capable of performing his/her routine and emergency duties?
    - Ideally the seafarer's doctor will have a working instructions that can be attached.Alternatively the person him-/herself can describe his/her duties
    - As a minimum requirement the position or type of position should be provided
    - Type of vessel, manning and trade area will also be very useful
- With the condition that the person has - how is the level of function for the various roles with regard to safety-critical duties?
    - E.g.: Concentration, attention, prioritisation, cooperation, reactivity, motivity, balance, sight, hearing, etc.
- What is the likelihood of the person becoming acutely unfit for work during the certificate period?
    - Which incident(s) may occur?
    - What consequences could this have if the person cannot be brought to a hospital?
- If he/she is using medication:
    - How is his/her functional level WITHOUT medication (medication can be lost)
    - How is his/her functional level WITH medication?
    - Are there adverse effects of the medication? Which ones?
    - What will happen in the event of sudden cessation of medical treatment?

- Need for check-ups and follow-up:
  - Does the seafarer has to go to check-ups within a specific time interval?
  - Where can or must the check-ups be carried out?

12.3 DOCUMENTS THAT SHOULD BE ATTACHED TO APPEAL OR APPLICATION FOR EXEMPTION TO THE APPELLATE BODY

The following documents should be attached

a) All cases:
   a. The person's appeal/application for exemption
   b. Copy of the seafarer's doctor's written decision including the basis for the decision which is being appealed, or which shows the seafarer's doctor's decision in exemption cases
   c. Copy of declaration(s) of unfitness or limited medical certificate(s)
   d. Copy of specialist statements, medical commentaries and other medical information relevant to the case
   e. Other information that could clarify the case, if any

b) Appeals:
   a. The seafarer's doctor's assessment which is given as grounds for the decision following receipt of appeal, and report/proposal to the appellate body

c) In case of postponed execution (only applicable to exemption cases):
   a. Copy of the information letter sent to the company or master in order to ask for consent to a postponed execution
   b. Copy of the written consent from the company or master in the event of postponed execution
   c. Copy of the medical certificate

12.4 CHECK LIST FOR LAYOUT OF APPLICATION FOR EXEMPTION OR APPEAL TO THE APPELLATE BODY

The following check list can be used when submitting documentation to the appellate body. Have you remembered all the documents? Have you remembered to comment on all the points mentioned in your own letter?

1. What is the enquiry related to?
   1.1. Appeal
   1.2. Application for exemption
2. Application/appeal from the person
3. Person
   3.1. Last, first and middle name
   3.2. Date of birth and national identity number
   3.3. Position on board
      3.3.1.Which position
      3.3.2.What are the duties
   3.4. Safety function YES/NO

3.4.1.Which function
3.4.2.What are the duties

4. Company
   4.1. Name of company
   4.2. Address, post code and city
5. Ship
   5.1. Name of ship
   5.2. Type of ship
   5.3. Manning of the workplace concerned
6. Trade area
7. Seafarer's doctor
   7.1. Name and address
   7.2. Tel
   7.3. Mobile
   7.4. E-mail address
8. Decision
   8.1. Declaration(s) in question
   8.2. Grounds for the seafarer's doctor's decision
      8.2.1. Legal basis
      8.2.2. Medical basis for the assessment
      8.2.3. Assessment of safety risk
      8.2.4. Recommendation to the appellate body with grounds
9. The seafarer's doctor's reconsideration before transfer to the appellate body with proposal
10. Appendix

13 VISION

13.1 EYESIGHT REQUIREMENTS

13.1.1 EYESIGHT EXAMINATION

- Distance vision shall be tested using the Snellen test type or equivalent. The requirements are set out in the STCW Code, Table A-I/9, see below
- Near vision shall be tested using the reading test type
- Colour vision shall be tested using the Ishihara pseudoisochromatic plates or equivalent.
- Persons who do not pass the Ishihara test may be referred to examination by way of lantern tests
- Lantern testing follows the International Recommendations for Colour Vision Requirements for Transport of the International Commission on Illumination (CIE-143-2001), or subsequent editions[3]
- Contact lenses or glasses may not be worn if their purpose is to improve colour vision.This includes visual aids with red-tinted glass that enhances the contrast between green, yellow and brown tones in such a way that an person with impaired colour vision may pass the Ishihara test
- Visual fields shall initially be tested using the Donders' method. Any indication of limited field of vision shall lead to referral to a clinical vision specialist for more detailed mapping of the visual field defect
- Limitations to night vision may be secondary to specific eye diseases or may follow ophthalmological procedures. Such limitations may also be found when testing low-contrast vision. Specialist assessment should be undertaken if reduced night vision is suspected
- Following refractive eye surgery and other ophthalmological procedures which may potentially impair eyesight, an examination by a specialist shall be carried out when the eyesight is presumed to have stabilised in order to map any occurrence of reduced contrast vision, reduced night vision, halo, stardust or similar effects. This is of the largest importance for persons that perform navigational watch functions

[3] STW 44/WP.3 Annex 7 «Interim Guidance on colour vision testing» recommends that new methods of colour vision testing are not introduced on a permanent basis before new and validated data are available, or, if applicable, revision of CIE 143-2001. (STW subcommittee meeting 29 April-3 Mai 2013).

The eyesight requirements are set out in the STCW Code, Table A-I/9: Minimum in-service eyesight requirements for seafarers on board ship

STCW Convention regulation	Category of seafarer	Distance vision aided[1]		Near/intermediate vision	Colour vision [3]	Visual fields[4]	Night blindness[4]	Diplopia (double vision)[4]
		One eye	Other eye	Both eyes together, aided or unaided				
I/11 II/1 II/2 II/3 II/4 II/5 VII/2	Masters, deck officers and ratings required to undertake look-out duties	0,5[2]	0,5	Vision required for ship's navigation (e.g. chart and nautical publication reference, use of bridge instrumentation and equipment, and identification of aids to navigation)	See Note 6	Normal visual fields	Vision required to perform all necessary functions in darkness without compromise	No significant condition evident
I/11 III/1 III/2 III/4 III/5 III/6 III/7 VII/2	All engineer officers, electro-technical officers, electro-technical ratings and ratings or others forming part of an engine-room watch	0,4[5]	0,4[5]	Vision required to read instruments in close proximity, to operate equipment, and to identify systems/components as necessary	See Noee 7 and No. 4.6.4 in the guide	Sufficient visual fields	Vision required to perform all necessary functions in darkness without compromise	No significant condition evident
I/11 IV/2	GMDSS Radio operators	0,4	0,4	Vision required to read instruments in close proximity, to operate equipment, and to identify systems/components as necessary		Sufficient visual fields	Vision required to perform all necessary functions in darkness without compromise	No significant condition evident

[1] Values given in Snellen decimal notation.
[2] A value of at least 0.7 in one eye is recommended to reduce the risk of undetected underlying eye disease.
[3.] As defined in the "International Recommendations for Colour Vision Requirements for Transport" by the Commission Internationale de l'Eclairage (CIE-143-2001 including any subsequent versions).
[4] Subject to assessment by a clinical vision specialist where indicated by initial examination findings.
[5] Engine department personnel shall have a combined eyesight vision of at least 0.4.
[6] CIE colour vision standard 1 or 2.
7 Based on the STCW Convention Part A-1/9, No. 5, the requirement for colour vision for engineers, electro-technical ratings etc. forming part of an engine-room watch is that their combined vision fulfils the requirements set out in table A-1/9.

13.2 RISKS RELATED TO IMPAIRED VISION

Impaired vision in persons on board ships may constitute a safety risk both when performing routine duties and in emergency situations.

The significance will vary according to the type of position and duties. Impaired vision constitutes the greatest risk for the navigational watch, followed by other safety functions on board where this could be of great significance. It will have the least significance in personnel not forming part of the safe manning.

Under ordinary circumstances the person's vision with visual correction will be decisive. The use of visual correction will be a main rule in emergency situations as well, but situations may arise where glasses or contact lenses are lost or damaged so that they cannot be used. With regard to contact lenses certain types of eye disorders may entail that the use of contact lenses is temporarily not possible.

The loss of lenses or glasses may be compensated for by bringing spare glasses or contact lenses on board. Temporary illness resulting in a temporary inability to use contact lenses can be compensated by the use of glasses during the period in question. Therefore any seaman who requires the use of glasses or contact lenses must be advised to take at least two pairs of glasses on board.

13.3 ENHANCED ASSESSMENT OF HIGH RISK POSITIONS

Several newer navigational aids such as radar, electronic charts, automatic position-fixing with AIS or GPS, monitoring of traffic in certain areas and improved marking of fairways have reduced the need for visual navigation. Despite this, there will still be situations where lookout is necessary and where it is necessary to be able to navigate and manoeuvre visually. Small boats do not always have radar reflectors, objects floating in the fairway may be dangerous to the ship or the radar image may be disrupted by the weather conditions, identification of lights, beacons and lighthouses will always be necessary, and it is always necessary to correctly read the colour coding in the fairway.

Visual acuity is put to the test when navigating high speed craft, especially if the waters are unclean, which you often see along the Norwegian coast. Increased light pollution from land through development and electrification of industrial plants, housing developments and transport makes it more difficult toseparate light signals intended for navigation from other light sources. An example of where you need to be extra vigilant is the position as navigator on passenger high-speed craft along the Norwegian coast.

13.4 REFRACTIVE EYE SURGERY

The use of refractive eye surgery in order to improve vision when using the Snellen's chart at the seafarer's doctor is generally not recommended. Some of these patients will experience problems with blurred vision, halo, stardust or glare, and the problem will be most severe in the

dark, when the pupil expands[4]. This compromises both night vision, contrast vision and normal visual acuity. This will not always be caught during the eye examination, so the person may still constitute a safety risk in the practical service on board, even if he or she passes the examination.

If the person undergoes such refractive eye surgery, visual acuity, contrast vision and night vision must be examined by a specialist following the surgery, and must be found satisfactory before a medical certificate can be issued.

Complications after laser refractive surgery

PRK/LASIK/LASEK	More common in PRK	More common in LASIK	More common in LASEK
Overcorrection/ undercorrection	Postoperative pain	Flap complications	Postoperative pain
Astigmatism	Delayed epithelial healing	Epithelial ingrowth	Stromal haze
Regression	Infection	Diffuse lamellar keratitis (Sands of Sahara)	Flap complications
Dry eye symptoms	Scarring/stromal haze		
Reduced contrast sensitivity			

PRK: phtorefractive keratectomy. LASIK: laser-assisted in situ keratomileusis. LASEK: Laser epithelial keratomileusis.

Source: UpToDate accessed 7th July 2014

13.4.1 EFFECT OF REFRACTIVE EYE SURGERY

Persons undergoing refractive eye surgery have a 90 - 99% chance of achieving 5/10 or better unaided vision[5] [6] and 57 - 79% achieve 5/5 or better unaided vision[7] [8] [9] [10]. The result of each study is based on each patient's refraction and the degree of astigmatism. Around 85% of the patients have 4/5 or better unaided vision, which makes them capable of functioning without corrective visual aids in daily life. Patient satisfaction following LASIK is usually high[11].

[4] McDonald MB, Carr JD, Frantz JM, Kozarsky AM, Maguen E, Nesburn AB, Rabinowitz YS, Salz JJ, Stulting RD, Thompson KP, Waring GO 3rd: Laser in situ keratomileusis for myopia up to -11 diopters with up to -5 diopters of astigmatism with the summit autonomous LADARVision excimer laser system. Ophthalmology. 2001;108(2):309.

[5] McDonald MB, Carr JD, Frantz JM, Kozarsky AM, Maguen E, Nesburn AB, Rabinowitz YS, Salz JJ, Stulting RD, Thompson KP, Waring GO 3rd. Laser in situ keratomileusis for myopia up to -11 diopters with up to -5 diopters of astigmatism with the summit autonomous LADARVision excimer laser system. Ophthalmology. 2001;108(2):309.

[6] el Danasoury MA, el Maghraby A, Klyce SD, Mehrez K. Comparison of photorefractive keratectomy with excimer laser in situ keratomileusis in correcting low myopia (from -2.00 to -5.50 diopters). A randomized study. Ophthalmology. 1999;106(2):411.

[7] El-Maghraby A, Salah T, Waring GO 3rd, Klyce S, Ibrahim O. Randomized bilateral comparison of excimer laser in situ keratomileusis and photorefractive keratectomy for 2.50 to 8.00 diopters of myopia. Ophthalmology. 1999;106(3):447.

[8] Kawesch GM, Kezirian GM. Laser in situ keratomileusis for high myopia with the VISX star laser. Ophthalmology. 2000;107(4):653

[9] Linebarger EJ, Hardten DR, Lindstrom RL: Diffuse lamellar keratitis: diagnosis and management. J Cataract Refract Surg. 2000;26(7):1072.

[10] Sakimoto T, Rosenblatt MI, Azar DT. Laser eye surgery for refractive errors. Lancet. 2006;367(9520):1432.

[11] Bailey MD, Mitchell GL, Dhaliwal DK, Boxer Wachler BS, Zadnik K. Patient satisfaction and visual symptoms after laser in situ keratomileusis. Ophthalmology. 2003;110(7):1371.

The degree of myopia, myopia versus hyperopia and occurrence of astigmatism are factors influencing the result following refractive surgery. Patients having low myopia without astigmatism have the best results, whereas patients having hyperopia with astigmatism have the most unpredictable results[12] [13] [14] [15] [16] [17] [18] [19].

A few other factors can also influence the result:

- Type of procedure used (PRK, LASIK, LASEK)
- The surgeon's skills and experience
- Quality control and maintenance of surgical equipment is crucial for precise ablation
- The type of laser will also have an effect. Newer generations of small flying spot laser beams (<100 microns) with eye tracking systems theoretically gives better results than older beams (4-5 mm) without eye tracking system[20]. Larger randomised studies are not available.

Short and Allan compared the results from six randomised studies (417 eyes) between PRK and LASIK used to correct myopia[21]. The vision stabilised quicker with LASIK, but the accuracy of the surgery was about the same. Six months after treatment with LASIK, there was a non-significantly larger number of treated eyes achieving unaided vision of 5/5 or better. There are no studies comparing LASIK to PRK with regard to hyperopia[22]. It is still uncertain whether the long term effects of PRK are different to those of LASIK.

12 El-Maghraby A, Salah T, Waring GO 3rd, Klyce S, Ibrahim O. Randomized bilateral comparison of excimer laser in situ keratomileusis and photorefractive keratectomy for 2.50 to 8.00 diopters of myopia. Ophthalmology. 1999;106(3):447

13 Dulaney DD, Barnet RW, Perkins SA, Kezirian GM. Laser in situ keratomileusis for myopia and astigmatism: 6 month results. J Cataract Refract Surg. 1998;24(6):758.

14 Buzard KA, Fundingsland BR: Excimer laser assisted in situ keratomileusis for hyperopia. J Cataract Refract Surg. 1999;25(2):197.

15 Yoo SH, Azar DT. Laser in situ keratomileusis for the treatment of myopia. Int Ophthalmol Clin. 1999;39(1):37.

16 Salah T, Waring GO 3rd, el Maghraby A, Moadel K, Grimm SB. Excimer laser in situ keratomileusis under a corneal flap for myopia of 2 to 20 diopters. Am J Ophthalmol. 1996;121(2):143.

17 Zadok D, Maskaleris G, Montes M, Shah S, Garcia V, Chayet A. Hyperopic laser in situ keratomileusis with the Nidek EC-5000 excimer laser. Ophthalmology. 2000;107(6):1132.

18 Corones F, Gobbi PG, Vigo L, Brancato R. Photorefractive keratectomy for hyperopia: long-term nonlinear and vector analysis of refractive outcome. Ophthalmology. 1999;106(10):1976.

19 Tabbara KF, El-Sheikh HF, Islam SM. Laser in situ keratomileusis for the correction of hyperopia from +0.50 to +11.50 diopters with the Keracor 117C laser. J Refract Surg. 2001;17(2):123.

20 McDonald MB, Deitz MR, Frantz JM, Kraff MC, Krueger RR, Salz JJ, Kraff CR, Maguen E, Matta CS, Nesburn AB, Piebenga LW. Photorefractive keratectomy for low-to-moderate myopia and astigmatism with a small-beam, tracker-directed excimer laser. Ophthalmology. 1999;106(8):1481.

21 Shortt AJ, Allan BD. Photorefractive keratectomy (PRK) versus laser-assisted in-situ keratomileusis (LASIK) for myopia. Cochrane Database Syst Rev. 2006;

22 Settas G, Settas C, Minos E, Yeung IY: Photorefractive keratectomy (PRK) versus laser assisted in situ keratomileusis (LASIK) for hyperopia correction. Cochrane Database Syst Rev. 2012;6:CD007112.

The results after 10 years are published in several studies. The results for PRK and LASIK for mild myopia are published by Alió et al for both low and high myopia[23] [24] [25] [26]. The studies include a total of 785 eyes, and show stable refraction and generally successful results. Myopic regression mostly occurs during the first two years following surgical treatment, and more rarely thereafter. New treatment was performed in 20-45% of the patients, usually more than two years after initial treatment and well tolerated.

In another study of 779 eyes in 402 individuals and a follow-up over five years following a LASIK procedure, the best corrected vision remained unchanged compared to one month post-surgery in 98% of the patients. 17.5% of the patients had undergone new surgery on average 2.5 years after the initial procedure[27].

Patient satisfaction following LASIK is generally high compared to other selective surgical procedures. In a systematic review of 19 articles on patients' quality of life and satisfaction following LASIK treatment, the average patient satisfaction with the surgical result was 95.4%[28].

13.5 PERSONS NOT COVERED BY THE STCW CONVENTION

All persons on board ships should achieve the minimum eyesight standard of 0.1 unaided in each eye (STCW Code, section B-I/9, paragraph 10). This standard may also be relevant to other seafarers to ensure visual capability under emergency conditions when visual correction may be lost or damaged.

Seafarers not covered by the STCW Convention's eyesight standards should have vision sufficient to perform their routine and emergency duties safely and effectively.

13.6 SHORT-SIGHTEDNESS / MYOPIA

Persons with severe short-sightedness have an increased risk of retinal detachment. Approximately 55% of persons with spontaneous retinal detachment experience this due to severe short-sightedness. When the myopia is -1 to -3 diopters, the risk is quadrupled. When the

[23] AlióJL, Muftuoglu O, Ortiz D, Artola A, Pérez-Santonja JJ, de Luna GC, Abu-Mustafa SK, Garcia MJ. Ten-year follow-up of photorefractive keratectomy for myopia of less than -6 diopters. Am J Ophthalmol. 2008;145(1):29.
[24] AlióJL, Muftuoglu O, Ortiz D, Pérez-Santonja JJ, Artola A, Ayala MJ, Garcia MJ, de Luna GC. Ten-year follow-up of laser in situ keratomileusis for myopia of up to -10 diopters. Am J Ophthalmol. 2008;145(1):46.
[25] AlióJL, Muftuoglu O, Ortiz D, Artola A, Pérez-Santonja JJ, de Luna GC, Abu-Mustafa SK, Garcia MJ. Ten-year follow-up of photorefractive keratectomy for myopia of more than -6 diopters. Am J Ophthalmol. 2008;145(1):37.
[26] AlióJL, Muftuoglu O, Ortiz D, Pérez-Santonja JJ, Artola A, Ayala MJ, Garcia MJ, de Luna GC. Ten-year follow-up of laser in situ keratomileusis for high myopia. Am J Ophthalmol. 2008;145(1):55.
[27] Kato N, Toda I, Hori-Komai Y, Sakai C, Tsubota K: Five-year outcome of LASIK for myopia. Ophthalmology. 2008;115(5):839.
[28] Solomon KD, Fernández de Castro LE, Sandoval HP, Biber JM, Groat B, Neff KD, Ying MS, French JW, Donnenfeld ED, Lindstrom RL, Joint LASIK Study Task Force: LASIK world literature review: quality of life and patient satisfaction. Ophthalmology. 2009;116(4):691.

myopia is more severe than -3, the risk is tenfold[29]. The general risk of retinal detachment, however, is quite low, around 1:10,000 per year[30] [31], so that in any case the risk is low.

13.7 COLOUR VISION

Reduced colour vision is a safety risk for persons on board ships, especially with navigational and watch functions. This risk consideration is essentially taken from the International Recommendations for Colour Vision Requirements for Transport (CIE 143-2001) from the International Commission on Illumination.

Reduced colour vision leads to the reduced ability to identify signal colours[32].

People with impaired colour vision also react slower when responding to signal lights[33] [34], and when they are reacting to colour coded computer screens[35].

Between ¼ and 1/3 of people with defective colour vision report difficulties in separating road traffic lights from street lighting. A smaller percentage have problems seeing brake lights. Persons with more severe colour vision deficiency have more difficulty with road traffic lights – up to 50% report such problems[36].

Many people with colour vision deficiency compensate by reading the context of the colour (position of traffic lights, the sharpness of the light, reactions of other road users). When such supporting factors are absent, these people can experience more difficulty.

Task or activity	% with difficulty
DRIVING	
Separating colours in traffic signal	29
Mixing street lights and traffic lights	26
Difficulty seeing brake lights	13
Difficulty seeing reading marking of roads	9
WORK	
Colour vision deficiency has affected career choice	34
Experienced difficulty due to reduced colour vision in current job	25
Difficulties due to reduced colour vision in previous job	23
ACTIVITIES IN EVERYDAY LIFE	
Choosing coloured products (clothes, paint, interior, cosmetics)	74
Work with craft or hobby (colour coded cable, thread, wool, paint, etc.)	39

[29] [Risk factors for idiopathic rhegmatogenous retinal detachment. The Eye Disease Case-Control Study Group. Am J Epidemiol. 1993;137(7):749. (PMID: 8484366) – Author not listed in the article.]
[30] Wilkes SR, Beard CM, Kurland LT, Robertson DM, O'Fallon WM.The incidence of retinal detachment in Rochester, Minnesota, 1970-1978. Am J Ophthalmol. 1982;94(5):670
[31] Haimann MH, Burton TC, Brown CK Epidemiology of retinal detachment. Arch Ophthalmol. 1982;100(2):289
[32] Vingrys and Cole, 1988
[33] Nathan et al, 1964
[34] Cole and Brown, 1966
[35] Cole and Macdonald, 1988
[36] Vingrys and Cole, 1989

Even through radar and satellite navigation has reduced the dependence of signal lights at sea, it is still essential for nautical personnel to be able to recognise navigation lights, port lights, beacons and lighthouses in order to navigate safely. The most important colours are red, green, white and sometimes yellow.

These signals must sometimes be observed from afar, and must be able to be observed under difficult conditions of visibility. When navigating along the coast and on rivers and lakes, interfering lights from the shore will make observation more difficult.

Fairway marking for daylight is also colour-coded, but it has also a significant shape which helps to interpret the markings. The colours used are green, yellow, orange, red and black.

Coloured flags are still used for ship-to ship and ship-to-shore communication.

Colour-coding is used extensively for piping and cables on board to indicate function.

The introduction of modern technology for navigation and ship control has increased the complexity of the visual presentation on computer screens both on the navigating bridge and in the engine-room. The computer screen often uses colour-coding to organise and separate or define complex information and to indicate operational status and to present alarms. Colour-coding on screens has been shown to increase the accuracy and speed of the decision-making[37] and defective colour vision has been shown to reduce the same qualities[38].

13.7.1 TYPES OF COLOUR VISION DEFICIENCY / COLOUR BLINDNESS

13.7.1.1 COLOUR VISION DEFICIENCY (ANOMALOUS TRICHROMACY / TRICHROMATICISM)

Protanomaly – red-weakness, 1% of all male. Deuteranomaly – green-weakness, 5% of all male.

13.7.1.2 COLOUR BLINDNESS (DICHROMACY)

Protanopia – red-blindness, 1% of all male.
Deuteranopia – green-blindness, 1% of all male.
Tritanopia – blue-blindness, rare.

Scholz et al[39] found that approximately 1/3 of anomalous trichromats and over half of dichromats could not find the target colours on a colour-coded sonar screen during 640 seconds of observation time, a task which 98% of those with normal vision managed in the assigned time.

[37] Christ, 1975; Macdonald and Cole 1988
[38] Cole and Macdonald, 1988
[39] Scholz et al [1995]

The STCW 2012 provides that CIE 143-2001 shall be the standard for colour vision testing for seafarers. This is also included in the ILO-IMO International guidelines for medical examinations of seafarers. According to CIE 143-2001 colour vision / colour vision deficiency / colour blindness is classified in different classes.

13.7.2 RECOMMENDED STANDARDS IN SHIPPING

Standard 1 shall be used for masters, deck officers and ratings required to undertake look-out duties on board ships of more than 500 tonnes.

Standard 2 shall be used for masters, deck officers and ratings required to undertake look-out duties on board ships of less than 500 tonnes approved for commercial traffic.

Standard 3 shall be used for engineer officers and engine department personnel, radio personnel and electro-technical officers/ratings on board ships of more than 500 tonnes and with propulsion power of more than 750 kW.

13.7.3 STANDARDS FOR COLOUR VISION RECOMMENDED BY CIE (COMMISSION INTERNATIONAL DE L'ECLAIRAGE).

1. CIE COLOUR VISION STANDARD 1 (Normal colour vision)
    a. Normal colour vision
    b. Passes the Ishihara Test for Color Blindness (38 pl)
        i. Errors on three or more charts is "fail"
        ii. When in doubt:
            1. Test with another PIC (see below)
            2. Anomaloscope
                a. Fail: MMP > ± 2 SD from population mean, or > 3 SD from normal mean.
            3. Lantern test (e.g. Holmes Wright Type B)
                a. Fail: Two or more errors naming signal colours on two runs (sequences) of nine pairs of colours.
    c. Another pseudoisochromatic (PIC) plate may be used as an alternative:
        i. American Optical Company Pseudo-isochromatic plates
        ii. Boström-Kugelberg Plates for Testing Colour Vision (Tabulae Pseudo-isochromaticae)
        iii. Dvorine Pseudo-isochromatic Plates
        iv. Standard Pseudo-isochromatic plates (SPP)
        v. The Hahn New Pseudoisochromatic colour vision test
        vi. American Optical Hardy-Rand-Rittler (AOHRR)

2. CIE COLOUR VISION STANDARD 2 (DEFECTIVE COLOUR VISION A)
    a. Mild colour vision deficiency, but able to identify signal colours correctly.
        i. If patient fails Ishihara test or other PIC, continue with:
        ii. Lantern test (Fail: 2 or more error on two runs of 9 pairs), such as:

1) Holmes Wright type A
2) Farnsworth
3) Optec 900
4) Beyne

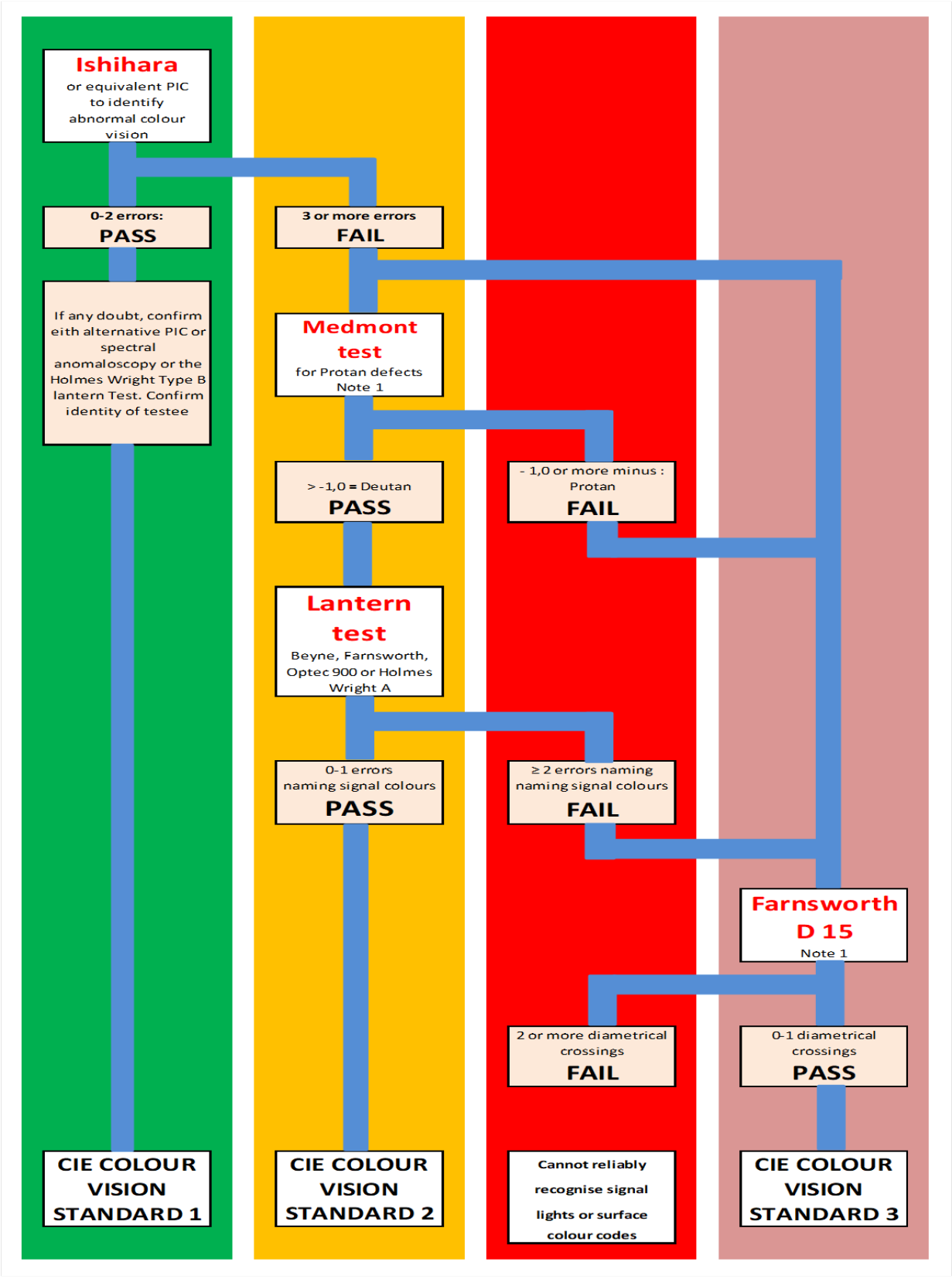
And a test for protan defective colour vision, such as:

5) Medmont C 100 or equivalent test
   - Fail: protan settings of -2.0 or more minus, on average, for three settings.
6) Anomaloscopy by an expert.
   - A protan diagnosis is indicated by an excess of red in a red plus green mixture of colours to match yellow and a significantly lower than normal yellow luminance setting.

3. CIE COLOUR VISION STANDARD 3 (DEFECTIVE COLOUR VISION B)
   a. Colour vision deficiency, but able to recognise signal colours correctly at short distances.
      i. If patient fails Ishihara test or other PIC, continue with:
         1. Farnsworth Dichotomous test panel D15 (Farnsworth D15) (Fail: 2 or more diametric crossings in the plot)
            or
         2. Anomaloscopy by an expert.

13.7.4 ALGORITHM FOR COLOUR VISION TESTING

The following algorithm could be used, and serve as a short overview of the testing procedure:



13.7.5 COLOUR VISION REQUIREMENTS FOR PERSONS FORMING PART OF AN ENGINE-ROOM WATCH[40]

There are different requirements for colour vision for different positions on board.

In accordance with the STCW Convention Part A-1/9 fifth paragraph, final sentence, the Norwegian Maritime Authority have decided as follows with regard to colour vision requirements for the above mentioned occupational groups[41]:

If a person as mentioned above satisfies all other requirement for vision in table A-I/9, it may be permitted that this person has poorer colour vision than provided in CIE standard 3. The person shall therefore be considered as fulfilling the colour vision requirements for serving in such a capacity and the point regarding colour vision on the medical certificate shall be filled out as satisfied.

If the person does not satisfy the other eyesight requirement following table A-I/9 while at the same time not fulfilling the colour vision requirements in CIE Standard 3, he or she does not fulfil the colour vision requirements for the position. The point regarding colour vision on the medical certificate shall be filled out as not satisfied.

Norway has made this interpretation of the STCW Convention in order to avoid that persons currently working in the engine-room are put ashore as a result of the new requirements for colour vision in the STCW Convention. Our experience is that there is no medical or safety-related justification for the requirement for colour vision in accordance with the CIE standard 3 for working in the engine-room. The current situation is that persons with defective colour vision are overrepresented within the concerned occupational groups, since several who initially applied to the navigator programme in maritime schools have switched to a mechanical and electro-engineering programme if they were found to have defective colour vision.

As there are no safety-related grounds for the stricter requirement in the STCW Code, we as flag State choose to take advantage of the flexibility set up by the STCW Convention in Part A-1/9 fifth paragraph.

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40 If the rating also forms part of a navigational watch, these exemption clauses do not apply to him/her.

41 Norway has made this interpretation of the STCW Convention in order to avoid that persons currently working in the engine-room are put ashore as a result of the new requirements for colour vision in the STCW Convention. Our experience is that there is no medical or safety-related justification for the requirement for colour vision in accordance with the CIE standard 3 for working in the engine-room. The current situation is that persons with defective colour vision are overrepresented within the concerned occupational groups, since several who initially applied to the navigator programme in maritime schools have switched to a mechanical and electro-engineering programme if they are found to have defective colour vision.
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In these cases it will consequently be correct to check the engineer fulfils the requirements for colour vision even if he or she does not pass the CIE standard 3 test.

In these cases it will consequently be correct to check the engineer fulfils the requirements for colour vision even if he/she does not pass the CIE standard 3 test.

He/she will not be fit for forming part of a navigational watch, and this shall be specified by the medical certificate.

14 HEARING

14.1 THE IMPORTANCE OF HEARING ON BOARD

14.1.1 SPEECH COMMUNICATION

Adequate hearing is necessary for verbal communication, either directly or via radio/telephone. Background noise can often interfere with the communication, and linguistic barriers may put additional demands on perception. Misinterpretation of speech communication may be critical for safety.

14.1.2 ALARMS

Acoustic signals are often used as alarms, and it is of course important that these are heard. Persons using hearing aids normally sleep without these, and compensating mechanisms must therefore be established in order to ensure that a safety-critical warning will also be heard by persons with hearing loss when they are sleeping.

14.1.3 DIFFERENCE BETWEEN MAN-TO-MAN SPEECH AND TELEPHONE/RADIO

Speech perception is generally better in direct conversation than in radio/telephone communication, since additional information may be deduced from body language, facial expressions, reading of lips, better situational understanding and similar.

14.2 HEARING REQUIREMENTS

14.2.1 THE HEARING REQUIREMENTS ARE PROVIDED IN THE STCW CONVENTION

Hearing requirements for seafarers in a position for which a certificate of competency is required pursuant to the STCW Convention

Frequency	500 Hz	1000 Hz	2000 Hz	3000 Hz
Best ear	Average hearing capacity at least 30 dB			
Weakest ear	Average hearing capacity at least 40 dB			

The hearing requirements are equivalent to hearing whispered speech at distances of 3 metres and 2 metres, respectively.

14.2.2 HEARING REQUIREMENTS FOR SEAFARERS NOT IN A POSITION FOR WHICH A CERTIFICATE OF COMPETENCY IS REQUIRED PURSUANT TO THE STCW CONVENTION

Seafarers performing duties not covered by the STCW Convention shall have satisfactory social hearing.

14.3 THE BASICS ON HEARING LOSS

Speech recognition is to a large extent dependent on the relation between signal and noise (Signal-Noise Ratio = SNR), but there is limited knowledge on which degree of hearing loss will entail failing to understand the spoken word. The frequencies 250-5,000 Hz are the important frequencies for recognition of speech, intermediate frequencies and treble are crucial for being able to distinguish what is being said. For many people who are hard of hearing, the hearing of unvoiced consonants and sibilants will in particular be affected. Speech sounds such as p/t/k, sh/ch, f/s/z/th are not heard or are easily confused. The same may apply to n/m/ng as well as b/d/g. In most people the hearing loss occurs in the frequency range 1,000-8,000 Hz. This is the range which is the most significant for the speech recognition, because this is where consonants and consonant combinations are perceived.

14.4 USE OF A HEARING AID

Even though the use of a hearing aid can compensate for loss of hearing to a certain degree, the person concerned will not get normal hearing when using such aids. The discrimination is still difficult, particularly when there is background noise. It is therefore not automatic that a person using a hearing aid has compensated for the loss of hearing to such a degree that the hearing requirements for issuance of a medical certificate are satisfied. Tinnitus may sometimes also complicate the issue.

14.4.1 TESTING OF PERSONS WITH A HEARING AID

It is necessary to test persons with a hearing aid in situations similar to those on board. It is not possible to test hearing with hearing aid by pure tone audiometer. Speech audiometry is used, and this must be performed with different types of background noise (conversational noise, wind noise, machinery noise). Satisfactory speech recognition must be achieved by speech audiometry under such conditions.

When performing speech audiometry, the speech recognition threshold (SRT) and word discrimination score are assessed. SRT is the lowest level at which the test person can correctly repeat 50% of the words in the test (the SNR which gives 50% correct answers). SRT is measured in decibel and is usually equal to the average pure-tone audiometry threshold ± 6 dB. The average threshold in pure-tone audiometry is the average of the frequencies 500, 1000 and 2000 Hz.

The speech discrimination score is the percentage of words that can be repeated directly at a given threshold, e.g. 40 dB over SRT. A poor word discrimination score may indicate that the hearing-loss is neurogenic, and that the effect of a hearing aid will be / is small, since sound

amplification does not increase speech recognition. This should be tested with varying degrees/types of background noise.

It has not been decided which limits should be set for speech audiometry in order to ensure hearing aids compensate sufficiently for the safety risk related to the hearing loss asto be acceptable. In these cases you need to rely on the ENT doctor's assessment.

14.5 SINGLE-SIDED HEARING

Persons with unilateral hearing loss may have difficulty in understanding communication from the hearing-impaired side, difficulty in localising sound and difficulty in understanding speech in the presence of background noise. In quiet conditions with little or no noise, speech discrimination is approximately the same as for persons with normal hearing without monaural hearing loss.

15 PHYSICAL CAPABILITY REQUIREMENTS

15.1 INTRODUCTION

The physical capability requirements for work at sea vary widely and have to take account of both routine and emergency duties. This requires sufficient physical ability in the following areas:

- strength;
- stamina;
- flexibility;
- balance and coordination;
- size – compatible with work in confined areas and moving through restricted openings;
- exercise capacity – heart and respiratory reserve; and
- fitness for specific tasks, such as being able to carry breathing apparatus formembers of the fire party.

Skills and related physical ability is described in STCW Code B – Table B-I/9. The Norwegian regulatory requirements are based on this table, which gives a good indication of the functions that seafarers must master.

Table B-I/9
Assessment of minimum entry level and in-service physical abilities for seafarers[3]

Shipboard task, function, event or condition[3]	Related physical ability	A medical examiner should be satisfied that the candidate[4]
Routine movement around vessel On moving deck Between levels Between compartments Note 1 applies to this row	Maintain balance and move with agility Climb up and down vertical ladders and stairways Step over coamings (e.g. Load Line Convention requires coamings to be 600 mm high) Open and close watertight doors	Has no disturbance in sense of balance Does not have any impairment or disease that prevents relevant movements and physical activities Is, without assistance[5], able to: Climb vertical ladders and stairways Step over high sills Manipulate door closing systems
Routine tasks on board Use of hand tools	Strength, dexterity and stamina to manipulate mechanical devices	Does not have a defined impairment or diagnosed medical

Movement of ship's stores Overhead work Valve operation Standing a for-hour watch Working in confined spaces Responding to alarms, warnings and instructions Verbal communication Note 1 applies to this row	Lift, pull and carry a load (e.g., 18 kg) Reach upwards Stand, walk and remain alert for an extended period Work in constricted spaces and move through restricted openings (e.g. SOLAS regulation II-I/3-6.5.1 requires openings in cargo spaces and emergency escapes to have the minimum dimensions of 600 mm x 600 mm. Visually distinguish objects, shapes and signals Hear warnings and instructions Give a clear spoken description	condition that reduces ability dto perform routine duties essential to the safe operation of the vessel Has ability to: Work with arms raised Stand and walk for an extended period Enter confined space Fulfil eyesight standards (table A-I/9) Fulfil hearing standards set by competent authority or take account of international guidelines Hold normal conversation
Emergency duties[6] on board Escape Fire fighting Evacuation Note 2 applies to this row	Put on a lifejacket or immersion suit Escape from smoke-filled spaces Take part in fire-fighting duties, including use of breathing apparatus Take part in vessel evacuation procedures	Does not have a defined impairment or diagnosed medical condition that reduces ability to perform emergency duties essential to the safe operation of the vessel Has ability to: Don lifejacket or immersion suit Crawl Feel for differences in temperature Handle fire-fighting equipment Wear breathing apparatus (where required as part of duties)

Notes:

[1] Rows 1 and 2 of the above table describe (a) ordinary shipboard tasks, functions, events and conditions, (b) the corresponding physical abilities which may be considered necessary for the safety of a seafarer, other crew members and the ship, and (c) high-level criteria for use by medical practitioners assessing medical fitness, bearing in mind the different duties of seafarers and the nature of shipboard work for which they will be employed.

[2] Row 3 of the above table describes (a) ordinary shipboard tasks, functions, events and conditions, (b) corresponding physical abilities which should be considered necessary for the safety of a seafarer, other crew members and the ship, and (c) high-level criteria for use by medical practitioners assessing medical fitness, bearing in mind the different duties of seafarers and the nature of shipboard work for which they will be employed.

[3] This table is not intended to address all possible shipboard conditions or potentially disqualifying medical conditions. Parties should specify physical abilities applicable to the category of seafarers (such as "Deck Officer" and "Engine rating"). The special circumstances or individuals and for those who have specialized or limited duties should receive due consideration.

[4] If in doubt, the medical practitioner should quantify the degree or severity of any relevant impairment by means of objective testes,m whenever appropriate tests are available, or by referring the candidate for further assessment.

[5] The term "assistance" means the use of another person to accomplish the task.

[6] The term emergency duties is used to cover all standard emergency response situations such as abandon ship or firefighting as well as the procedures to be followed by each seafarer to secure personal survival.

15.2 THE SEAFARER'S DOCTOR'S ROLE IN THE FUNCTIONAL ASSESSMENT

The seafarer's doctor is responsible for testing physical capability when there is an indication for it. He may alternatively use an assistant for the actual test procedure, or have an agreement with a training centre or a physical therapist who can perform the actual testing.

Conditions which may entail loss of physical capability must lead to testing being undertaken, see below point on medical conditions and physical capability.

15.3 MEDICAL CONDITIONS AND PHYSICAL CAPABILITY

Limitations in physical capability may arise from a range of medical conditions, such as:

- high or low body mass / obesity;
- severely reduced muscle mass;
- musculoskeletal disease, pain or limitations to movement;
- a condition following an injury or surgery;
- lung disease;
- heart and blood vessel disease; and
- neurological diseases.

15.4 PHYSICAL CAPABILITY ASSESSMENT

Physical capability testing shall be undertaken when there is an indication for it, for instance because of the presence of one of the above conditions or because of other concerns about an person's physical abilities.

The aspects that are tested will depend on the reasons fortesting.

Table B-I/9 in the STCW Convention gives recommendations for physical abilities to be assessed for the various functions. See above.

The below recommendation shows approaches that may be used to assess whether the requirements are met.

- Observed ability to perform routine and emergency duties safely and effectively.
- Tasks that simulate normal and emergency duties.
- Assessment of cardiorespiratory reserve, including spirometry and ergometric tests. This will predict maximum exercise capacity and hence indirectly the seafarer's ability to perform physically demanding work. A large reserve will also indicate that heart and lung performance is less likely to be compromised throughout the period of validity of the medical certificate. The benchmark test is measurement of maximum oxygen uptake

($VO_{2\ max}$)[42], but this requires dedicated equipment. Step tests such as the Chester[43] [44] or the Harvard[45] are simpler alternatives that may be used for screening. If step tests are abnormal, they should be further validated by $VO_{2\ max}$ or treadmill stress tests.

- Informal testing of cardiorespiratory reserve could be performed, for instance climbing stairs (three to six flights of stairs) and assessing any distress, shortness of breath and similar, plus the speed of pulse rate decline on stopping. This is not readily reproducible between practioners but can be used for repeat assessment by the same medical practitioner.
- Clinical assessment of strength, mobility, coordination, etc.

Additional information may come from activities recently or regularly undertaken, as described by the seafarer, such as:

- physically demanding duties on the vessel (carrying heavy items, handling mooring equipment, etc.);
- attendance at a physically demanding course, e.g.fire fighting, helicopter escape, STCW basic training or similar; and
- a confirmed personal pattern of regular exercise.
- When a case is submitted for consideration by the Appelate Body, it is important that the testing is done in an objective way, making it possible for the Appelate Body to assess the case independently.

15.5 INTERPRETATION OF RESULTS

- Is there any evidence that the person is not able to perform his or her routine and emergency duties safely and effectively?
- Are there any observed limitations to strength, flexibility, stamina or coordination?
- What is the outcome of any test for cardiorespiratory reserve?
    - Test performance limited by shortness of breath, musculoskeletal or other pain, or exhaustion. Causes need to be investigated and taken into account in determining physical capability.
    - Unable to complete the test.
    - Completed but stressed (cardiovascular or respiratory) or with poor recovery after stopping.
    - Completed to good or average standard.

  Discuss subjective feelings during the test with the person and also go over experiences of fitness and capability when doing normal tasks and emergency drills (e.g. man-

[42] Hem Erlend and Leirstein Svein: Testing av utholdenhet. http://olympiatoppen.no/fag/utholdenhet/testlaboratoriet/tester/media3223.media
[43] Sykes K, Roberts A: The Chester step test – a simple yet effective tool for the prediction of aerobic capacity. Physioterapy 90 (2004) 183-188
[44] Watkins J: Step tests of cardiorespiratory fitness suitable for mass testing. Br J. Sports Med 1984 June; 18(2): 84-89
[45] Ryhming I. A modified Harvard step test for the evaluation of physical fitness. Arbeitsphysiologie. 1953;15(3):235–250.

overboard drills or lifeboat drills). Obtain corroboration from others if performance at work is uncertain.

15.6 DECISION-MAKING

Information from a range of sources may be required and many of these are not easily accessed in the course of a medical examination.

- Is there any indication that physical capability may be limited? (stiffness, obesity, history of heart disease, etc.)
  - If NO – no test necessary.
  - If YES – consider which tests or observations should be carried out in order to determine the person's capability to perform their duties.
- Do the test results indicate that capabilities may be limited?
  - NO – provided there are no underlying conditions that affect the conduct of the assessment. →Unrestricted medical certificate
  - YES – but duties can be modified so that the person can work in a safe and effective way, without putting excess responsibilities on others. → Limited medical certificate
  - YES – but the cause of limitation can be remedied. Currently incompatible with reliable performance of essential duties safely and effectively. → Declaration of temporary unfitness
  - YES – and cause of limitation cannot be remedied nor duties modified. Incompatible with reliable performance of essential duties safely and effectively. → Declaration of permanent unfitness

16 USE OF MEDICATION

16.1 INTRODUCTION

Medication can play an important part in enabling seafarers to continue to work at sea. However some have side effects that can affect safe and effective performance of duties and some have other complications that may increase the likelihood of illness at sea.

The paragraph on use of medication is only concerned with the use of continuing prescribed medication.

The use of oral medication at sea may be prevented by nausea and vomiting, and illness may arise if the medication is no longer taken and therefore does not provide protection (epilepsy, hormones, etc.).

The seafarer's doctor will need to assess the known adverse effects of each medication used and the individual's reaction to it.

If medication is clinically essential for the effective control of a condition, e.g. insulin, anticoagulants and psychopharmaceuticals, it is dangerous to stop it in an attempt to be fit for work at sea.

16.2 RESTRICTED MEDICINES - AND OTHER MEDICINES

Medicines in the Norwegian prescription group A (narcotics and CNS stimulants) and B (tranquillizers and other medicines with an addictive effect) are no longer specifically mentioned in the regulations. This does not, however, imply that these medicines should not be judged as seriously as earlier. On the contrary – it implies that also other medicines could have unwanted effects: drowsiness, reduced alertness, reduced concentration abilities, mood changes – just tomention a few.

The seafarer's doctor shall assess the safety risk connected to regular medication in ALL CASES, whether or not the medicines are used daily or on demand whilst serving on board ship.

I all such cases the person shall carry a certificate from the seafarer's doctor that these medicines are assessed and that the person is allowed to us them whilst in service.

The risk assessment for medical conditions also includes the use of medicines.

16.3 RISK ASSESSMENT IN PERSONS USING MEDICATION

It is not unusual to see that the seafarer's doctor focuses more on the medication use than on the condition for which the medication is used. For example they focus more on the use of methylphenidate than the condition ADHD, focus more on the use of antidepressants than on the underlying depression, etc.

When persons use medication, three factors should be assessed:

1) Functional ability in areas significant for safety WITHOUT medication
    a. Illustrates the necessity of using medication
    b. What you must be prepared for if you lose the medication during service on board
2) Functional ability in areas significant for safety WITH medication
    a. Illustrates the necessity of using medication
3) The medication in use, including
    a. Adverse effects
    b. Risk of complication
    c. Risk of abuse
    d. Storage and distribution on board
    e. Refilling
    f. Legislation in other countries (including customs regulations and border crossings) and how this is enforced.
        i. The medication may be prohibited in some places.
        ii. Possession of the medication may in some places be considered violation of the law.

Functions that could easily be affected in a safety-critical way when using medication are, for example,the ability to concentrate, alertness and vigilance, ability to react, acts of impulse, emotional state, etc.

16.4 ISSUE OF DECLARATION REGARDING USE OF PRESCRIBED MEDICATION

The seafarer's doctor shall ensure that the person has written documentation outlining the use of their medications. This should be in a form that can be shown to any official who may question the presence of the medication on board. This is particularly important for those medications that are legally prescribed controlled drugs (prescription group A and B in Norway) or those drugs which may be abused.

All seafarers who pass the medical examination, and who use prescribed medication, shall be provided with a declaration from the seafarer's doctor, including:

- a specification of the name of the medication;
- dosage; and
- a confirmation that permission has been granted for using the medication when on duty on board ship.

Below is the declaration form that should be completed by the seafarer's doctor for all persons using medicines on a regular basis:

Sjøfartsdirektoratet
Norwegian Maritime Authority

Erklæring fra sjømannslege om bruk av faste medikamenter/
Declaration from the Seafarer's Doctor regarding use of regular medicines

Arbeidstaker/*Employee*	
Etternavn/*Family name*	Fødselsdato/*Date of birth*
For- og mellomnavn/*First and middle name*	Stilling/*Position*

Nevnte arbeidstaker har fått helseerklæring for arbeid på norske skip og flyttbare innretninger/
The above mentioned employee has got a medical certificate for work on board Norwegian ships and offshore mobile units

Helseerklæring nr/*Medical Certificate No*	Utløpsdato/*Expiry date*

Jeg har vurdert eventuell sikkerhetsrisiko knyttet til bruk av nedenstående medikamenter under utførelse av tjenesten/*I have considered the possible safety risk related to the use of the below mentioned medicines whilst on duty.*

Jeg bekrefter at medikamentene tillates brukt under utførelsen av tjenesten om bord og ikke vil utgjøre noen sikkerhetsrisiko/*I confirm that the medicines are allowed during work on board, and do not imply a safety risk.*

Preparat/*Preparation*	Substans/*Substance*	Dosering/*Dosage*

Dato/*Date*	Sjømannslegens navn/*Name of seafarers' doctor*	Sjømannslegens signatur/*Signature of seafarers' doctor*

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16.5 SHORT-TERM TREATMENT WITH MEDICATION

Medicinal treatment of non-chronic illnesses shall as a rule be completed before a medical certificate can be issued.

Use of such medication is not included in the requirement for declaration regarding the use of prescribed medication.

It is the company's and the master's responsibility to have routines in place that cover short-term treatment and use of over-the-counter drugs.

16.6 MEDICATIONS THAT CAN IMPAIR ROUTINE AND EMERGENCY DUTIES

- Medications affecting the central nervous system functions (e.g. sleeping tablets, antipsychotics, some analgesics, some anti-anxiety and anti-depression treatments, anti-epileptics and antihistamines).
- Medications that increase the likelihood of sudden incapacitation (e.g. insulin, some of the older anti-hypertensives and medications predisposing to seizures).
- Medication impairing vision (e.g. hyoscine and atropine, but the list is long. A search carried out in Felleskatalogen (Norwegian equivalent to Physicians' Desk Reference) returned 190 hits in product monographs)[46].

16.7 MEDICATIONS THAT CAN HAVE SERIOUS ADVERSE CONSEQUENCES

- Excessive bleeding from injury or spontaneous bleeding (e.g. warfarin). Individual assessment of likelihood needed. Anticoagulants such as warfarin or dicoumarin normally have a likelihood of complications that is incompatible with work at sea but, if coagulation values are stable and closely monitored, work that does not carry an increased likelihood of injury and that is within reach of a helicopter with evacuation capacity may be considered.
- Dangers from cessation of medication use (hormones, insulin, anti-epileptics, anti-hypertensives, oral anti-diabetics, etc.).
- Antibiotics and other anti-infection agents.
- Anti-metabolites and other cancer treatments.
- Medications supplied for use at individual discretion (asthma treatments or antibiotics for recurrent infections).

[46] www.felleskatalogen.no searched 10.09.2013

16.8 MEDICATIONS THAT REQUIRE LIMITATION OF PERIOD AT SEA BECAUSE OF SURVEILLANCE REQUIREMENTS

A wide range of agents, such as anti-diabetics, anti-hypertensives and replacement therapy (hormones) may require close follow-up by a medical practitioner / specialist, and may therefore be incompatible with work at sea.

16.9 ISSUE OF MEDICAL CERTIFICATES IN THE EVENT OF MEDICATION USE

The seafarer's doctor must base his or her decision on reliable information regarding the use of medication, the side effects of the medication, the underlying condition and the need to treat it, and make make his or her assessment of the use of medication following a personal examination of the the person.

- UNFITNESS
  - The use of medication is incompatible with the reliable performance of routine and emergency duties safely or effectively if:
    - there is a risk of life-threatening consequences if medication is not taken as prescribed;
    - there is a risk of cognitive impairment when the medication is taken as prescribed;
    - there is a risk of severe adverse effects likely to be dangerous at sea, e.g. risk of bleeding when using anticoagulants.
- MEDICAL CERTIFICATE WITH LIMITATION
  - There is a risk of adverse effects, but these only develop over time, hence work in near-coastal waters may be acceptable.
- MEDICAL CERTIFICATE WITH TIME LIMITATION
  - Surveillance of medication effectiveness or side effects is needed more frequently than the full duration of a medical certificate.
- MEDICAL CERTIFICATE WITHOUT LIMITATIONS
  - No impairing side effects, no requirements for regular surveillance and no risk of life-threatening consequences if the medication is not taken.

16.9.1 ANTITHROMBOTIC THERAPY (2015, REVIEWED 2016)

The safety risk is connected to the risk of bleeding, or to the failure to reduce the risk of thrombosis related to the underlying condition.

In general, newer antihrombotic agents are regarded as having the same bleeding likelihood as warfarin, and the same risk assessment apply.

In general, it is not advised that patients on antithrombotic therapy (except for ASA) serve in positions where they may be exposed to trauma and increased likelihood of bleeding, or out of range of helicopters with a MEDEVAC capability.

There has been some confusion regarding the use of the term «anticoagulant» in the guidance. Strictly speaking an «anticoagulant drug» is a drug that works to prevent the coagulation (clotting) of blood, and as such inhibit thrombus formation. Some antithrombotic drugs, like antiplatelet drugs which are used for the same purpose (to prevent thrombus formation) through decreasing platelet aggregation are usually not regarded as “anticoagulants”.

Antithrombotic drugs include antiplatelet drugs, anticoagulants and thrombolytic drugs. However, according to the ATC-code, there is no clear distinction between these groups, and antithrombotic drugs are classified as, Vitamin K-antagonists, heparines, platelet aggregation inhibitors, enzymes, direct thrombin-inhibitors, direct Factor Xa-inhibitors, and “other” antithrombotic agents.

The clear distinction between these agents today was not so clear some years ago, and the tradition to use “anticoagulant” meaning “antithrombotic” is reflected in the regulations and in the guidance. Often “anticoagulation” is used also in official documents from authorities or professional bodies including both antiplatelet agents and anticoagulant agents. In Norwegian documents aimed at presenting antithrombotic therapy to the public, the authorities often use the term “blood-thinning” agents or “blood-thinners”, which both are confusing terms, as none of these agents actually makes the blood thinner (meaning reducing viscosity). The same tradition is seen in other countries.

The similarities in indication, and the similarities in bleeding risk, make it reasonable to compare newer antithrombotic drugs with warfarin. The only exception is acetylsalicylic acid (ASA) which has a lower bleeding risk than the others.

The use of the term “anticoagulant” in the regulations and the guidance thus should be understood as covering all antithrombotic agents, except ASA.

16.9.1.1 ASA THERAPY

The main adverse effect of aspirin is an increased risk of bleeding, chiefly from the gastrointestinal (GI) tract but also very rarely from intracranial vessels.

Meta-analyses of randomized trials have demonstrated that five years of treatment with 325 mg aspirin daily produces a nearly one percent absolute increase in risk of GI bleeding compared to placebo (2.47 versus 1.42 percent with placebo at an average of 28 months and 1.3 versus 0.5 percent at variable follow-up)[47] [48] [49].

[47] UpToDate: Benefits and risks of aspirin in secondary and primary prevention of cardiovascular disease, accessed 15. July 2014
[48] Derry S, Loke YK. Risk of gastrointestinal haemorrhage with long term use of aspirin: meta-analysis. BMJ 2000; 321:1183.
[49] Weisman SM, Graham DY. Evaluation of the benefits and risks of low-dose aspirin in the secondary prevention of cardiovascular and cerebrovascular events. Arch Intern Med 2002; 162:2197.

In indirect comparisons in the worldwide meta-analysis conducted by the Antithrombotic Trialists' Collaboration, there were no significant differences between 75 and 325 mg aspirin and risks of major extracranial bleeding based on data from three individual randomized trials[50].

The risk for severe bleeding during ongoing ASA therapy is increased by 70%[51] versus placebo, but absolute risk was only modestly increased.

Some patients need to be treated with ASA even if they have experienced gastric haemorrhage or ulcer formation. It is common to recommend proton-pump inhibitors for as long as the patient must use ASA, even if this is for life[52].

In a study of the effect of using proton-pump inhibitors in patients who have experienced ulcer complications during long-term use of ASA, it was found that the treatment with proton-pump inhibitors reduced the risk of recurrence from 14.8% to 1.6%[53].

The risk of stopping aspirin therapy in connection with a cardiovascular or cerebrovascular disorder which is being treated with ASA, is far too great to defend this. In a study by Sung et al. in 2010 the difference between the placebo group and the ASA group was significant after 8 weeks, with total mortality in the ASA group of 1.3%, against 12.9% in the placebo group[54]. The study was quite small, and only low-dose aspirin (80 mg) was used.

Long term treatment after minor stroke (TIA and RIND) reduces the total vascular mortality with approximately 15 % and nonfatal cerebrovascular and myocardial infarction with 30 %. The reocclusion after aortocoronary bypass is also reduced[55].

16.9.1.2 WARFARIN (MAREVAN®)

The treatment with warfarin may involve a safety risk on board due to its risk of causing bleeding. Intracerebral bleeding causes approximately 90 % of the deaths and most of the permanent disability in patients with warfarin-associated bleeding[56].

[50] Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. BMJ 2002; 324:71.
[51] McQuaid KR og Laine L (Systematic review and meta-analysis of adverse events of low-dose aspirin and clopidogrel in randomized controlled trials, Am J Med. 2006;119(8):624)
[52] Yeomans ND, Tulassay Z, Juhász L, Rácz I, Howard JM, van Rensburg CJ, Swannell AJ, Hawkey CJ: A comparison of omeprazole with ranitidine for ulcers associated with nonsteroidal antiinflammatory drugs. Acid Suppression Trial: Ranitidine versus Omeprazole for NSAID-associated Ulcer Treatment (ASTRONAUT) Study Group. N Engl J Med. 1998;338(11):719.
[53] Lai KC, Lam SK, Chu KM, Wong BC, Hui WM, Hu WH, Lau GK, Wong WM, Yuen MF, Chan AO, Lai CL, Wong J: Lansoprazole for the prevention of recurrences of ulcer complications from long-term low-dose aspirin use. N Engl J Med. 2002;346(26):2033
[54] Sung JJ, Lau JY, Ching JY, Wu JC, Lee YT, Chiu PW, Leung VK, Wong VW, Chan FK: Continuation of low-dose aspirin therapy in peptic ulcer bleeding: a randomized trial. Ann Intern Med. 2010;152(1):1.
[55] http://legemiddelhandboka.no/Legemidler/74869/?ids=74870#i74870
[56] Fang MC, Go AS, Chang Y, et al. Death and disability from warfarin-associated intracranial and extracranial hemorrhages. Am J Med 2007; 120:700.

Dependent on the dosage, warfarin treatment increases the risk for intracranial haemorrhage (ICH) two- to five-fold[57] [58] [59].

The dosage is individual, which means that patients need to be closely followed up, particularly in the initial period before they know their individual dose. Certain types of vegetables (deep green, e.g. broccoli) may increase the effect of Marevan. It is therefore important that the dose (once per day) is taken at the same time and with approximately the same relation to consumption of foodstuffs (distance to meal, contents of meal) every time.

The stability of the therapy, the need for monitoring by a doctor, the underlying condition, the service on board and the trade area are all factors which need to be taken into consideration on an individual basis in the risk assessment.

There is an increased risk of bleeding caused by Warfarin in the elderly and women, in the event of diabetes, cancer, high blood pressure, acute or chronic alcoholism, liver disease, kidney disease, anaemia, poor follow-up of treatment, prior stroke or cerebral haemorrhage, ulcer, co-use of acetylsalicylic acid, NSAIDs, platelet aggregation inhibitors, antibiotics, remedies against high cholesterol, antiarrhythmics, INR > 3.0, INR > 1.2 before start of treatment, or if prior bleeding on warfarin with INR within therapeutic range.

It is difficult to assess the risk for bleeding in a particular patient, since the risk varies a lot with several different concurrent factors. Several indices have been prepared in order to try and determine the risk for different patient groups. Low-risk groups have a bleeding risk over a four year period of around 3%, while high-risk groups can have a risk of over 50%[60].

The most severe bleeds are often from gastrointestinal tract. 15% are intracranial. This is a special risk at sea, since not only the blood loss is of significance, but the lack of intracranial expansion possibilities mean that even small bleeds may be serious and require immediate treatment at hospital - which often is not possible if you are far from land.

The interaction between warfarin and medication, foodstuffs and other disorders leads to a real risk of adverse events even in well-regulated patients.

The potential effect of anticoagulation in high-risk patients was examined in a study which assessed the effect of long-term treatment with warfarin (INR 2-2.85) over six months or indefinitely in patients who had had a second episode of DVT, but without detected thrombophilia. Long-term treatment with warfarin (Marevan) was found to be highly efficient in

[57] Rosand J, Eckman MH, Knudsen KA, et al. The effect of warfarin and intensity of anticoagulation on outcome of intracerebral hemorrhage. Arch Intern Med 2004; 164:880.
[58] Hart RG, Tonarelli SB, Pearce LA. Avoiding central nervous system bleeding during antithrombotic therapy: recent data and ideas. Stroke 2005; 36:1588.
[59] García-Rodríguez LA, Gaist D, Morton J, et al. Antithrombotic drugs and risk of hemorrhagic stroke in the general population. Neurology 2013; 81:566.
[60] Outpatient bleeding risk index

preventing new episodes of thrombosis (2.6% compared to 21% over four years). This effect was to a certain degree counteracted by the increased risk of major haemorrhage (8.6% compared to 2.3%) There was no difference in mortality between the two groups[61].

16.9.1.3 DABIGATRAN (PRADAXA®), RIVAROXABAN (XARELTO®) AND APIXABAN (ELIQUIS®)

Mainly based on the RE-LY, ROCKET-AF and ARISTOTLE trials, these drugs have been approved for the prevention of stroke in non-valvular atrial fibrillation.

Although dabigatran, rivaroxaban, and apixaban are promising agents for stroke prevention in atrial fibrillation, it is important to point out that there are no specific antidotes to reverse their anticoagulant effects. In addition, these agents are not used in patients with mechanical heart valves because the RE-ALIGN trial comparing dabigatran with warfarin found that dabigatran was associated with more ischemic strokes and more wound bleeding than warfarin[62].

Rivaroxaban and apixaban have not been compared with warfarin in patients with prosthetic heart valves.

There are great differences between the centres participating in the RE-LY study regarding how long the patients had been in therapeutic range (TTR). In the ebst studies, showing TTR 72,5% or above, there is no difference between effect and complication risk between dabigatran and warfarin[63].

So far, there is no reason to believe that the safety risk is reduced in persons on ships by replacing warfarin therapy with newer oral antithrombotic agents. One may gain smoething on easier follow-up, but uncertainty regarding bleeding likelihood and the lack of antidotes implies that the new agents should be regarded as comparable to warfarin in the risk assessment.

16.9.2 TREATMENT WITH CNS STIMULANTS

The use of CNS stimulants for ADHD in adults has increased over the last decades. Seafarer's doctors therefore will have to assess whether the use of such medicines on board ships is justified.

In this context, the persons capability to carry out duties shall be assessed in two different scenarios – with and without the use of medicines.

CNS stimulants are restricted preparations in most countries. In some countries they are extremely strict. The person may experience trouble on replenishment and trouble with some

[61] Schulman S, Granqvist S, Holmström M, Carlsson A, Lindmarker P, Nicol P, Eklund SG, Nordlander S, Lärfars G, Leijd B, Linder O, Loogna E: The duration of oral anticoagulant therapy after a second episode of venous thromboembolism. The Duration of Anticoagulation Trial Study Group. N Engl J Med. 1997;336(6):393

[62] Eikelboom JW, Connolly SJ, Brueckmann M, et al. Dabigatran versus warfarin in patients with mechanical heart valves. N Engl J Med 2013; 369:1206.

[63] Hansen J-B: Dokumentasjon for atrieflimmer og nye antikoagulantia, Indremedisineren 03:2012

authorities regarding the medicines he/she is carrying as illicit drugs. In addition there is a risk that such medicines may be stolen. The person therefore has a higher risk for being without necessary medicines on using CNS stimulants as compared to other medicines.

The underlying condition therefore is as important to assess as the medical condition whilst on CNS stimulants, and the use of the medicines themselves.

Due to the potential for abuse, misuse and recreational use the use of such substances in patients with known addiction to narcotics/drugs or alcohol is not recommended. Chronic abuse of CNS stimulants can lead to considerable tolerance and psychological addiction with a varying degree of deviant behaviour. Some psychotic symptoms can occur, particularly as response to parenteral abuse.

The preparations are known among drug addicts, and it must be taken into consideration that people other than the person may be interested in trying the preparation, which calls for the need for adequate storage.

It is important that a proper risk is carried out in each individual case and that this is properly documented and registered. Having done this and providing the doctor has found the risk acceptable, a certificate stating that the person is allowed to use the medicines whilst in service on board ship may be issued.

The below table[64] gives an overview of which substance concentrations of full blood that produce clinical influence corresponding to different levels of blood alcohol concentration – 0.2, 0.5 and 1.2 per mille.

Substance	Corresponding to 0,2 per mille (µmol/L full blood)	Corresponding to 0,5 per mille (µmol/L full blood)	Corresponding to 1,2 per mille (µmol/L full blood)
CNS stimulants			
Amfetamine	0,3	*	*
Cocaine	0,08	*	*
MDMA	0,25	*	*
Metamfetamine	0,3	*	
* Connection between substance concentration in full blood and accident risk / driving capabilities is variable or sparsely documented. Pronounced influenced can be noted at low concentrations, especially some time after consumption of substantial amounts of amfetamine/metamfetamine			

[64] Fakta om rusmiddelgrenser i trafikken, http://www.fhi.no/tema/rusmidler/rusmiddelgrenser-i-trafikken - visited 16th July 2014

16.9.3 BENZODIAZEPINES

Medication which has a sedative effect may constitute a threat to the safety on board, because it could have a large impact onreaction time, concentration and alertness.

Comparisons have been made between the intake of diazepam and alcohol concentration in the blood with regard to driving. They found that an intake of 15 mg of diazepam corresponds to a BAC of 0.1, if the person is not accustomed to the use. The skills needed for driving are quite similar to the skills needed on board a ship in many situations.

The effects may linger for a long while, dependening on the drug that is being taken. The half-life, i.e. the time it takes before the serum concentration has been halved, is crucial, and medications with a long half life are particularly associated with an increased accident risk.

Active ingredient	**Trade name**	**Half-life**
Diazepam	(Stesolid, Valium, Vival)	20-100 hours
Oksazepam	(Alopam, Sobril)	10-15 hours
Klonazepam	(Klonazepam, Rivotril)	20-60 hours
Nitrazepam	(Apodorm, Mogadon)	18-38 hours
Alprazolam	(Xanor, Xanor Depot)	9-20 hours
Midazolam	(Midazolam)	1.5-2.5 hours

A large problem is that many people think that they are less influenced by the medication than they actually are.

Anxiolytics not part of the benzodiazepine group usually do not have an unacceptable effect on reaction times, alertness and attentiveness. One exception is HYDRALAZIN (Atarax), which is quite sedative.

The below table[65] gives an overview of which substance concentrations of full blood that produce clinical influence corresponding to different levels of blood alcohol concentration – 0.2, 0.5 and 1.2 per mille.

Letting people work on board ships under the influence of such substances corresponds to letting people work on board ships under the influence of alcohol.

[65] Fakta om rusmiddelgrenser i trafikken, http://www.fhi.no/tema/rusmidler/rusmiddelgrenser-i-trafikken - visited 16th July 2014

Substance	Corresponding to 0,2 per mille (µmol/L full blood)	Corresponding to 0,5 per mille (µmol/L full blood)	Corresponding to 1,2 per mille (µmol/L full blood)
Benzodiazepines			
Alprazolam	0,01	0,02	0,05
Diazepam	0,2	0,5	1,2
Phenazepam	0,005	0,015	0,03
Flunitrazepam	0,005	0,01	0,025
Clonazepam	0,004	0,01	0,025
Nitrazepam	0,06	0,15	0,35
Oxazepam	0,6	1,5	3
Zolpidem	0,1	0,25	0,6
Zopiclone	0,03	0,06	0,15

Benzodiazepines will only on exceptional occasions be justified as a regular medicine. On these rare occasions it is necessary to judge the safety risk the use of the substance implies. As a general rule, such medicines is not in accordance with safe conduct of ordinary and emergency duties on board ships.

17 COMMON MEDICAL CONDITIONS

17.1 INTRODUCTION

It is not possible to develop a comprehensive list of fitness criteria covering all possible conditions and the variations in their severity, symptomatology, prognosis and treatment.

The principles underlying the approach adopted in the table below may often be extrapolated to conditions not covered by it. Analog assessment should be used. The seafarer's doctors must in any case assess whether the person is medically and physically fit to reliably perform his or her routine and emergency duties safely and effectively.

The table of medical conditions is laid out as follows:

Column 1: WHO International Classification of Diseases, 10. Edition (ICD-10). Codes are listed as an aid to collection and comparison of data for statistics and research purposes.

Column 2: The common name of the condition or group of conditions, with a brief statement on its relevance to work at sea.

Column 3: Description of conditions that are incompatible with work at sea. This column should be consulted first.

Column 4: Description of conditions that should entail a medical certificate with limitation or time limitation. This column should be consulted if the person does not fit the criteria in column 3.

Column 5: Description of conditions that are compatible with a medical certificate without limitations. This column should be consulted only when the person does not fit the criteria in columns 3 or 4.

For some conditions, one or more columns have been given the description "Not applicable". This is used where this type of medical certificate is either not relevant or not appropriate.

Terms used:

P: Permanent unfitness (red category)
T: Temporary unfitness (red category)
R: Medical certificate with restrictions in position or trade area (yellow category)
L: Medical certificate with restrictions in period of validity (yellow category)
H: Medical certificate without restrictions in position, trade area or validity (green category)

Unfitness means a condition which is incompatible with the reliable performance of routine and emergency duties safety and effectively.

17.2 INFECTIOUS AND PARASITIC DISEASES

17.2.1 GASTROINTESTINAL INFECTIONS

A00–09	Gastrointestinal infection Transmission to others - recurrence	T– If detected while onshore. (current symptoms or awaiting test results on carrier status); or confirmed carrier status until elimination demonstrated	Not applicable	Non-catering personnel: When satisfactorily treated Catering personnel: Medical certificate depends on individual medical assessment. Bacteriological clearance may be required.

Whilst most cases of acute gastrointestinal infection and diarrhoea are self limiting they can cause significant morbidity and mortality. In the confined environment of a ship there is also a significant risk of the spread of gastrointestinal infection particularly if the affected person is a food handler. It is therefore vital that a person should be deemed temporarily unfit whilst he/she has acute symptons and/or there is doubt with regards to carrier status. An individual risk assessment should be carried out in each case before the person is issued with an unrestricted certificate.

Reviewed 2014

17.2.2 PULMONARY TB(2015)

A15-16	Pulmonary TB Transmission to others, recurrence (testing according to Regulations on tuberculosis control)	T – Positive screening test or clinical history - until investigated. If infected - until treatment stabilised and lack of infectivity confirmed. P – Relapse or severe residual damage	Not applicable	Successful completion of a course of treatment in accordance with the Regulations on tuberculosis control (and WHO Treatment of Tuberculosis guidelines).

Tuberculosis carries the risk of limitations in physical capabilities due to the illness itself or complications of it, a risk of infection for others on board and a need for treatment and control which could be made difficult by working on board a ship.

The Appelate Body may consider limited and restricted coastal service, provided the person is compliant with treatment demands, has good understanding of the disease and is physically fit. It is further a prerequisite that there is a written agreement regarding Direct Observed Therapy (DOT) with the Officer in charge of medical care on board, and an agreement with the tuberculosis coordinator at the hospital onshore where the treatment is supervised (Norway).

Examination with regard to tuberculosis follows ***FOR-2002-06-21-567 Regulations on control of tuberculosis***. It is not prepared for persons in particular, but should be used as far as possible. Chapter 3 describes how the tuberculosis examination shall be carried out.

Chapter 3 reads:

17.2.2.1 SECTION 3-1. OBLIGATION TO UNDERGO TUBERCULOSIS EXAMINATION

The following persons are required to undergo a tuberculosis examination:

Persons from countries with a high occurrence of tuberculosis, who are staying in the country for more than three months and who are not exempt from the requirement for work permit or residence permit, along with refugees and asylum seekers. The tuberculosis examination includes tuberculin testing of this group and x-ray examination of persons over the age of 15.

Persons coming from or having stayed at least three months in countries with a high occurrence of tuberculosis, and who are to take up or continue in a position in the health and care services, in teaching positions or in other positions related to child care. The obligation also applies to persons in training for or working as trainees in such positions.

Other persons who there is a medical reason to suspect are or have been at risk of being infected with tuberculosis.

A person infected with a tuberculous disease has a duty to accept the individual infection control guidelines provided by the medical practitioner to prevent the disease from being transmitted to others and a duty to let himself be placed in isolation if necessary.

Tuberculosis examinations pursuant to these Regulations shall be free of charge for the person required to undergo such examination. Vaccination of tuberculin negative persons against tuberculosis shall be free of charge for the persons concerned. Travel expenses in connection with visiting a doctor for a tuberculosis examination and/or vaccination shall be free of charge for the person concerned.

The Norwegian Armed Forces provides guidelines for the examination of military personnel.

17.2.2.2 SECTION 3-2. EXECUTION

Examination of persons as mentioned in section 3-1 shall be executed as soon as possible.

Refugees and asylum seekers shall be examined within fourteen days after entry into the country.

Persons as mentioned in section 3-1 No. 2 shall be examined before taking up the position. The employer has a duty to see to that tuberculosis examinations are carried out prior to appointment.

17.2.2.3 SECTION 3-3. FOLLOW-UP

If a tuberculosis examination reveals symptoms or signs which could indicate tuberculous disease, the person concerned shall be referred to a diagnostic station, a paediatric ward, or a pulmonary or infectious disease department of an out-patient clinic for further evaluation and additional examinations. Upon suspicion of contagious pulmonary tuberculosis an investigation shall be initiated immediately.

A specialist in pulmonology, infectious diseases or a paediatrician shall be responsible for initiating treatment and selecting treatment regime. The treatment shall take place in accordance with recommended international rules for tuberculosis control, including (usually) directly observed treatment.

The specialist shall immediately notify the tuberculosis coordinator who is responsible for establishing a treatment plan for the patient for the entire treatment period. The treatment plan shall be established in cooperation with the specialist, the patient and the municipal medical officer. Follow-up and control, including observation of ingestion of tuberculosis medication, shall take place in cooperation with the municipal health service.

Patients with multidrug-resistant tuberculosis shall be treated at the hospital designated by the regional health trust.

17.2.2.4 SECTION 3-4. EXEMPTION FROM TUBERCULOSIS EXAMINATION

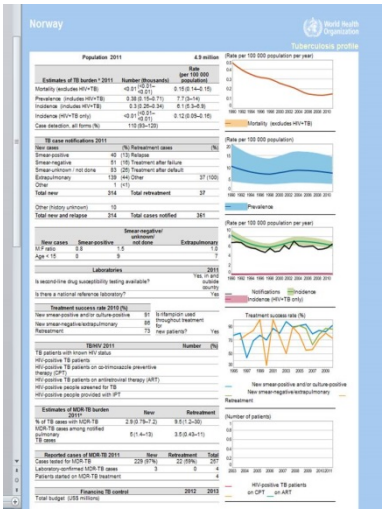
Decisions on exemption from the duty to undergo tuberculosis examination, cf. section 3-1, are made by the municipal medical officer together with the hospital doctor designated by the municipal health service pursuant to section 7-3 third paragraph of the Act relating to control of communicable diseases. The county medical officer shall make decisions on appeals against decisions.

A form which can be used for requesting support in a decision regarding the screening of persons for tuberculosis can be found on the website of the Norwegian Institute of Public Health[66]. New forms are expected by October 2014. Persons shall be assessed in accordance with the category “immigrant worker”.

To conclude , we can say that if 1) the person comes from a country with a TB incidence >40/100 000/year, 2) there is information regarding previous tuberculosis, 3) there is information regarding environmental infection or 4) there is clinical suspected tuberculosis, there is an indication to carry out a tuberculosis examination. The method of examination is decided by the Norwegian Institute of Public Health. During examination of persons on ships, usually it is sufficient to to exclude active tuberculosis by means of chest X-ray (CXR). If findings during clinical

[66] http://www.fhi.no/publikasjoner-og-haandboker/tuberkuloseveilederen/skjemaer-og-maler

examination or CXR raise suspicion of active tuberculosis, further examination of sputum or more advanced radiological methods must be carried out to exclude active tuberculosis before going to sea.



17.2.2.5 OCCURRENCE OF TUBERCULOSIS

Updated information about the occurrence of tuberculosis in the world can be found on the WHO's website: http://www.who.int/tb/country/en/ in the form of a so-called "tuberculosis profile".

Through a simple search function (name of country) you can find all the important key figures for each country including prevalence, incidence, mortality.

This is useful supportive documentation when deciding whether an person needs to be further examined for tuberculosis. An example from Norway is given in the figure to the left.

17.2.3 SEXUALLY TRANSMISSIBLE INFECTIONS

A50-64	Sexually transmissible infections Acute deterioration, recurrence	T – If detected while onshore - until diagnosis confirmed, treatment initiated and impairing symptoms resolved. P – Untreatable impairing late complications	R – Consider near-coastal if oral treatment regime in place and symptoms resolved.	On successful completion of treatment.

Sexually transmitted infections (STIs) are a major public health problem in developed and developing countries. Complications of untreated STIs include upper genital tract infections, infertility, cervical cancer, and enhanced transmission and acquisition of the human immunodeficiency virus (HIV). The person should be made temporarily unfit whilst a potential STI is investigated and treatment initiated and until he/she is confirmed to have no symptoms that may impair their ability to perform their duties. Once this is the case a certificate, with or without restrictions can be issued.

Reviewed 2014

17.2.4 HEPATITIS A

B15	Hepatitis A Transmissible by food or water contamination	T – Until jaundice resolved and liver function tests returned to normal.	Not applicable	On full recovery

The significance of Hepatitis A virus (HAV) is that is can be transmitted by food or water contamination. The incidence of Hepatitis A has declined since the introduction of a vaccine (in 1996). The spread is faecal-oral and there is the risk of infecting other crew members.

In the acute phase, and as long as there is a danger of infection, the person must be considered unfit. Restrictions in the service or trade area is normally not relevant. Upon full recovery a medical certificate without restrictions is the normal decision.

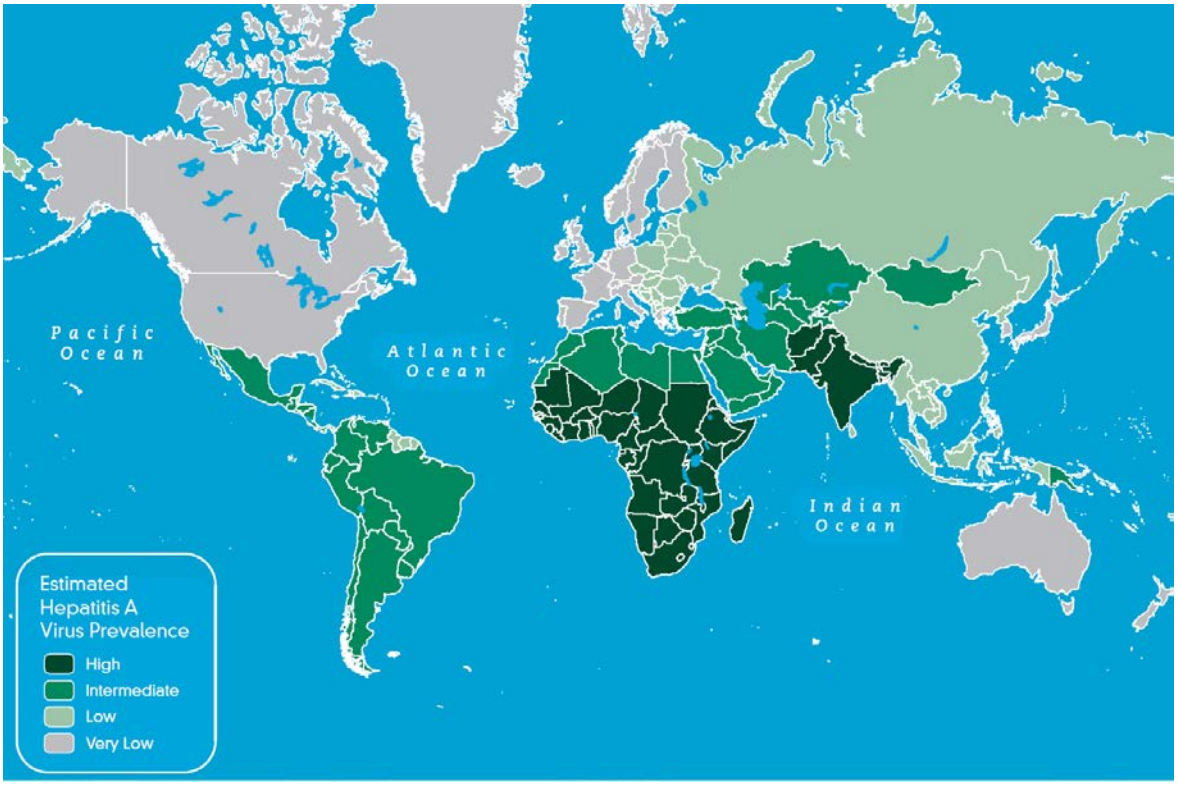
The most common risk factor in contracting HAV is international travel (up to 50% of the cases). Other risk factors include contact with infected persons (10%), homosexual activity (9%), food- or waterborne outbreaks (7%), child or person in a daycare centre (4%) and injection drug use (3%)[67].

Because the disease is usually self-limited, the treatment is supportive. Occasional patients require hospitalization (20 percent in the large outbreak[68]). Patients who develop fulminant infection require aggressive supportive therapy, possibly liver transplantation.

Approximately 85 percent of HAV-infected individuals have full clinical and biochemical recovery within three months, and nearly all have complete recovery by six months[69].

Fatalities due to hepatitis A are more common with advancing age and in patients with chronic hepatitis C[70] [71]. Reported case fatality rates are 0.1 percent in infants and children, 0.4 percent between the ages of 15 and 39, and 1.1 percent in those over age 40[72].

[67] http://www.uptodate.com/contents/overview-of-hepatitis-a-virus-infection-in-adults?detectedLanguage=en&source=search_result&search=hepatitis+A&selectedTitle=1%7E150&provider=noProvider
[68] Wheeler C, Vogt TM, Armstrong GL, et al. An outbreak of hepatitis A associated with green onions. N Engl J Med 2005; 353:890.
[69] Koff RS. Clinical manifestations and diagnosis of hepatitis A virus infection. Vaccine 1992; 10 Suppl 1:S15.
[70] Vento S, Garofano T, Renzini C, et al. Fulminant hepatitis associated with hepatitis A virus superinfection in patients with chronic hepatitis C. N Engl J Med 1998; 338:286.
[71] Vogt TM, Wise ME, Bell BP, Finelli L. Declining hepatitis A mortality in the United States during the era of hepatitis A vaccination. J Infect Dis 2008; 197:1282.
[72] Centers for Disease Control. Hepatitis Surveillance Report 1990; 53:23.



Occurrence of Hepatitis A in the world as of July 17th 2013

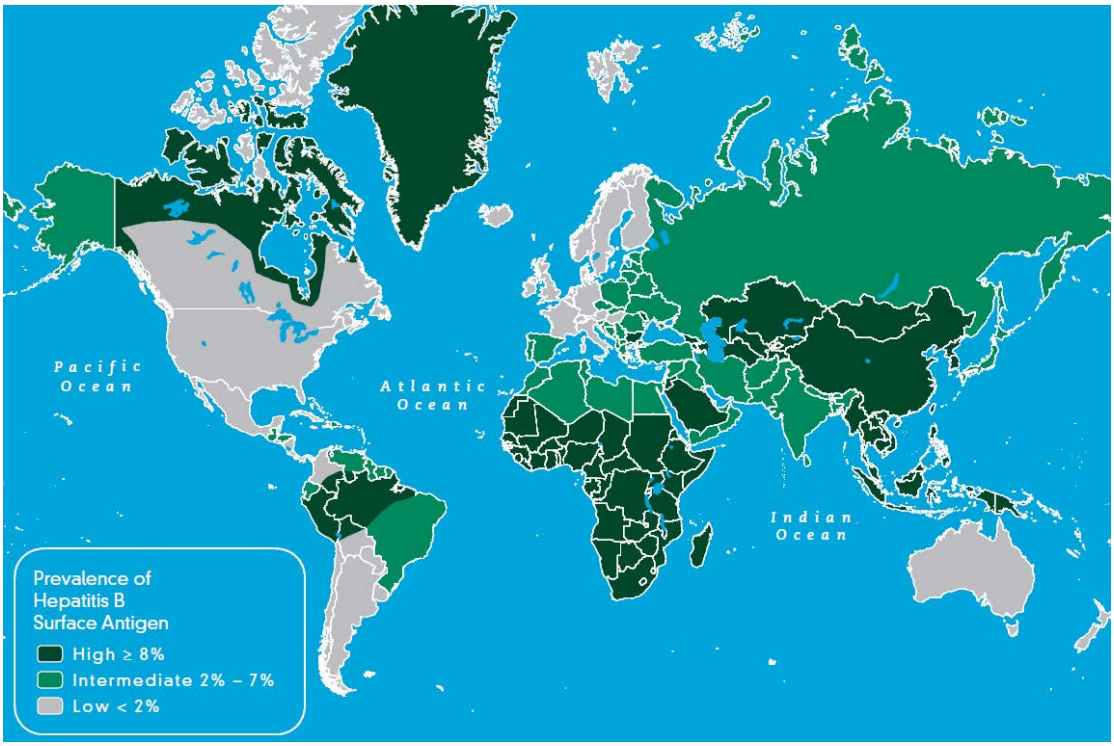
Source: http://wwwnc.cdc.gov/travel/yellowbook/2012/chapter-3-infectious-diseases-related-to-travel/hepatitis-a

Reviewed 2014

17.2.5 HEPATITIS B

B16-19	Hepatitis B, C, etc. Transmissible by contact with blood or other bodily fluids. Possibility of permanent liver impairment and liver cancer.	T – Until jaundice resolved and liver function tests returned to normal. P – Persistent liver impairment with symptoms affecting reliable, safe and effective performance of duties	R, L – Uncertainty about total recovery or lack of infectivity. Case-by-case assessment based on duties on board and trade area.	On full recovery and confirmation of low level of infectivity.

The significance of Hepatitis B is firstly the impaired physical capability and secondly the risk of complications which could lead to an acute exacerbation on board. It is not considered necessary to take theinfectious risk into consideration, as the disease is only transmitted by contact with blood and bodily fluids. As long as there is jaundice or the liver enzymes are abnormal, the person is unfit. In the event of chronic hepatitis, permanent unfitness is a likely decision. Restrictions in validity or trade area could be considered if there is uncertainty regarding full recovery or the infectious status. Upon full recovery an unrestricted medical certificate is the most common decision.



Occurrence of Hepatitis B in the world as at July 16th 2013.

Source: http://wwwnc.cdc.gov/travel/yellowbook/2012/chapter-3-infectious-diseases-related-to-travel/hepatitis-b.htm

Hepatitis B can be both acute and chronic. During the acute phase, manifestations range from subclinical or anicteric hepatitis to icteric hepatitis and, in some cases, fulminant hepatitis.

Approximately 70% of patients with acute Hepatitis B have subclinical or anicteric hepatitis, whilst 30% develop icteric hepatitis. The disease can be more severe in patients coinfected with other Hepatitis viruses[73] although fulminant hepatitis is unusual, and occurs in approximately 0.1 to 0.5% of patients[74].

The rate of progression from acute to chronic Hepatitis B is determined primarily by the age at infection. The rate is approximately 90% for a perinatally acquired infection[75], 20 to 50% for infections acquired between the age of one and five years[76], and less than 5% for an adult-acquired infection[77].

[73] Liaw YF, Tsai SL, Sheen IS, Chao M, Yeh CT, Hsieh SY, Chu CM: Clinical and virological course of chronic hepatitis B virus infection with hepatitis C and D virus markers. Am J Gastroenterol. 1998;93(3):354.
[74] Wright TL, Mamish D, Combs C, Kim M, Donegan E, Ferrell L, Lake J, Roberts J, Ascher NL:Hepatitis B virus and apparent fulminant non-A, non-B hepatitis. Lancet. 1992;339(8799):952.
[75] Stevens CE, Beasley RP, Tsui J, Lee WC: Vertical transmission of hepatitis B antigen in Taiwan. N Engl J Med. 1975;292(15):771.
[76] Beasley RP, Hwang LY, Lin CC, Leu ML, Stevens CE, Szmuness W, Chen KP: Incidence of hepatitis B virus infections in preschool children in Taiwan. J Infect Dis. 1982;146(2):198
[77] Tassopoulos NC, Papaevangelou GJ, Sjogren MH, Roumeliotou-Karayannis A, Gerin JL, Purcell RH:Natural history of acute hepatitis B surface antigen-positive hepatitis in Greek adults. Gastroenterology. 1987;92(6):1844.

There are studies suggesting that the complete eradication of HBV rarely occurs after recovery from acute HBV infection and that latent infection can maintain the T cell response for decades following clinical recovery, thereby keeping the virus under control[78]. It is not clear how often latent infection can lead to liver cirrhosis, but the use of immunosuppressants can lead to reactivation of the virus.

Approximately 30 to 50% of patients with chronic HBV infection have a past history of acute hepatitis. Many are asymptomatic. The sequelae of chronic HBV infection varies from an inactive carrier state to the development of cirrhosis, liver failure, hepatocellular carcinoma (HCC), extrahepatic manifestations, and death.

The prognosis appears to vary with the clinical setting.

17.2.5.1 CHRONIC CARRIERS:

- Long-term follow-up studies of HBsAg positive blood donors have shown that the majority remain asymptomatic with a very low risk of cirrhosis or HCC[79] [80] [81].
- In a 16-year follow-up study of 317 HBsAg positive blood donors, only three died from HBV-related cirrhosis and none developed HCC[82].
- HBV-infected patients from endemic areas and in patients with chronic hepatitis:[83] [84] [85] [86].

The estimated five-year rates of progression are as follows[87]:

- Chronic hepatitis to cirrhosis - 12 to 20%
- Compensated hepatitis to liver failure - 20 to 23%
- Compensated cirrhosis to HCC - 6 to 15%

[78] Rehermann B, Ferrari C, Pasquinelli C, Chisari FV: The hepatitis B virus persists for decades after patients' recovery from acute viral hepatitis despite active maintenance of a cytotoxic T-lymphocyte response. Nat Med. 1996;2(10):1104.
[79] Villeneuve JP, Desrochers M, Infante-Rivard C, Willems B, Raymond G, Bourcier M, CôtéJ, Richer G:A long-term follow-up study of asymptomatic hepatitis B surface antigen-positive carriers in Montreal. Gastroenterology. 1994;106(4):1000.
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[82] Villeneuve JP, Desrochers M, Infante-Rivard C, Willems B, Raymond G, Bourcier M, CôtéJ, Richer G:A long-term follow-up study of asymptomatic hepatitis B surface antigen-positive carriers in Montreal. Gastroenterology. 1994;106(4):1000.
[83] Fattovich G, Brollo L, Giustina G, Noventa F, Pontisso P, Alberti A, Realdi G, Ruol A: Natural history and prognostic factors for chronic hepatitis type B. Gut. 1991;32(3):294
[84] Fattovich G, Giustina G, Schalm SW, Hadziyannis S, Sanchez-Tapias J, Almasio P, Christensen E, Krogsgaard K, Degos F, Carneiro de Moura M: Occurrence of hepatocellular carcinoma and decompensation in western European patients with cirrhosis type B. The EUROHEP Study Group on Hepatitis B Virus and Cirrhosis. Hepatology. 1995;21(1):77.
[85] Liaw YF, Tai DI, Chu CM, Chen TJ: The development of cirrhosis in patients with chronic type B hepatitis: a prospective study. Hepatology. 1988;8(3):493.
[86] Liaw YF, Lin DY, Chen TJ, Chu CM: Natural course after the development of cirrhosis in patients with chronic type B hepatitis: a prospective study. Liver. 1989;9(4):235.
[87] Fattovich G, Bortolotti F, Donato F: Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol. 2008;48(2):335.

The cumulative survival rate at each of these stages of progressive disease is[88 89 90 91 92]:

- Compensated cirrhosis - 85% at five years
- Decompensated cirrhosis - 55 to 70% at one year and 14 to 35% at five years

A number of factors influence the prognosis of chronic HBV infection and these are too extensive to discuss here.

Reactivation is common in patients who receive immunosuppressive therapy, but rarely occurs spontaneously.

It is not uncommon to find coinfection with Hepatitis C virus (HCV) or Hepatitis D virus (HDV), resulting in a worse prognosis[93]. HCV has been estimated to be present in 10 - 15 % of patients with HBV-associated chronic hepatitis, cirrhosis, or HCC[94]. 62% of patients with HCV infection had evidence of exposure to HBV, while 6% had chronic HBV infection[95].

Reviewed 2014

17.2.6 HIV AND AIDS

B20-24	HIV+ Transmissible by contact with blood or other bodily fluids. Progression to HIV-associated diseases or AIDS.	T – Until stabilised on treatment with CD4 level of >350 or when treatment changed and tolerance of new medication uncertain. P – Non-reversible impairing HIV-associated disease. Continuing impairing effects of medication.	R, L – HIV+ and low likelihood of progression; on no treatment or on stable medication without side effects, but requiring regular specialist surveillance.	HIV+, no current impairment and very low likelihood of disease progression. No side effects of treatment or need for frequent monitoring.

[88] Fattovich G, Giustina G, Schalm SW, Hadziyannis S, Sanchez-Tapias J, Almasio P, Christensen E, Krogsgaard K, Degos F, Carneiro de Moura M: Occurrence of hepatocellular carcinoma and decompensation in western European patients with cirrhosis type B. The EUROHEP Study Group on Hepatitis B Virus and Cirrhosis. Hepatology. 1995;21(1):77.
[89] Liaw YF, Lin DY, Chen TJ, Chu CM: Natural course after the development of cirrhosis in patients with chronic type B hepatitis: a prospective study. Liver. 1989;9(4):235.
[90] Fattovich G, Bortolotti F, Donato F:Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol. 2008;48(2):335.
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[93] Mimms LT, Mosley JW, Hollinger FB, Aach RD, Stevens CE, Cunningham M, Vallari DV, Barbosa LH, Nemo GJ:Effect of concurrent acute infection with hepatitis C virus on acute hepatitis B virus infection. BMJ. 1993;307(6912):1095.
[94] Liaw YF: Role of hepatitis C virus in dual and triple hepatitis virus infection. Hepatology. 1995;22(4 Pt 1):1101.
[95] Bini EJ, Perumalswami PV: Hepatitis B virus infection among American patients with chronic hepatitis C virus infection: prevalence, racial/ethnic differences, and viral interactions. Hepatology. 2010;51(3):759.

17.2.6.1 GENERAL

Since HIV is transmitted through blood and bodily fluids, the risk of transmission is most often related to lifestyle, such as sexual relations and practices, the use of injected illicit drugs and the adequacy of infection control practices in clinical care.

Because of the form of transmission and consequent stigma associated to those with such conditions, legal and ethical aspects become important in the assessment of a patient with HIV.

The risk of infection in the workplace will only be present in connection with accidents where blood has been spilt. With normal precautions this is not a problem. The risk of sudden incapacitation is very low in the early stages of HIV infection, and should not entail unfitness. Side-effects of some forms of treatments may reduce performance.

Control and follow-up of treatment could be rendered difficult when not sailing near-coastal[96].

HIV infection which is being treated will generally not affect the working capacity, other than the occurrence of any side effects. It is also necessary to check and follow-up the treatment at certain intervals. The need for follow-up can determine whether restrictions should be set for validity or trade area. Most patients undergoing treatment for HIV infection can work as normal, as long as the treatment is followed and follow-up is carried out.

Untreated HIV infection could be very serious, both in the short term in some cases and in the long term in most cases.

17.2.6.2 PRIMARY HIV INFECTION

The presence of symptoms and a prolonged illness of more than 14 days appears to correlate with more rapid progression to AIDS[97] [98]. In the study by Pedersen et al, it was found that the risk of progression to AIDS within three years was 78% in those with acute symptoms and illness lasting more than 14 days, compared to 10% in those with symptoms and illness of a shorter duration and/or who had only mild symptoms.

[96] Carter T: Handbook for medical examiners, http://handbook.ncmm.no
[97] Pedersen C, Lindhardt BO, Jensen BL, Lauritzen E, Gerstoft J, Dickmeiss E, Gaub J, Scheibel E, Karlsmark T: Clinical course of primary HIV infection: consequences for subsequent course of infection. BMJ. 1989;299(6692):154.
[98] Niu MT, Stein DS, Schnittman SM: Primary human immunodeficiency virus type 1 infection: review of pathogenesis and early treatment intervention in humans and animal retrovirus infections. J Infect Dis. 1993;168(6):1490.

17.2.6.3 SEROCONVERSION

Seroconversion occurs within 4 to 10 weeks after exposure, and ≥95% seroconvert within six months[99] [100] [101].

17.2.6.4 CLINICAL LATENT PERIOD

This is the period after seroconversion until significant symptoms start to appear. With appropriate treatment this can last for decades although the period may be much shorter.

Viral load is the most important predictor of progressive disease in the early stages of HIV infection whilst the CD4 count is an important prognostic indicator in late stage disease[102] [103] [104].

The CD4 count is usually 1000/mm^3 in the early stages of the disease, but this decreases to 780/mm^3 at six months post-seroconversion in untreated patients and to 670/mm^3 at one year[105]. Some untreated patients have a more rapid progression and one study found that 28% of patients had a CD4 count of < 350/mm^3 at 36 weeks, and 50% at 72 weeks[106].

17.2.6.5 EARLY SYMPTOMATIC HIV INFECTION

During the early stages of HIV infection the person may note the occurrence of various conditions associated with HIV infection, but which also occur in association with many other disorders eg candidiasis, leukoplakia, zoster, neuropathy, cervical dyplasia, cervical carcinoma in situ, fever, diarrhea, ITP, lesteriosis, etc. These conditions are not pathognomonic for HIV infection.

17.2.6.6 AIDS

This is associated with the presence of severe immunosuppression and is classified intodifferent categories by the CD4 count.

- >500/mm^3 in category A1,

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[99] Coutlée F, Olivier C, Cassol S, Voyer H, Kessous-Elbaz A, Saint-Antoine P, He Y, Fauvel M: Absence of prolonged immunosilent infection with human immunodeficiency virus in individuals with high-risk behaviors. Am J Med. 1994;96(1):42.
[100] Simmonds P, Lainson FA, Cuthbert R, Steel CM, Peutherer JF, Ludlam CA:HIV antigen and antibody detection: variable responses to infection in the Edinburgh haemophiliac cohort. Br Med J (Clin Res Ed). 1988;296(6622):593.
[101] Sheppard HW, Busch MP, Louie PH, Madej R, Rodgers GC: HIV-1 PCR and isolation in seroconverting and seronegative homosexual men: absence of long-term immunosilent infection. J Acquir Immune Defic Syndr. 1993;6(12):1339.
[102] Schacker TW, Hughes JP, Shea T, Coombs RW, Corey L: Biological and virologic characteristics of primary HIV infection. Ann Intern Med. 1998;128(8):613.
[103] Mellors JW, Rinaldo CR Jr, Gupta P, White RM, Todd JA, Kingsley LA:Prognosis in HIV-1 infection predicted by the quantity of virus in plasma. Science. 1996;272(5265):1167.
[104] Giorgi JV, Lyles RH, Matud JL, Yamashita TE, Mellors JW, Hultin LE, Jamieson BD, Margolick JB, Rinaldo CR Jr, Phair JP, Detels R, Multicenter AIDS Cohort Study: Predictive value of immunologic and virologic markers after long or short duration of HIV-1 infection. J Acquir Immune Defic Syndr. 2002;29(4):346.
[105] Stein DS, Korvick JA, Vermund SH: CD4+ lymphocyte cell enumeration for prediction of clinical course of human immunodeficiency virus disease: a review. J Infect Dis. 1992;165(2):352.
[106] Hogan C, DeGruttola V, Daar E, et al. A finite course of ART during early HIV-1 infection modestly delays need for subsequent ART initiation: ACTG A5217, the SETPOINT Study. 2010 Conference on Retroviruses and Opportunisic Infections, Abstr. #134.

- 200-499/mm^3 in category A2,
- • < 200/mm^3 in category A3.

In the Multicenter AIDS Cohort Study, the median CD4 count at the time of an AIDS-defining complication was 67/mm^3. However, approximately 10% of patients developed an AIDS-defining diagnosis with a CD4 count above 200/mm^3[107].

The median time from the onset of severe immunosuppression (defined as a CD4 cell count < 200/mm^3) to an AIDS-defining diagnosis is 12 to 18 months in persons not receiving antiretroviral treatment[108].

Patients with advanced HIV infection have a CD4 cell count below 50/mm^3. Median survival is then only 12 to 18 months in the absence of antiretroviral therapy[109] [110].

17.2.6.7 ASYMPTOMATIC PERSONS

"LONG-TERM NONPROGRESSORS"

Some patients exhibit remarkable clinical stability and remain asymptomatic over many years without antiretroviral therapy, ie. no symptoms for at least 10 years, no antiretroviral therapy, CD4 count > 500/mm^3. Longitudinal studies show that 4 to 7% of HIV-infected patients satisfy these criteria[111] [112].

Other studies show that as many as 13% of men who have sex with men (MSM) and are HIV infected at a young age will remain asymptomatic for more than 20 years without treatment[113].

DECLARATION FROM SPECIALIST

Specialist advice is essential for each individual assessment. The specialist's declaration must include prognostic factors, the treatment given and side-effects, if any, the stability of treatment with specification of the CD4 count, the need for controls (frequency) and where the controls must/should/can be carried out.

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[107] Taylor JM, Sy JP, Visscher B, Giorgi JV: CD4+ T-cell number at the time of acquired immunodeficiency syndrome. Am J Epidemiol. 1995;141(7):645.
[108] Karon JM, Buehler JW, Byers RH, Farizo KM, Green TA, Hanson DL, Rosenblum LS, Gail MH, Rosenberg PS, Brookmeyer R: Projections of the number of persons diagnosed with AIDS and the number of immunosuppressed HIV-infected persons--United States, 1992-1994. MMWR Recomm Rep. 1992;41(RR-18):1.
[109] Yarchoan R, Venzon DJ, Pluda JM, Lietzau J, Wyvill KM, Tsiatis AA, Steinberg SM, Broder S:CD4 count and the risk for death in patients infected with HIV receiving antiretroviral therapy. Ann Intern Med. 1991;115(3):184.
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[111] Hughes MD, Stein DS, Gundacker HM, Valentine FT, Phair JP, Volberding PA: Within-subject variation in CD4 lymphocyte count in asymptomatic human immunodeficiency virus infection: implications for patient monitoring. J Infect Dis. 1994;169(1):28.
[112] Gottlieb GS, Sow PS, Hawes SE, Ndoye I, Redman M, Coll-Seck AM, Faye-Niang MA, Diop A, Kuypers JM, Critchlow CW, Respess R, Mullins JI, Kiviat NB: Equal plasma viral loads predict a similar rate of CD4+ T cell decline in human immunodeficiency virus (HIV) type 1- and HIV-2-infected individuals from Senegal, West Africa. J Infect Dis. 2002;185(7):905.
[113] Wei X, Ghosh SK, Taylor ME, Johnson VA, Emini EA, Deutsch P, Lifson JD, Bonhoeffer S, Nowak MA, Hahn BH: Viral dynamics in human immunodeficiency virus type 1 infection. Nature. 1995;373(6510):117.

ASSESSMENT

Some individuals will require frequent controls, and some have a greater risk of rapid impairment during the validity period. As long as the CD4 count is not stable, or is below 350/mm^3, the person is considered unfit, but could possibly return to work if the CD4 count stabilises above 350/mm^3. If there are adverse effects of the treatment, or if complications/AIDS-related conditions have arisen, permanent unfitness will be the right decision. If there are doubts regarding stability, improvement or stage of infection, the medical certificate should be restricted, both in validity and trade area. An unrestricted medical certificate is only applicable in cases where a low level of infection and full clinical recovery is documented.

Reviewed 2014

17.2.7 OTHER INFECTIONS				
A00-B99	Other infections Personal impairment, infection of others	T– If detected while onshore: until free from risk of transmission and capable of performing duties. P – If continuing likelihood of repeated impairing or infectious recurrences.	Case-by-case assessment based on nature of infection.	Full recovery and confirmation of low level of infectivity.

Any infection carries the risk to the person of personal impairment to the degree that he/she cannot perform their duties but also the risk to other crew members of the transmission of infection. In addition it should be considered if the person is fit to travel to the point of embarkation by whatever means is necessary. Airlines in particular have their own guidelines on passengers travelling whilst suffering from an infectious disease[114].

In addition it is a legal requirement that certain infectious diseases are notified to port health officials and in some cases, this can then lead to the quarantine of the ship with significant impact on the ship's schedule.

Reviewed 2015

[114] IATA Medical Manual: ISBN 978-92-9252-195-0

17.3 NEOPLASMS

C00-D48	Malignant neoplasms, including lymphoma, leukaemia and related conditions. Recurrence, especially acute complications, e.g. acute spontaneous bleeding and seizures.	T – Until investigated, treated and prognosis assessed P – Continuing impairment with symptoms affecting work at sea and with high likelihood of recurrence	L – Limited to interval between specialist reviews if: – cancer diagnosed <5 years ago; and – there is no current impairment of ability to perform normal or emergency duties or to live at sea; and – there is a low likelihood of recurrence and minimal risk of requirement for urgent medical treatment / hospitalisation R – If any continuing impairment does not interfere with essential duties and any recurrence is unlikely to require emergency medical treatment / hospitalisation.	Cancer diagnosed more than 5 years ago, or specialist reviews no longer required and no current impairment with low continuing likelihood of impairment from recurrence. To be confirmed by specialist report with evidence for opinion stated.

17.3.1 SIGNIFICANCE

The significance of these with regard to safety is related to the risk of impairment, either due directly to the disease, the effects from spread or the complications or side-effects of treatment. This may sometimes entail a risk to other members of the crew and to the safe operation of the ship. The general aspects of risk assessment will have a varying degree of significance depending on the diagnosis, type, stage, treatment and individual factors.

This group is so heterogeneous that it is not possible to give detailed guidelines for individual types of tumours. There is ongoing, rapid development in the treatment of many malignant conditions, and prognostic assessments based on older studies will therefore quickly become out-dated.

17.3.2 SPECIALIST ADVICE

Specialist advice must be solicited in all such cases. The declaration must in particular include the diagnosis, type, stage, treatment, effect of treatment, any side-effects and the prognosis for acute impairment which could lead to the loss of capabilities, risk to others on board, risk to the ship, and possibly risk to the person himself.

ASSESSMENT

As long as the condition is inconclusive, untreated or with an unclear prognosis, the person must be considered unfit. In the event of persistent disease leading to loss of or impaired capabilities, or the significant risk of complications as a result of the disease or the treatment, the right decision will be permanent unfitness. When the condition is considered resolved and check-ups are no longer necessary, an unrestricted medical certificate can be issued. Other factors to be considered include whether restrictions should be set for position, trade area or validity, or other special terms.

Reviewed 2015

17.4 D50-89 BLOOD AND BLOOD-FORMING ORGANS, IMMUNE MECHANISM

17.4.1 ANAEMIA

D50-59	Anaemia/Haemoglobinopathies Reduced exercise tolerance. Episodic red cell breakdown.	T – Distant waters, until haemoglobin normal and stable P – Severe recurrent or continuing anaemia or impairing symptoms from red cell breakdown that are untreatable	R, L – Consider restriction to near-coastal waters and regular surveillance if reduced haemoglobin level but asymptomatic	Normal levels of haemoglobin

There are many different causes of anaemia. Anaemia is generally secondary to other diseases, and these need to be investigated. The safety assessment can vary a lot depending on the underlying condition.

ASSESSMENT

As long as the anaemia is inconclusive, not completely investigated and/or symptomatic with impairment, unfitness is the correct conclusion.

An unrestricted medical certificate is only appropriate when the haemoglobin has normalised, the underlying cause has been eliminated or treated, there is no requirement for follow-up within the validity periodand no expectation of impairment of working capability during this time.

Reviewed 2015

17.4.2 SPLENECTOMY

D73	Splenectomy (history of surgery) Increased susceptibility to certain infections	T – Post surgery until fully recovered	R – Case-by-case assessment. Likely to be fit for near-coastal and temperate work but may need restriction on service in tropics.	Case-by-case assessment

An overview article on vascular complication can be found in "Blood"[115]

Following splenectomy the incidence of severe sepsis is approximately 8%. Another risk is thromboembolic disease, which can occur as frequently as in 35% of patients[116].

The risk of thrombosis following splenectomy was only recognised a few years ago. Portal vein thrombosis occurs in 5 to 37% of patients within the first two months after surgery, and the majority occur within the first two weeks[117] [118] [119] [120] [121].

The long-term risks are dependent on the original cause for the removal of the spleen, and will vary significantly. In a study of 8860 patients the prevalence of deep vein thrombosis was 32%, portal vein thrombosis 16% and pulmonary embolism 13%[122].

Pulmonary hypertension is another complication of splenectomy. In a study of patients with pulmonary hypertension the incidence of patients post splenectomy was 8.6 to 11.5% compared to 0 to 0.6% in the control group (patients with another pulmonary disease)[123] [124] [125].

Streptococcus Pneumoniae and Haemophilus Influenzae have a particularly high tendency of causing infection after splenectomy. The same applies to malaria and a few other tropical diseases[126]. This means that it is not advisable for people without a spleen to sail in the Tropics. It also means that is is important to vaccinate against Pneumococcus and Haemophilus Influenzae

ASSESSMENT

Patients post splenectomy should not be issued a medical certificate until it has been confirmed that their vaccination status is satisfactory. They should not serve in the Tropics due to the risk of tropical diseases, malaria in particular.

Reviewed 2014

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[115] Shelley E. Crary and George R. Buchanan; Vascular complications after splenectomyu for hematologic disorders. Blood 2009 114: 2861-2868.
[116] Petroianu A, Federal University of Minas Gerais, Brazil; «THE SPLEEN», Bentham Books, eISBN 978-1-60805-273-8
[117] Ikeda M, Sekimoto M, Takiguchi S, et al. High incidence of thrombosis of the portal venous system after laparoscopic splenectomy: a prospective study with contrast-enhanced CT scan. Ann Surg 2005;241(2):208-216.
[118] Hassn AM, Al-Fallouji MA, Ouf TI, Saad R. Portal vein thrombosis following splenectomy. Br J Surg 2000;87(3):362-373
[119] Chaffanjon PC, Brichon PY, Ranchoup Y, Gressin R, Sotto JJ. Portal vein thrombosis following splenectomy for hematologic disease: prospective study with Doppler color flow imaging. World J Surg 1998;22(10):1082-1086
[120] Stamou KM, Toutouzas KG, Kekis PB, et al. Prospective study of the incidence and risk factors of postsplenectomy thrombosis of the portal, mesenteric, and splenic veins. Arch Surg 2006;141(7):663-669
[121] Skarsgard E, Doski J, Jaksic T, et al. Thrombosis of the portal venous system after splenectomy for pediatric hematologic disease. J Pediatr Surg 1993;28(9):1109-1112
[122] Taher A, Isma'eel H, Mehio G, et al. Prevalence of thromboembolic events among 8860 patients with thalassaemia major and intermedia in the Mediterranean area and Iran. Thromb Haemost 2006;96(4):488-491
[123] Jais X, Ioos V, Jardim C, et al. Splenectomy and chronic thromboembolic pulmonary hypertension. Thorax 2005;60(12):1031-1034.
[124] Hoeper MM, Niedermeyer J, Hoffmeyer F, Flemming P, Fabel H. Pulmonary hypertension after splenectomy? Ann Intern Med 1999;130(6):506-509.
[125] Bonderman D, Jakowitsch J, Adlbrecht C, et al. Medical conditions increasing the risk of chronic thromboembolic pulmonary hypertension. Thromb Haemost 2005;93(3):512-516
[126] Kesinee Chotivanich, Rachanee Udomsangpetch, Rose McGready, Stephane Proux, Paul Newton, Sasithon Pukrittayakamee, Sornchai Looareesuwan and Nicholas J. White: Central Role of the Spleen in Malaria Parasite Clearance; J Infect Diseases Vol. 15, No 10, 1538-1541.

17.4.3 OTHER DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS

D50-89	Other diseases of the blood and blood-forming organs Spontaneous bleeding, reduced exercise tolerance, low resistance to infections	T – While under investigation P – Chronic coagulation disorders	Case-by-case assessment for other conditions	Case-by-case assessment

17.4.3.1 ANTIPHOSPHOLIPID SYNDROME

Some patients with antiphospholipid syndrome (APS) may have fluctuations in the INR and may have difficulty maintaining a stable INR. This is due to several factors, such as antiprothrombin antibodies, variations in thromboplastin reagents and lupus anticoagulants. The seafarer's doctor therefore has to be certain that the INR in the person concerned really is stable before the person can be issued a restricted or unrestricted certificate based on individual risk assessment.

In a series of 1000 patients with either primary or secondary APS[127] the following occurrences were found:

- Deep vein thrombosis 32%
- Thrombocytopenia 22%
- Livedo reticularis 20%
- Stroke 13%
- Superficial thrombophlebitis 9%
- Pulmonary embolism 9%
- Fetal loss 8%
- Transient ischemic attack 7%
- Hemolytic anaemia 7%

The condition is thus no trivial disease when it comes to the risk of complications. There are, however, large variations of APS, dependent on subtype and which antibodies are detected. The risk can therefore not be determined based on the diagnosis alone. More detailed medical information is necessary in order to determine the individualised risk.

The prognosis for patients with APS is dependent upon the clinical manifestations that lead to diagnosis. As an example, the prognosis is particularly poor during the initial episode of care when the patient presents with multisystem disease as seen in the catastrophic antiphospholipid

[127] Cervera R, Piette JC, Font J, et al. Antiphospholipid syndrome: clinical and immunologic manifestations and patterns of disease expression in a cohort of 1,000 patients. Arthritis Rheum 2002; 46:1019.

syndrome (CAPS). The best data on morbidity and mortality are those from an international retrospective study of 1000 patients who were seen during the period of 1999 to 2004[128]. In this group which consisted of 82% females (98.5% Caucasian), the mean age at entry was 42 years, and 53% had primary APS (36% with systemic lupus erythematosus (SLE)). Among these patients, 77% received treatment, 54% with oral anticoagulants and 45% with aspirin. Recurrent thrombotic or thromboembolic events occurred in 166 patients (strokes 23, transient ischemic attacks 23, deep vein thrombosis 21, pulmonary embolism 21 and myocardial infarction). Patients with thromboses or thromboembolic events had been receiving treatment with oral anticoagulants with a target INR of 2.0 to 3.0 (n = 90) or aspirin (n = 49), or were untreated (n = 27).

Other morbid events included seizures (n = 17), heart valve thickening or dysfunction (n = 17), and microangiopathic hemolytic anaemia (n = 9).

Obstetric outcomes in 77 women who had one or more pregnancies included live births (n = 63), pre - eclampsia or eclampsia (n = 8), early pregnancy loss (n = 18), premature birth (n = 28), and intrauterine growth retardation (n = 11).

Mortality in this cohort was 5.3%. Causes of death included bacterial infection (n = 11), myocardial infarction (n = 10), stroke (n = 7), haemorrhage (n = 6), CAPS (n = 5), and pulmonary embolism (n = 6).

Patients who survive the initial episode remain at risk for recurrent events.

Treatments with oral anticoagulation (or aspirin) may reduce, but does not eliminate, the risk of recurrent thrombotic, thromboembolic, or obstetrical adverse outcomes.

ASSESSMENT

This means that the diagnosis of APS usually is not compatible with service on board ships.

17.4.4 INHERITED TROMBOPHILIA

Inherited thrombophilia is a genetic tendency to venous thromboembolism. The Factor V Leiden mutation is the most common cause (40 - 50% of cases). Prothrombin gene mutation and deficiencies in Protein S, Protein C and antithrombin account for most of the remaining cases, whilst rare causes include the dysfibrinogenemias[129]. The total incidence of an inherited

[128] Cervera R, Khamashta MA, Shoenfeld Y, Camps MT, Jacobsen S, Kiss E, Zeher MM, Tincani A, Kontopoulou-Griva I, Galeazzi M, Bellisai F, Meroni PL, Derksen RH, de Groot PG, Gromnica-Ihle E, Baleva M, Mosca M, Bombardieri S, Houssiau F, Gris JC, Quérél, Hachulla E, Vasconcelos C, Roch B, Fernández-Nebro A, Piette JC, Espinosa G, Bucciarelli S, Pisoni CN, Bertolaccini ML, Boffa MC, Hughes GR, Euro-Phospholipid Project Group (European Forum on Antiphospholipid Antibodies)Morbidity and mortality in the antiphospholipid syndrome during a 5-year period: a multicentre prospective study of 1000 patients. Ann Rheum Dis. 2009;68(9):1428).

[129] Mateo J, Oliver A, Borrell M, Sala N, Fontcuberta J: Laboratory evaluation and clinical characteristics of 2,132 consecutive unselected patients with venous thromboembolism--results of the Spanish Multicentric Study on Thrombophilia (EMET-Study). Thromb Haemost. 1997;77(3):444

thrombophilia in individuals with a deep vein thrombosis (DVT) ranges from 24 to 37 % overall compared with approximately 10% in controls.

The lifetime probability of developing thrombosis for persons with thrombophilia compared with those with no defect is as follows: 8.5 time higher for protein S deficiency, 8.1 for antithrombin deficiency, 7.3 for protein C deficiency and 2.2 for factor V Leiden[130] .

17.4.4.1 PROTEIN C DEFICIENCY

There are two major subtypes of Protein C deficiency. Type I deficiency is the more common, with the plasma Protein C concentration being approximately 50% of normal. In type II deficiency plasma concentration is normal, but the functional activity of Protein C is decreased.

Three clinical syndromes are associated with Protein C deficiency:

1) Deep vein thrombosis (DVT)
2) Neonatal purpura fulminans (only in newborns)
3) Warfarin-induced skin necrosis in certain heterozygous teenagers and adults.

The lifetime probability of developing thrombosis compared with those with no defect is 7.3 times higher for carriers of Protein C deficiency (8.5 for Protein S deficiency, 8.1 for Antithrombin III deficiency and 2.2 for Factor V Leiden mutation)[131].

The initial episode of DVT is apparently spontaneous in approximately 70% of cases, whilst the remaining 30% are connected with specific risk factors eg pregnancy, parturition, oral contraceptives, surgery, trauma.

Most patients are asymptomatic until their twenties, with increasing numbers experiencing DVT as they reach the age of 50. The median age of initial DVT is 45 years in unselected patients and 30 years in members of thrombophilia families[132].

Approximately 60% of patients develop recurrent venous thrombosis and about 40 percent have signs of pulmonary embolism[133].

[130] Martinelli I, Mannucci PM, De Stefano V, et al. Different risks of thrombosis in four coagulation defects associated with inherited thrombophilia: a study of 150 families. Blood 1998; 92:2353.
[131] Koster T, Rosendaal FR, Briët E, van der Meer FJ, Colly LP, Trienekens PH, Poort SR, Reitsma PH, Vandenbroucke JP: Protein C deficiency in a controlled series of unselected outpatients: an infrequent but clear risk factor for venous thrombosis (Leiden Thrombophilia Study). Blood. 1995;85(10):2756
[132] Lensen RP, Rosendaal FR, Koster T, Allaart CF, de Ronde H, Vandenbroucke JP, Reitsma PH, Bertina RM: Apparent different thrombotic tendency in patients with factor V Leiden and protein C deficiency due to selection of patients. Blood. 1996;88(11):4205
[133] Broekmans, AW, Bertina, RM. Protein C. In: Recent Advances in Blood Coagulation, volume four, Poller, L (Ed), Churchill Livingstone, New York 1985. p.117

Treatment with anticoagulants, usually warfarin, gives good results. Long-term treatment with warfarin reduces the risk of recurrent thromboses to 2.6% over a four-year period, whilst short-term treatment (six months) gives a risk of recurrence of 21% over a four-year period[134].

Warfarin-induced skin necrosis typically occurs during the first several days of warfarin therapy.

Warfarin therapy reduces functional and immunologic measurements of Protein C, making it difficult to diagnose individuals by blood testing after warfarin therapy is initiated.

17.4.5 PROTEIN S DEFICIENCY

Protein S deficiency leads to an increased risk of thrombosis.

The clinical presentation of patients with heterozygous Protein S (PS) deficiency is similar to that of Antithrombin or Protein C deficiency. In patients found to be heterozygous (gene material in both DNA chains) for Protein S deficiency, 55% developed DVT, and it was recurrent in 77% of these cases. Most patients had various combinations of DVT (74%), superficial thrombophlebitis (72%) and pulmonary embolism (38%), either in succession or simultaneously. The age at the first thrombotic event ranged from 15 to 68 years, with mean age 28 years, and at age 35 years the probability to be still free of thrombosis was only 32%. 56% of the thrombotic events were not preceded by a precipitating condition. In these respects Protein S deficiency is similar to Protein C deficiency[135].

17.4.6 FACTOR V LEIDEN MUTATION

The Factor V Leiden mutation is the most common cause of inherited thrombophilia , accounting for 40 - to 50% of cases. It exists in both heterozygous and homozygous form.

Heterozygotes account for 99% of patients with the Factor V Leiden mutation. The prevalence varies from on average 0.45% in Asians to 5.3% in Caucasians, but there are examples of local prevalence as high as 15% incertain parts of Sweden, Greece and Lebanon[136]. In Norway we estimate that approximately 8% of the population are carriers of the mutation.

Homozygotes account for approximately 1% of patients, but are at much greater risk of developing clinical disease.

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[134] Schulman S, Granqvist S, Holmström M, Carlsson A, Lindmarker P, Nicol P, Eklund SG, Nordlander S, Lärfars G, Leijd B, Linder O, Loogna E: The duration of oral anticoagulant therapy after a second episode of venous thromboembolism. The Duration of Anticoagulation Trial Study Group. N Engl J Med. 1997;336(6):393

[135] Engesser L, Broekmans AW, Briët E, Brommer EJ, Bertina RM : Hereditary protein S deficiency: clinical manifestations. Ann Intern Med. 1987;106(5):677

[136] Rees DC, Cox M, Clegg JB: World distribution of factor V Leiden. Lancet. 1995;346(8983):1133

The risk connected with Factor V Leiden mutation is related to the increased incidence of deep vein thrombosis, with or without pulmonary embolism. There is also an increased risk of cerebral, mesenteric and portal vein thrombosis[137].

Approximately 10 - 26% of patients with venous thrombosis are carrier of the Factor V Leiden mutation. The risk of deep vein thrombosis in homozygous factor V Leiden mutants is less than in homozygous protein C or protein S mutants and the relative risk of new venous thromboses in heterozygous Leiden V mutants without additional risks is quite low. Even though it is seven times as high as in the general population, the yearly risk is only 0.06%, which gives an overall risk within a normal certificate period of 0.12%. This is seen as a very low likelihood. In homozygotes the risk is approximately 80 times as high as for the general population, and this results in a yearly likelihood of 0.5-1.0% (very low likelihood), or 1.0-2.0% over a two year period (very low/low likelihood).

It is unclear how large the likelihood of a second episode of thrombosis is for Leiden mutants. The general view is that there is is an increased likelihood, but it is difficult to set a reliable figure on it.

Episodes of thrombosis rarely occur spontaneously, they are most often triggered by a combination of Leiden mutation and other risk factors, such as trauma, surgery, immobilisation, prolonged air travel, oestrogen therapy, obesity, etc. Anticoagulation therapy is therefore used in risk situations, i.e. surgical procedures, long-term immobilisation. It is also considered on an individual basis in other situations, such as e.g. pregnancy. In recurrent thrombosis lifelong warfarin treatment is usually recommended.

Reviewed 2015

17.5 E00-90 ENDOCRINE, NUTRITIONAL AND METBOLIC DISEASES

17.5.1 DIABETES MELLITUS TYPE I

E10	Diabetes – Insulin-dependent Acute impairment from hypoglycaemia. Complications from loss of blood glucose control. Increased likelihood of visual, neurological and cardiac problems.	T – From start of treatment until stabilised P – If poorly controlled or not compliant with treatment. History of hypoglycaemia or loss of hypoglycaemic awareness. Impairing complications of diabetes.	R, L – Subject to evidence of good control, full compliance with treatment and recommendations and good hypoglycaemia awareness. Fit for near-coastal duties without solo watchkeeping. Time limited until next specialist check-up. Must be under regular surveillance.	Not applicable

[137] Stolz E, Kemkes-Matthes B, Pötzsch B, Hahn M, Kraus J, Wirbartz A, Kaps M: Screening for thrombophilic risk factors among 25 German patients with cerebral venous thrombosis. Acta Neurol Scand. 2000;102(1):31

Diabetes mellitus types I and II principally have the same associated problems although the prevalence and severity vary.

The long-term complications are an increased occurrence of cardiovascular disease, neuropathy, retinopathy and peripheral neuropathy.

High blood sugar (hyperglycaemia) is a threat to alertness, concentration and reaction times and will cause a reduction or loss of consciousness as it increases.

Low blood sugar (hypoglycaemia) is generally a larger safety risk on board ships compared to hyperglycaemia. Severe hypoglycaemia can lead to unconsciousness and/or seizures, and can cause temporary or permanent brain damage and even death. The symptoms of hypoglycaemia usually occur suddenly and can include cold sweats, cold and pale skin, exhaustion, nervousness or tremors, anxiety, abnormal tiredness or weakness, confusion, lack of concentration, drowsiness, feeling of extreme hunger, eye complications, headaches, nausea and palpitations.

The average patient with type I diabetes suffers many episodes of asymptomatic hypoglycaemia, where plasma glucose concentration can be below 3 mmol/L (2.8 to 3.3) for as much as 10% of the time[138].

On average they suffer two episodes of symptomatic hypoglycaemia per week, thousands of episodes over a lifetime with diabetes, and on average one serious episode per year. Severe hypoglycaemic events have been reported to range from 62 to 170 episodes per 100 patient years in type 1 diabetes[139].

Insulin therapy is imperative in type I diabetes. It can be a challenge to set the level of insulin so that hyperglycaemia and hypoglycaemia is avoided. This requires the patient to be competent in self-monitoring and to test frequently.

17.5.1.1 LONG-ACTING INSULIN

Levemir (detemir) is a long-acting insulin and side effects are usually caused by the pharmacological effects of the insulin. The total percentage of treated patients expected to experience side effects is estimated at 12%. Hypoglycaemia is the most commonly reported side effect and can occur if the insulin dosage is too high in relation to the insulin need. In studies severe hypoglycaemia has been found in approximately 6% of patients[140].

[138] Cryer, PE. Hypoglycemia in diabetes. Pathophysiology, prevalence, and prevention. American Diabetes Association, Alexandria VA, 2009
[139] Cryer PE, Axelrod L, Grossman AB, Heller SR, Montori VM, Seaquist ER, Service FJ, Endocrine Society: Evaluation and management of adult hypoglycemic disorders: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2009;94(3):709
[140] Source: Felleskatalogen, Levemir® monograph

RISK ASSESSMENT FOR INSULIN-TREATED TYPE I DIABETES

The general prevalence of hypoglycaemia of 6% means that it is not acceptable for persons with type I diabetes and ongoing treatment with long-acting insulin to have watch-keeping duties or a safety function. It is also debatable whether the risk is too high for other duties on board, unless it has been documented that the person is in better control of his or her diabetes than the average person. Sailing in a trade area where there are no acceptable evacuation possibilities is not advisable. Upon documented stable control without known hypoglycaemic events, service on board ships in a trade area where the person at all times can be reached by helicopter with evacuation capacity may be acceptable in a position without watch-keeping duties or a safety function.

Reviewed 2015

17.5.2 DIABETES MELLITUS TYPE II

E11-14	Diabetes – Non-insulin treated, on other medication Progression to insulin use, increased likelihood of visual, neurological and cardiac problems.	T – Distant waters and watchkeeping until stabilised P – Impairing complications of diabetes	R – Until stabilised: Near-coastal waters. Non-watchkeeping duties. R – If minor side effects from medication or when using sulphonylureas: Near-coastal waters. Non-watchkeeping duties. L – Time limited if compliance with treatment and advice poor or medication needs frequent review. Check diet, weight and cardiovascular risk.	When stabilised, in the absence of impairing complications
E11-14	Diabetes – Non-insulin treated, on diet Progression to insulin use, increased likelihood of visual, neurological and cardiac problems.	T – Distant waters and watchkeeping until stabilised	R – Until stabilised: Near-coastal waters. Non-watchkeeping duties. L – Time limited if compliance with advice poor or there is need of frequent controls. Check diet, weight and vascular risk factor control.	When stabilised, in the absence of impairing complications

The global prevalence of diabetes has been estimated at 2.8%[141] and in the Western world the prevalence has increased in recent years in parallel with the increase in BMI. The lifetime risk for diabetes is now 30% for men and 35% for women in the USA and Type II diabetes accounts for over 90% of the cases. Patients with diabetes have a very high likelihood of having co-occurring obesity, lipid abnormalities and high blood pressure[142] [143].

A number of different conditions occur more frequently in people with type II diabetes, e.g. renal failure, retinopathy, amputations, myocardial infarction, heart failure, stroke, infections, hypoglycaemia related to treatment and hyperglycaemia related to insufficient treatment, sometimes with electrolyte imbalance.

More than 65% of adults with diabetes die of a myocardial infarction, stroke or peripheral arterial disease[144].

The lifetime risk of end-stage complications of diabetes is around 5% for end-stage renal disease, <5% for blindness and approximately 7-8% for amputations[145].

Aggressive and effective clinical treatment of blood pressure, lipids and blood sugar,the use of Aspirin and abstinence from tobacco can reduce the risk of serious cardiovascular complications by more than 50%.

Chronic renal failure occurs in approximately 40% of patients over a certain time, with a lifetime risk of renal failure of approximately 5%. The risk of chronic renal failure increases with uncontrolled blood pressure and blood glucose increasing the risk of cardiovascular disease between 4 and 10-fold. The target blood pressure for diabetic patients with renal failure is a systolic blood pressure of < 120 mmHg.

The acute safety risk is connected to a blood sugar that is too high or too low which could both compromise judgement, reaction times, concentration, alertness and decisiveness. Some long-term complications could also entail acute problems, such as stroke and myocardial infarction.

Many diabetics also have impaired functional ability, which can become evident in physical testing.

There are such large variations in the functional ability of diabetics that it is not possible to make an adequate assessment based on statistics and general considerations. An individual

[141] Wild S, Roglic G, Green A, et al. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care. 2004;27:1047-1053

[142] National Collaborating Centre for Chronic Conditions. Type 2 diabetes: national clinical guideline for management in primary and secondary care (update). London: Royal College of Physicians, 2008. http://www.nice.org.uk/nicemedia/pdf/CG66diabetesfullguideline.pdf (last accessed 25 March 2010 - Narayan KM, Boyle JP, Thompson TJ, et al. Lifetime risk for diabetes mellitus in the United States. JAMA. 2003;290:1884-1890

[143] DeFronzo RA. Pathogenesis of type 2 diabetes mellitus. Med Clin North Am. 2004;88:787-835

[144] Haffner SM, Lehto S, Ronnemaa T, et al. Mortality from koronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med. 1998;339:229-234

[145] National Diabetes Data Group. Diabetes in America. 2nd edition. NIH Publication No. 95-1468. http://diabetes.niddk.nih.gov/dm/pubs/america (last accessed 25 March 2010

assessment is imperative in order to determine whether an person constitutes an unacceptable safety risk or whether he fulfils the requirements of section 1 of the Regulations.

Reviewed 2015

17.5.3 OBESITY AND OVERWEIGHT

E65-68	Obesity/abnormal body mass – high or low Accident to self, reduced mobility, reduced exercise tolerance. Increased likelihood of diabetes, cardiovascular diseases and arthritis.	T – If safety-critical duties cannot be performed, physical capability or exercise tolerance is poor. Measures have been implemented to improve physical capability and exercise tolerance with a prospect of improvement. P – If safety-critical duties cannot be performed, physical capability or exercise tolerance is poor. Attempts to improve the situations have failed. NB: Body mass index (BMI) is a useful indicator of when additional assessment of physical assessment is needed. BMI should not form the sole basis for decisions on unfitness. In the event of BMI over 35, testing is mandatory.	R, L – Time limited and restricted to near-coastal waters or to restricted duties if the person is unable to perform certain tasks but able to meet routine and emergency capabilities for assigned safety-critical duties.	Physical capability and exercise tolerance performance is average or better, weight steady or reducing and no co-morbidity

17.5.3.1 DEFINITION OF OBESITY AND OVERWEIGHT

The WHO defines overweight and obesity based on the following criteria:

- UNDERWEIGHT: BMI < 18.5,
- NORMAL WEIGHT: BMI 18.5-24.9,
- OVERWEIGHT: BMI > 25,
- OBESE CLASS I: BMI > 30,
- OBESE CLASS II: BMI > 35,
- OBESE CLASS III (morbid obesity): BMI > 40.

17.5.3.2 USE OF BMI AS A SCREENING METHOD

BMI is suitable for finding people to examine more closely, as this is a good measurement of body size.

It is, however, important to remember that this is just an index, not a conclusion. There are, for example, large differences between Caucasians and Asians, and it is being debated whether other limits should be used in Asia.

BMI is a poor predictor of cardiovascular risk related to being overweight or obese, but it is, however, a good predictor of impaired working capacity, and a high BMI is a good reason to examine more closely. (ROC value is 0.64 for males and 0.59 for females.)

There are a variety of indices which can be used for evaluating cardiovascular risk in overweight and obese patients including waist circumference and Conicity index (CI)[146]. (Index C ROC value for males is 0.80, females 0,75, whilst Waist Circumference ROC value for males is 0.73 and females 0.66.)

For all practical purposes the seafarer's doctor will be able to use BMI in order to find the people who there is reason to examine more closely.

17.5.3.3 SAFETY RISK IN OVERWEIGHT/OBESITY - IMPAIRED PHYSICAL CAPABILITIES

Being overweight or obese can lead to impaired physical capabilities so that the physical ability requirements set out in STCW Code A table A-I/9 are not satisfied.

The safety risk to other members of the crew and to the operation of the ship is related to the increased risk of being overweight or obese and can involve:

1. Overweight and obese persons can have difficulty performing their routine and emergency duties
2. They have increased risk of other diseases which could cause sudden indisposition (myocardial infarction, stroke, etc.)
3. They have increased risk of falling ill in a way which requires evacuation from the ship or that the ship must deviate from its planned route to continue to another port
4. They may have to pass on their duties to others on board, so that these crew members get an increased working load

With regards to the possibility of being evacuated, the waist circumference and actual weight are factors which could be significant for the ability to

1. be carried/moved by others
2. be strapped securely onto a stretcher for evacuation and
3. get into position (and possibly be secured with seat belts) in a lifeboat.

[146] Haun DR, Pitanga FJ, Lessa I. Universidade de Trás-os-Montes e Alto Douro: Waist-height ratio compared to other indicators of obesity as predictors of high koronary risk. Rev Assoc Med Bras. 2009 Nov-Dec;55(6):705-11

Body weight exceeding 120 kg and a waist circumference exceeding 115 cm could quickly lead to problems with regard to these questions.[147].

A number of disorders have an increased prevalence in overweight and obese patients. These include: hypertension, glucose intolerance, dyslipidemia, with increased risk of cardiovascular disease, diabetes, renal disease and obstructive sleep apnoea. There is also an increased risk of colon, breast, oesophageal, uterine, ovarian, kidney and pancreatic cancer. In addition there is potential physical disability which increases as the weight increases and which can be present to a varying degree. The load on the musculoskeletal system leads to more wear and tear than in others and there is an increased risk of gout, depression, hernia, gallstone and varicosity.

The increased risk for heart failure is estimated at 5% for men and 7% for women for each BMI increase of 1 (kg/m^2)[148]. Total mortality increases by 30% for each 5 point increase of BMI above 25[149].

The increasing mortality we see when the BMI increases is mainly related to cardiovascular disease. Mean survival is reduced by 2 to 4 years at a BMI of 30-35 kg/m^2 and reduced by 8 to 10 years at a BMI of 40-45 kg/m^2 - this is comparable to the effects of smoking. However, it has also been found that the risks related to being overweight and obese decrease with age. For persons over the age of 55, no particular increase in mortality is found[150].

Being moderately overweight is found to have little significance as a risk factor as long as the physical fitness is maintained[151].

Both a reduction in mortality and a significant improvement in conditions related to being overweight and obese, such as hypertension, insulin resistance and an unfavourable lipoprotein profile, can be achieved through improved physical fitness, independent of weight loss[152] [153].

The general risk consideration alone cannot be used to decide if a person is fit or not. There are large individual variations and an individual risk assessment of the ability to perform ordinary and emergency duties and an estimation of the individual risk for becoming unwell, having to hand over duties to others, having to be treated on board or be evacuated is necessary in order to decide whether a health declaration can be issued.

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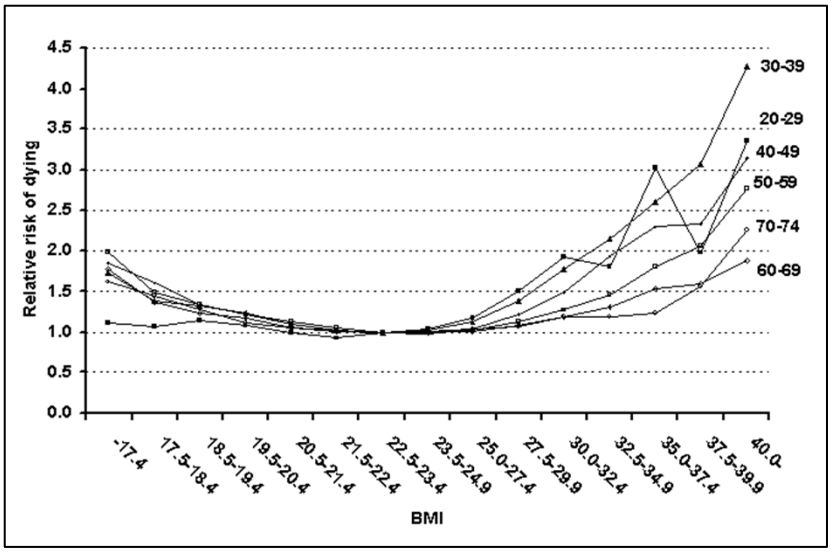
[147] Guide to Regulations concerning health requirements for persons working on installations in the petroleum industry at sea, section 16.6.2 – page 32. (IS-1879)
[148] Kenchaiah S, Evans JC, Levy D, et al. Obesity and the risk of heart failure. N Engl J Med 2002; 347: 305-13).
[149] Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. Lancet 2009; 373: 1083-96
[150] Bender R, Føckel CH, Trautner C, Spraul M, Berger M. Effect of Age on Excess Mortality in Obesity. JAMA 1999; 281:1498-1504
[151] Gaesser GA, Rich RG. Effects of high- and low-intensity exercise training on aerobic capacity and blood lipids. Med Sci Sports Exerc 1984; 16: 269-74
[152] Martin WH 3d, Dalsky GP, Hurley BF, Matthews DE, Bier DM, Hagberg JM et al. Effect of endurance training on plasma free fatty acid turnover and oxidation during exercise. Am J Physiol 1993; 265: E708 - 14
[153] Bergman BC, Brooks GA. Respiratory gas-exchange ratios during graded exercise in fed and fasted trained and untrained men. J Appl Physiol 1999; 86: 479 - 87

When the person is found to be obese, the seafarer's doctor needs to record the person's physical capabilities, assess his or her body size and look at the risk of disease related to obesity.



Ref: Engeland A, Bjørge T, Selmer RM, Tverdal A. Height and body mass index in relation to total mortality. Epidemiology 2003; 14: 293-9. Hans Th Waaler: height, weight and mortality. The Norwegian Experience. Rapport 4, 1984, Statens institututt for folkehelse.

17.5.3.4 EXAMINATION OF OVERWEIGHT/OBESE PERSONS

When the person is found to be obese, it is important to consider and record the following factors:

- Physical abilities:
  - Mobility
  - Strength
  - Fitness
- No decision has been made on which method to use for testing fitness. The method to be used should be standardised, so that it is comparable and verifiable, and results can be measured in the form of one or more values. The Chester Step test, Harvard step test, treadmill test, exercise bicycle test, stress test ECG, oxygen intake are all applicable test methods.
- Cardiovascular risk:
  - Diabetes and lipid abnormalities
  - Concurrent smoking?
  - Family risk?
- Actual body size:
  - Will the person be able to get through a manhole?
  - Will the person be able to get through a helicopter window?
  - Will he/she be able to fasten their seatbelt in a free-fall lifeboat?
  - Will he/she be able to get into a survival suit?
  - Will it be possible to get him/her onto a stretcher and evacuated?
  - Will people be able to carry him/her if they get sick or injured?

17.5.3.5 THE ASSESSMENT OF PATIENTS WHO HAVE UNDERGONE BARIATRIC SURGERY

Bariatric surgery is becoming more popular in many nations and is indicated for patients with a BMI over 40 or a BMI over 35 with significant comorbidities[154] in whom non operative therapies have not been successful. The procedures may achieve restriction, malabsorbtion or both. Once the individual has recovered from the surgery one of the main ongoing risks is that of hernia[155]. Hernia may be incisional or internal, the latter often resulting in small bowel obstruction.

Incisional hernias occur at a higher incidence after open gastric bypass (GBP) at a rate of about 20 %[156]. Laparoscopic gastric bypass (LGBP) has a lower rate (0.2%) of incisional hernias[157].

Internal hernias, on the other hand, occur more frequently in LGBP than in the open procedure and this is a significant clinical problem, since internal hernia is the most common cause of small bowel obstruction (SBO) after LGBP. Retrospective reviews have found the incidence of SBO after LGBP to be between 1.8 and 9.7 % and patients most commonly present with abdominal pain, and may also have symptoms of small bowel obstruction. The time of presentation varies greatly and may occur within one week of the initial operation or up to three years postoperatively. However, the majority of cases occur between 6 and 24 months postoperative[158]. Hence for the first two years the risk of small bowel obstruction after LGBP is moderate and it is advisable to consider a restriction to coastal waters where medical care can be reached quickly in case of the onset of symptoms.

Reviewed 2014

[154] Burguera B, Agusti A, Arner P, Baltasar A, Barbe F, Barcelo A, Breton I, Cabanes T, Casanueva FF, Couce ME, Dieguez C, Fiol M, Fernandez Real JM, Formiguera X, Fruhbeck G, Garcia Romero M, Garcia Sanz M, Ghigo E, Gomis R, Higa K, Ibarra O, Lacy A, Larrad A, Masmiquel L, MoizéV, Moreno B, Moreiro J, Ricart W, Riesco M, Salinas R, Salvador J, Pi-Sunyer FX, Scopinaro N, Sjostrom L, Pagan A, Pereg V, Sánchez Pernaute A, Torres A, Urgeles JR, Vidal-Puig A, Vidal J, Vila M. Critical assessment of the current guidelines for the management and treatment of morbidly obese patients; J Endocrinol Invest. 2007 Nov;30(10):844-52.

[155] http://bariatrictimes.com/internal-hernia-after-laparoscopic-gastric-bypass-a-review-of-the-literature/comment-page-1/

[156] Sugerman HJ, Kellum JM, Jr., Reines HD, et al. Greater risk of incisional hernrecurrence with prefascial polypropylene mesh. Am J Surg 1996;171(1):80–4.

[157] Rosenthal RJ, Szomstein S, Kennedy CI, Zundel N. Direct visual insertion of primary trocar and avoidance of fascial closure with laparoscopic Roux-en-Y gastric bypass. Surg Endosc2007;21(1):124–8.

[158] Capella RF, Iannace VA, Capella JF. Bowel obstruction after open and laparoscopic gastric bypass surgery for morbid obesity. J Am Coll Surg 2006;203(3):328–35.

17.5.4 OTHER ENDOCRINE CONDITIONS

E 00-90 Not listed separately	Other endocrine and metabolic disease (thyroid, adrenal including Addison's disease, pituitary, ovaries, testes) Likelihood of recurrence or complications	T – Until treatment established and stabilized without adverse effects P – If continuing impairment, need for frequent adjustment of medication or increased likelihood of major complications	R, L – Case-by-case assessment with specialist advice if any uncertainty about prognosis or side effects of treatment. Need to consider likelihood of impairing complications from condition or its treatment, including problems taking medication, and consequences of infection or injury while at sea	If medication stable with no problems in taking at sea and surveillance of conditions infrequent, no impairment and very low likelihood of complications Addison's disease: The risks will usually be such that an unrestricted certificate should not be issued

17.5.4.1 ADDISON'S DISEASE

Symptoms and sign on adrenal insufficiency depends on how quickly adrenal function is lost, the degree of production of mineralocorticoids and the degree of stress. The onset is often graduous and may be asymptomatic, especially if an Addison crisis (acute adrenal failure) is not elicited by other disease or stress.

Such crisis may occur in the following situations:

- In a patient earlier undiagnosed with primary adrenal failure, exposed to serious infection or other acute serious stress.
- In a patient with known adrenal failure, who fails to increase the dosage of glucocorticoids during infection or stress, or who suffers from prolonged vomiting following gastrointestinal infection or other gastrointestinal disease.
- Following bilateral adrenal infarction ord bilateral adrenal haemorrhage.
- On rare occasions it is also seen in secondary or tertiary adrenal failure (following acute lack of cortisol in infarction of the pituitary gland)
- In patiens where sudden discontinuation of clucocorticoid therapy casues secondary adrenal failure.

Symptoms of acute adrenal failure / Addison crisis are circulatory shock, but patients often suffer from reduced appetite, nausea, vomiting, abdominal pain, bodily weakness, increased tiredness, inertia, fever, confusion or coma. Blood pressure is low (sometimes also blood glucose) and in some cases serious electrolyte disturbances.

Addison crisis urgently needs intravenous glucocorticoid and electrolyte treatment. Immediate hospitalization is necessary. The condition stabilizes in two or three days.

Following trauma, serious stress or during infection, doses need to be increased. Sometimes intravenous infusion is necessary to compensate for the body's own reduced production of cortisol.

This is the reason why Addison's disease generally is not regarded compliant with service in the bridge watch or in a safety function, and should be carefully considered also in all other positions on board ship.

If a medical certificate is issued, the trade area should be restricted to near coastal (within reach of helicopter with a MEDEVAC capability).

Reviewed 2016

17.6 F 00-99 MENTAL, COGNITIVE AND BEHAVIOURAL DISORDERS

17.6.1 ALCOHOL ABUSE AND DEPENDENCY

F 10	Alcohol abuse (dependency) Recurrence, accidents, erratic behaviour/ safety performance	T – Until investigated and stabilized and criteria for fitness met. Until one year after initial diagnosis or one year after any relapse P – If persistent or there is co-morbidity likely to progress or recur while at sea	R, L – Time limited, not to work as master in charge of vessel or without close supervision and continuing medical monitoring, provided that: treating physician reports successful participation in rehabilitation programme; and there is an improving trend in liver function tests	After three years from end of last episode without relapse and without co-morbidity

17.6.1.1 SAFETY RISKS UNDER INFLUENCE OF ALCOHOL

Alcohol is implicated in the morbidity and mortality from trauma[159], mainly related to the Blood Alcohol Concentration (BAC). In Norway, the legal BAC limit for driving is 0.02 percent. This may vary from country to country, and in most of the United States, the legal BAC limit for driving is 0.08 percent. Simulated driving ability is impaired with BACs as low as 0.02 percent[160] and the risk of involvement in a collision while driving doubles at a BAC of only 0.05 percent[161]. Alcohol intake is also associated with a greater severity of injury in motor vehicle accidents[162]. The risk of driving accidents is greatest in the first two years of exposure to alcohol[163]. Impairment in

[159] Vinson DC, Mabe N, Leonard LL, et al. Alcohol and injury. A case-crossover study. Arch Fam Med 1995; 4:505
[160] LOOMIS TA, WEST TC. The influence of alcohol on automobile driving ability; an experimental study for the evaluation of certain medicological aspects. Q J Stud Alcohol 1958; 19:30.
[161] Voas RB. Issues in cross-national comparisons of crash data. Addiction 1993; 88:959.
[162] Híjar M, Flores M, López MV, Rosovsky H. Alcohol intake and severity of injuries on highways in Mexico: a comparative analysis. Addiction 1998; 93:1543.
[163] Asch T, Levy D. The minimum legal drinking age and traffic fatalities. Rutgers University, NIAAA 1986.

simulated flying ability is demonstrated at the level of 0.04 percent for pilots[164]. Injuries during operation of boats[165], bicycles[166] [167] and snowmobiles[168] are related to alcohol use.

A number of maritime accidents has happened due to crew being under the influence of alcohol[169] [170].

Falls, drowning, burns, hypothermia, and occupational injuries are more prevalent in drinkers, particularly heavy drinkers[171].

An analysis of 1150 respondents from the 1990 National Alcohol Survey (USA) suggests that the risk of injury increases with even one drink daily[172]. In summary, no safe level of alcohol use exists for the use of potentially dangerous equipment.

17.6.1.2 LEGAL ISSUES

The Norwegian Maritime Code No 39 of 24th June 1994, as latest amended by Act No 16 of 9th May 2014, Section 143, sets the maximum level for individuals in the bridge watch or who perform safety duties and on ships of 15 m length and above of BAC to 0.02 percent or an amount of alcohol in the body having the potential to produce such a BAC. Breath alcohol concentration shall not be more than 0.01 percent. In fact this means that persons on watch cannot consume alcohol at all.

17.6.1.3 MORBIDITY AND MORTALITY

There is a considerable safety risk connected to individuals drinking only in their free periods on shore, or who is under treatment for alcohol abuse and dependency.

The seafarer's doctor must assess the likelihood for concomitant somatic diseases as well as psychiatric diseases and cognitive diseases, which are known to follow heavy alcohol consumption.

[164] Morrow D, Leirer V, Yesavage J, Tinklenberg J. Alcohol, age, and piloting: judgement, mood, and actual performance. Int J Addict 1991; 26:669.
[165] Mengert, P, Sussman, ED, DiSario, R. A study of the relationship between the risk of fatality and blood alcohol concentration of recreational boat operators. Report CG-D-09-92. U.S. Coast Guard, Washington DC 1992.
[166] Olkkonen S, Honkanen R. The role of alcohol in nonfatal bicycle injuries. Accid Anal Prev 1990; 22:89.
[167] Li G, Baker SP, Smialek JE, Soderstrom CA. Use of alcohol as a risk factor for bicycling injury. JAMA 2001; 285:893.
[168] Waller J, Lamborn K. Snowmobiling: Characteristics of owners, patterns of use and injuries. Accid Anal Prev 1975; 7:213.
[169] EMSA: MARITIME ACCIDENT REVIEW 2009
[170] Drazen Cuculica, Alan Bosnara, Valter Stembergaa, Miran Cokloa, Nebojsa Nikolicb, Emina Grgurevicc: Interpretation of blood alcohol concentration in maritime accidents – A case report. Forensic Science Internatioanl Supplement Series 2009; Vol 1:1; 36-37
[171] Romelsjo A. Alcohol consumption and unintentional injury, suicide, violence, work performance, and inter-generational effects. In: Alcohol and Public Policy: Evidence and Issues, Holder HD, Edwards G (Eds), Oxford University Press, New York 1995. p.114.
[172] Cherpitel CJ, Tam T, Midanik L, et al. Alcohol and non-fatal injury in the U.S. general population: a risk function analysis. Accid Anal Prev 1995; 27:651.

17.6.1.4 TREATMENT OF ALCOHOL ABUSE AND DEPENDENCE

Regarding the likelihood for remission and relapse, a good overview of the "Rates and predictors of relapse after natural and trated remission from alcohol use disorders" is given by Rudolf H. Moos and Bernice S. Moos, first published online 24 Jan 2006[173]

Among treated individuals, short-term remission rates vary between 20 and 50%, depending on the severity of the disorder and the criteria for remission[174] [175]. Initial studies suggested that between 5 and 45% of untreated individuals with alcohol use disorders may achieve some improvement or remission[176] [177]. Subsequent studies estimated untreated remission rates to range from 50 to 80% or more, depending on the severity of alcohol problems. However, these studies focused primarily on general population or media-recruited samples; that is, on individuals who had not initiated help-seeking and who may have had less severe and as yet unrecognized problems[178] [179].

In a meta-analysis of alcoholism treatment outcome studies, average short-term abstinence rates were 21% for untreated individuals in waiting-list, no-treatment or placebo conditions, compared to 43% for treated individuals [180] [181]. Similarly, Weisner, Matzger & Kaskutas[182] found that treated alcohol-dependent individuals had higher 1-year non-problem use outcomes (40% versus 23%) than did untreated individuals. Overall, these studies suggest that, especially among individuals who recognize their alcohol problems, treated individuals achieve higher remission rates than do untreated individuals.

In treated samples, estimated long-term relapse rates have varied between 20 and 80%[183] [184].

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[173] Rudolf H. Moos* andBernice S. Moos. Rates and predictors of relapse after natural and treated remission from alcohol use disorders. Addiction. Volume 101, Issue 2, pages 212–222, February 2006

[174] Miller WR, Walters ST, Bennett ME. How effective is alcoholism treatment in the United States? J Stud Alcohol 2001;62: 211–20

[175] Monahan S, Finney J. Explaining abstinence rates following treatment for alcohol abuse. A quantitative synthesis of patient, research design, and treatment effects. Addiction 1996;91: 787–805.

[176] Armor DJ, Meshkoff JE. Remission among treated and untreated alcoholics. Adv Subst Abuse 1983;3: 239–69

[177] Roizen R, Cahalan D, Shanks P. 'Spontaneous remission' among untreated problem drinkers. In: KandelDB, editor. Longitudinal research on drug use: empirical findings and methodological issues. Washington, DC: Hemisphere; 1978, pp. 197–221.

[178] Blomqvist J. Paths to recovery from substance misuse. change of lifestyle and the role of treatment. Subst Use Misuse 1996;31: 1807–52.

[179] Cunningham JA. Resolving alcohol-related problems with and without treatment: the effects of different problem criteria. J Stud Alcohol 1999;60: 463–6.

[180] Monahan S, Finney J. Explaining abstinence rates following treatment for alcohol abuse. A quantitative synthesis of patient, research design, and treatment effects. Addiction 1996;91: 787–805.

[181] Moyer A, Finney JW. Outcomes for untreated individuals involved in randomized trials of alcohol treatment. J Subst Abuse Treat 2002;23: 247–52.

[182] Weisner C, Matzger H, Kaskutas LA. How important is treatment? One-year outcomes of treated and untreated alcohol-dependent individuals. Addiction 2003;98: 901–11.

[183] Finney J, Moos R, Timko C. The course of treated and untreated substance use disorders: remission and resolution, relapse and mortality. In: McCradyB, EpsteinE, editors. Addictions: a comprehensive guidebook. New York: Oxford University Press; 1999, pp. 30–49.

[184] Jin H, Rourke SB, Patterson TL, Taylor MJ, Grant I. Predictors of relapse in long-term abstinent alcoholics. J Stud Alcohol 1998;59: 640–6.

This means that there is good evidence that even with successful treatment the risk of relapse is high, and that there is good reason to set specific conditions even on completion of treatment for those who are issued with a restricted medical certificate.

Reviewed 2014

17.6.2 DRUG DEPENDENCE/PERSISTENT SUBSTANCE ABUSE

F 11-19	Drug dependence/persistent substance abuse, includes both illicit drug use and dependence on prescribed medications Recurrence, accidents, erratic behaviour/safety performance	T – Until investigated and stabilized and criteria for fitness met. Until one year after initial diagnosis or one year after any relapse P – If persistent or there is co-morbidity likely to progress or recur while at sea	R, L – Time limited, not to work as master in charge of vessel or without close supervision and continuing medical monitoring, provided that: – treating physician reports successful participation in rehabilitation programme; and – evidence of completion of unannounced/ random programme of drug screening for at least three months with no positives and at least three negatives; and – continuing participation in drug screening programme	After three years from end of last episode without relapse and without co-morbidity

The below table[185] gives an overview of which substance concentrations of full blood that produce clinical influence corresponding to different levels of blood alcohol concentration – 0.2, 0.5 and 1.2 per mille.

[185] Fakta om rusmiddelgrenser i trafikken, http://www.fhi.no/tema/rusmidler/rusmiddelgrenser-i-trafikken - visited 16th July 2014

Substance	Corresponding to 0,2 per mille (µmol/L full blood)	Corresponding to 0,5 per mille (µmol/L full blood)	Corresponding to 1,2 per mille (µmol/L full blood)
Cannabis			
THC	0,004	0,01	0,03
GHB			
GHB	100	300	1200
Hallucinogenes			
Ketamine	0,2	0,500	1,2
LSD	0,003	*	*
Opioids			
Buprenorphine	0,002	*	*
Methadone	0,08	*	*
Morphine	0,03	0,08	0,2
* Connection between substance concentration in full blood and accident risk / driving capabilities is variable or sparsely documented. Pronounced influenced can be noted at low concentrations, especially some time after consumption of substantial amounts of amfetamine/metamfetamine			

17.6.2.1 LONG-TERM TREATMENT OF OPIOID DEPENDENCE

Opioid use disorder is a chronic, relapsing illness. Patients with opioid dependence who have gone through the acute withdrawal period from opioids have completed only the first step toward successful long-term recovery. Long-term maintenance treatment is typically needed; treatment options include non-medication, abstinence-based treatment or medication maintenance with opioid agonists (methadone or buprenorphine) and opioid antagonists (naltrexone)[186].

Long-term treatment with opioid agonists vary in different countries. Usually methadone or buprenorphine (like in Norway) is used, but some countries use heroin. In Norway this is regulated through a specific treatment programme *"Legemiddelassistert Rehabilitering"* or "LAR" (Medicine Assisted Rehabilitation).

Some individuals will be on treatment for many years/life-long. Others will reduce the doses in cooperation with the LAR-doctor over months or years.

One study carried out by Hauri-Bionda[187] examined the driving/fitness capacity of patients treated with methadone under thorough medical supervision. They found that methadone had no significant unfavourable impact on the psychophysical performances in driving ability. However, the study was carried out on 34 testpersons on a low dose methadone, and examined only indirectly factors that might influence driving, through neuropsychological tests, not on driving or in a simulator. Impotant functions as concentration, awareness, reaction time, memory, perception and sensorimotor coordination was tested.

[186] www.UpToDate.com: Treatment of opioid abuse and dependence, visited 18th July 2014
[187] Hauri-Bionda R, Bär W, Friedrich-Koch A: Driving fitness/driving capacity of patients treated with methadone, Schweiz Med Wochenschr. 1998;128(41):1538

The relapse rate is quite high. In one study of 352 patients from 16 substance addiction treatment facilities in Norway, 160 (45.4%) experienced a relapse after their prior treatment[188].

The provisions under ICD F11-19 in Appendix E is described as also covering prescribed medication, which also includes legally prescribed long-term medication with opioids. It has to our knowledge, not been demonstrated that individuals receiving LAR-treatment has so low rate of recurrence, accidents, erratic behaviour that they can be regarded as able to perform ordinary and emergency duties safely.

The likelihood of relapse on LAR-treatment is so high that it is regarded not compliant with work on board ships. Only after ended treatment with opioids, a restricted or unrestricted medical certificate may be considered.

Reviewed 2014

17.6.3 PSYCHOSIS

F 20-31	Psychosis (acute) – whether organic, schizophrenic or other category listed in the ICD. Bipolar (manic depressive disorders) Recurrence leading to changes to perception/cognition, accidents, erratic and unsafe behaviour	Following single episode with provoking factors: T – Until investigated and stabilized and conditions for fitness met. At least three months after episode	R, L – Time limited, restricted to near coastal waters and not to work as master in charge of vessel or without close supervision and continuing medical monitoring, provided that: – person has insight; – is compliant with treatment; and – has no adverse effects from medication	Case-by-case assessment at least one year after the episode, provided that provoking factors can and will always be avoided
		Following single episode without provoking factors or more than one episode with or without provoking factors: T – Until investigated and stabilized and conditions for fitness met. At least two years since last episode P – More than three episodes or continuing likelihood of recurrence. Criteria for fitness with or without restrictions are not met	R, L – Time limited, restricted to near coastal waters and not to work as master in charge of vessel or without close supervision and continuing medical monitoring providing that: – the person has insight; – is compliant with treatment; and – has no impairing adverse effects from medication	Case-by-case assessment to exclude likelihood of recurrence at least five years since end of episode if no further episodes; no residual symptoms; and no medication needed during last two years

[188] Trond Nordfjærn. Relapse patterrns among patients with substance use disorders. J Subst Use: 2011, Vol. 16;4,313-329

17.6.3.1 PARANOID PSYCHOSES

Paranoid psychoses imply a substantial safety risk to all service on board ships, even when apparently controlled with medication or after apparently complete remission.

The disease pattern of paranoid psychoses are variable, but the conditions must be regarded as chronic. A variety of physical and psychological strains as well as changes in diurnal rhythm and daily routines can precipitate to a relapse.

There are few prognostic studies of paranoid psychoses[189]. A Norwegian study demonstrated that out of 26 patients with paranoia, 13 had unchanged paranoia after 14 years, whilst eight had got schizophrenia[190]. Another study found that six patients with paranoia had an unchanged level of performance eight years later[191].

Such conditions usually will not be compliant with the regulations, and it will usually not be defensible to grant an exemption from the requirements.

It is also necessary to be aware of symptoms that can be a safety risk, even if the diagnosis paranoid psychosis is not confirmed. Symptoms which relate to a paranoid personality disorder[192], for example, abnormal suspiciousness, isolated withdrawal, tendency to interpret others' actions as hostile, persistent tendency to self-reference or a tenacious sense of personal rights, can be a safety threat, without having a diagnosis of paranoid psychosis. Self-reference sometimes develops to paranoid psychosis[193].

17.6.3.2 SCHIZOPHRENIA

Schizophrenia is a psychiatric condition which can include chronic and relapsing psychosis, often associated with distorted social and occupational function[194]. The disease is ranked by WHO as on of the "top ten" disesases of the global burden of disease[195].

Symptoms are divided into:

- Positive symptoms
  - Hallucinations
  - Delusions
  - Disordered thoughts and speech
- Negative symptoms
  - Deficits of normal emotional responses

[189] Birkeland SF. Paranoia. Ugeskr Læger 2007; 169: 3566-70
[190] Retterstol N, Opgjordsmoen S. Differences in diagnosis and long-term course and outcome between monosymptomatic and other delusional disorders. Psychopathology 1994; 27: 240-6
[191] Jørgensen P. Forløbet af vrangforestillinger [disp]. Århus: Aarhus Universitet, Det Sundhedsvidenskabelige Fakultet, 1999
[192] ICD F60.0
[193] Ulrik Malt: Selvhenføring. (2012-03-11) I Store norske leksikon. Hentet fra http://snl.no/.sml_artikkel/selvhenføring
[194] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR), American Psychiatric Association, Washington, DC 2000
[195] Murray CJL, Lopez AD. The Global Burden of Disease, Harvard University Press, Cambridge, MA 1996. p.21

  - Poverty of speech
- Distorted cognition
  - Attention
  - Memory
  - Performance ability
- Changes in affection
- Anxiety

There are many different couses of schizophrenia. One study describes eight different disease patterns[196]. Most of them start abruptly, have relapsing symptoms for a while and later on have no or only mild symptoms. Approximately 20% have a stereotypical gradual onset, continuous symptoms and a poor prognosis. Other studies have demonstrated that there are groups with quite good prognosis[197] [198].

In a 15-25 year follow-up study of 644 patients, approximately half of them had a favourable course with minimal or no symptoms at all and were working[199]. Earlier in the course functional recovery is rare. Only 14 % of 188 patients with schizophrenia or schizoaffective disorder were without symptoms 2-5 years after disease onset[200].

Most of the symptoms of the disease can be a threat to safety on board, depending on the degree. Work on board ships often follows irregular rhythms, isolation from family and environment at home, include periods of sleep deprivation and overtime work. Working and living with only a few colleagues, difficulty of follow-up from a general practitioner or a psychiatrist/psychologist is to be expected. All these factors can easily lead to a relapse in vulnerable individuals, especially people with self-reference symptoms or paranoid delusions.

The condition usually will not be compatible with work on board ships in any job position, except for very special cases with full recovery without residual symptoms, long time stability and very little need for follow-up, with or without medication.

17.6.3.3 BIPOLAR DISEASE (F31.0-F31.9)

Updated 2017-02-10

Bipolar disease usually is not compatible with service in any job position on board ship.

The reason for this is the considerable safety risk related to a person with hypomania, mania, depression or psychotic symptoms, regardless of their position on board.

[196] Blueler M. The Schizophrenic Disorders: Long-Term Patient and Family Studies, Yale University Press, London 1978
[197] Harding CM, Brooks GW, Ashikaga T, Strauss JS, Breier A: The Vermont longitudinal study of persons with severe mental illness, I: Methodology, study sample, and overall status 32 years later. Am J Psychiatry. 1987;144(6):718
[198] Hopper K, Harrison G, Janca A, Sartorius N. (Eds): Recovery from Schizophrenia: An International Perspective; A Report from the WHO Collaborative Project, The International Study of Schizophrenia, Oxford University Press, New York 2007
[199] Harrison G et al. Recovery from psychoti illness: a 15-25-year international follow-up study. Br J Psychiatry (2001) 178: 506-517
[200] Robinson DG, Woerner MG, McMeniman M, Mendelowitz A, Bilder RM: Symptomatic and functional recovery from a first episode of schizophrenia or schizoaffective disorder. Am J Psychiatry. 2004;161(3):473

Lifetime likelihood of the disease is 0,5-1,6%, equal for men and women. Onset is usually between 19 and 29 years of age. There is a considerable hereditariy trait.

Most of the literature uses the earlier classification of bipolar disease, which is the reason why we use this classification, even if newer consensus in some countries recommend another classification.

There are four types of bipolar disease:

- Bipolar I
- Bipolar II
- Cyclotymia
- Unspecified bipolar disease

Bipolar I is diagnosed in patients with one or more episodes of mania or mixed episodes (mania and depression). Hypomania is also seen frequently.

Even if the course of Bipolar I in patients nearly always includes at least one episode with major depression, this is not always the case[201] [202] [203].

In a prospective study of 163 Bipolar I patients who were followed for 15-20 years, episodes of manias without major depression (unipolar mania) were observed in 4% [204].

Bipolar II is diagnosed in patients with a medical history of at least one major depressive episode and at least one hypomanic episode, but no history of mania or mixed episodes[205].

Cyclothymic disorder is diagnosed in patients with numerous episodes of both hypomanic symptoms or episodes and depressive symptoms which do not meet the criteria for a major depressive episode. These symptoms often relapses over a period of two to several years, during which time the patient is not symptom free for more than two months at a time. This is a huge strain and usually leads to psychosocial dysfunction [206].

Unspecified bipolar disease is diagnosed in patients who have bipolar characteristics which cannot be classified as Bipolar I, II or Cyclothymia[207]

The diseases have several possible courses. “Rapid cycling” is defined as four or more episodes during a 12 month period. Seasonal variation is also seen.

Shulman KI, Tohen M: Unipolar mania reconsidered: evidence from an elderly cohort. Br J Psychiatry. 1994 Apr;164(4):547-9

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, American Psychiatric Association, Washington, DC, 2000

[206] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, American Psychiatric Association, Washington, DC, 2000

[207] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, American Psychiatric Association, Washington, DC, 2000

Episodes can easily be precipitated by stress, sleep deprivation, irregular daily rhythm, and drug and alcohol abuse.

Typical characteristics of episodes are:

- Increased activity or physical restlessness
- Loquaciousness and pressure of speech
- Flight of thought and a racing thoughts
- Reduced sleep
- Unrealistic, grandiose beliefs about one's abilities or powers
- Increased distractibility and continuously changing activities and plans
- Reduced concentration ability
- Frivolity, foolhardiness, improper behaviour, atypical for the individual

Management of the conditions consists of psychological treatment in combination with medication. One important part of the prevention of relapse is to avoid situations which earlier have led to acute episodes. Regular follow-up by GP/psychiatrist/psychologist is necessary.

The condition can change very quickly, and close follow-up is needed.

The disease is relapsing and a considerable cause of disability to work, even if the course following one single episode varies a lot. Studies have demonstrated that two years after the first episode, only 36% of the patients had regained the functional level they had before the episode and 40% had experienced relapsing episodes during that period (20% manic and 20% depressive episodes)[208].

One study of 172 patients with bipolar I disorder reported an 85 percent relapse rate over five years[209]. Another study showed that Bipolar II patients have a relapse frequency of 60% within 4 years[210]. It has also been demonstrated that up to 73% of the patients with bipolar disease who take their prescribed medicines, will have a relapse in five years[211].

Each new episode seems to reduce the threshold for precipitation of additional episodes.[212]

Bipolar II has a higher tendency for chronicity than Bipolar I [213]. For a large proportion of patients with BD, residual, sub-syndromal symptoms persist between major syndromal episodes, and studies have shown that many patients with bipolar disorder are symptomatic for approximately 50% of the time over follow-up periods of greater than 10 years[214].

[208]Tohen M, Hennen J, Zarate C, et al. Harvard first episodes project: predictors of recovery and relapse. Bipolar Disord 2002;4(suppl 1):135-136
[209] Keller MB, Lavori PW, Coryell W, et al. Bipolar I: a five-year prospective follow-up. J Nerv Ment Dis 1993; 181:238.
[210] McAllister-Williams, R. Hamish (January 1, 2006). "Relapse prevention in bipolar disorder: a critical review of current guidelines". Journal of Psychopharmacology 20 (2): 12-16
[211] Gitlin MJ, Swendsen J, Heller TL. et al. Relapse and impairment in bipolar disorder. Am J Psychiatry 1995; 152: 1635-40
[212] Post RM. Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. Am J Psychiatry 1992; 149: 999-1010
[213] Randall, Carol (2010). "1". Neuropsychological emotion processing abnormalities in bipolar disorder I and II (Ph. D thesis). University of Nevada. Retrieved 19 October 2011
[214] Fagiolini A1, Forgione R, Maccari M, Cuomo A, Morana B, Dell'Osso MC, Pellegrini F, Rossi A. Prevalence, chronicity, burden and borders of bipolar disorder. J Affect Disord. 2013 Jun;148(2-3):161-9. doi: 10.1016/j.jad.2013.02.001. Epub 2013 Mar 7.

RISK CONSIDERATIONS

Even if patients who have got the diagnosis of Bipolar disease seem to have a mild degree of symptoms, nobody can guarantee that there will not be a relapse. Relapse can occur very quickly. A stable environment, regular routines of daily living, sufficient sleep, absence of stress and the absence of use of drugs and alcohol are necessary measures to avoid relapse.

During symptom free periods, the patient will be able to function normally and many will be able to perform their regular duties, even if many patients with bipolar disorder are symptomatic for approximately 50% of the time.

Work on board ship can lead to changes in diurnal rhythm, work overload, reduced ability to contact those at home and reduced follow up from a medical doctor or psychologist.

Due to the uncertainty connected with the diagnosis of Bipolar disease it cannot be recommended that exemptions from the regulations are granted. There may be exceptions, where specific individual cases mean that the general risks of the diagnosis are significantly less than indicated in the studies.

Reviewed 2014

17.6.4 AFFECTIVE DISORDERS

F 32-38	Mood/affective disorders Severe anxiety state, depression, or any other mental disorder likely to impair performance Recurrence, reduced performance, especially in emergencies	T – While acute, under investigation or if impairing symptoms or side effects of medication present. At least three months on stable medication P – Persistent or recurrent impairing symptoms	R, L - Restrict to near-coastal waters; not to work as master in charge of ship; only when person: - has good functional recovery; - has insight; - is fully compliant with treatment and the advice given; - has no adverse effects; and - has a low[iii] likelihood of recurrence	Case-by-case assessment to exclude likelihood of recurrence after at least two years with no further episodes and with no medication or on medication with no impairing adverse effects
F32-38	Mood/affective disorders Minor or reactive symptoms of anxiety/depression Recurrence, reduced performance, especially in emergencies	T – Until symptom free. If person is on medication, the medication must be on a stable dose and free from impairing adverse effects P – Persistent or recurrent impairing symptoms	R, L – Time limited and consider geographical restriction if on stable dose of medication and free from impairing symptoms or impairing side effects from medication	Case-by-case assessment after one year from end of episode if symptom free and off medication or on medication with no impairing effects

Please note that bipolar disease (F31.0-F31.9) is not assessed according to the section 'Mood/affective disorders' F32-38, rather according to the section of 'Psychosis' (F20-31), see 17.6.3.3 Bipolar disease above.

17.6.4.1 DEPRESSION

Major depression can appear any time from childhood to old age, but mainly at the age of 30-40 years[215].

In most cases complete remission occurs within 3-4 months, either spontaneously (especially mild cases) or on treatment. One observation study of 92 patients over 23 years found an average duration of symptoms of 12 weeks[216]. Other studies have demonstrated an average of 16 weeks[217], while 20 weeks have been found in yet another study[218]. For those who had more than one episode, it was difficult to predict the length of the episodes.

Unipolar depression was the fourth leading cause of dysfunction in the world in 2002, and is calculated to be the second most important cause of dysfuntion in the coming decades[219].

Depressions can increase the risk of developing coronary disese, diabetes and stroke, and worsen the prognosis for other concomitant diseases[220].

Suicidal risk is increased to 63 % higher than the average population[221]. While comparison of suicide prevalence rates in the whole population across various countries is difficult due to differences in nature, quality, and availability of reporting, as well as collection and analysis of data related to suicide, the WHO provides some comparative international data. Male suicide rates are highest in post-communist countries such as Lithuania (68.1/100,000), Belarus (63.3/100,000), and Russia (58.1/100,000), whereas female suicide rates are highest in Asian countries such as China (14.8/100,000), Korea (14.1/100,000), and Japan (13.1/100,000)[222].

Up to 90 % of the patients with depression suffer at least one relapse. The risk of relapse is highest in the first weeks or months after an acute episode.

We talk about a major depression when five or more of the following symptoms are present most of the day, nearly daily for at least two weeks:

- Lowered mood
- Inability to experience pleasure in activities that were formerly enjoyed
- Insomnia or increased need of sleep
- Changes in appetite and body weight
- Psychomotoric retardation or agitation
- Reduced energy
- Reduced ability tdo concentrate

- Thoughts and feelings of worthlessness, inappropriate guilt or regret, helplessness, hoplessness and self-hatred.
- Recurrent thoughts about death or suicide

Even a minor or moderate depression may reduce alertness, concentration and performance to an extent that can be a safety risk in some situations. During an acute episode of a depression the person will not be fit for duty.

17.6.4.2 SEASONAL AFFECTIVE DISORDER (SAD)

The life time risk for depression and bipolar disease with seasonal variation varies between 0.4 and 2,9% in American, Canadian and British population studies[223] [224] [225] [226]. Some estimates are as high as 9.7% [227], due to different diagnostic criteria. Autumn and winter onset of major depressive episodes are more usual than other seasonal fluctuations in mood[228] [229].

The average age of onset is between 20 and 30 years, decreasing with age towards older populations[230] [231].

SAD is about 3-5 times as frequent in females than males with a higher difference between the sexes than that seen in non-seasonal depression[232]. The prevalence in children and youth is from 3.3-4.2% with the incidence increasing among girls during puberty[233] [234].

Season-related affective disorder prevalence can be higher in populations living at northern latitudes.[235]although this has been difficult to reproduce in European cohort studies.

This may be due to other influencing factors, for example genetic variation, cultural differences and climate differences[236]. About 20% of patients with SAD have a bipolar type I or II

[223] Blazer DG, Kessler RC, Swartz MS. Epidemiology of recurrent major and minor depression with a seasonal pattern: the National Comorbidity Survey. Br J Psychiatry. 1998;172:164-167

[224] Levitt AJ, Boyle MH, Joffe RT, et al. Estimated prevalence of the seasonal subtype of major depression in a Canadian community sample. Can J Psychiatry. 2000;45:650-654

[225] Levitt AJ, Boyle MH. The impact of latitude on the prevalence of seasonal depression. Can J Psychiatry. 2002;47:361-367

[226] Michalak EE, Lam RW. Seasonal affective disorder: the latitude hypothesis revisited. Can J Psychiatry. 2002;47:787-788

[227] Magnusson A, Axelsson J, Karlsson MM, et al. Lack of seasonal mood change in the Icelandic population: results of a cross-sectional study. Am J Psychiatry. 2000;157:234-238

. 1987;144:1602-1603

Treatment of seasonal affective disorder. Expert Rev Neurother. 2006;6:1039-1048

SM-IV-TR). Washington, DC: American Psychiatric Press; 2000

Magnusson A, Partonen T. The diagnosis, symptomatology, and epidemiology of seasonal affective disorder. CNS Spectr. 2005;10:625-634

clinical pattern in seasonal affective disorder (SAD) over time in a German-speaking sample. Eur Arch Psychiatry Clin Neurosci. 2002;252:54-62

Psychiatry. 1995;152:1016-1019

Carskadon MA, Acebo C. Parental reports of seasonal mood and behavior changes in children. J Am Acad Child Adolesc Psychiatry. 1993;32:264-269

[235] Mersch PP, Middendorp HM, Bouhuys AL, et al. Seasonal affective disorder and latitude: a review of the literature. J Affect Disord. 1999;53:35-48

[236] Radua J, Pertusa A, Cardoner N. Climatic relationships with specific clinical subtypes of depression. Psychiatry Res. 2010;175:217-220

disease[237] and the incidence of SAD may be higher in some populations with anxiety, ADHD and premenstrual dysphoric conditions[238] [239] [240].

Alcohol consumption can also increase as a form of self-medication of SAD-symptoms in some populations[241].

SAD tends to be a relapsing condition, with up to 70% of patients suffers from relapsing episodes of autumn and winter depression[242].

The course pattern of untreated SAD can be chronic and disabling, and can be associated with a considerable need for health services[243] [244]. Effective treatment of SAD requires early detection, information, light therapy and pharmacological therapy which must be supervised by the health service.

Complications are related to the sudden cessation of SSRI, comorbid anxiety, medicine abuse and a certain suicide risk in connection with SSRI / SNRI treatment. There is an increased risk of suicide in children, youth and young adults with major symptoms or with other psychiatric conditions, especially in the first months of treatment on antidepressants[245].

SAFETY RISK

This is related to the seriousness of the underlying condition. Several symptoms of depression are incompatible with service on board ship. Consideral improvement can, however, be expected on medication. Even if depression seems to be "neutralized" on medication, there is a certain likelihood of sequelae, side effects of medication and effects of self-cessation of therapy which can be a safety threat. The need for follow-up within the health service on shore may also restrict service at sea. Voyages with a long time at sea, a long way from home and with isolation and changes in climate could also be unfavourable.

It is not possible to assess the safety risk for the single person from general considerations. An individual risk assessment must be carried out.

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[237] White DM, Lewy AJ, Sack RL, et al. Is winter depression a bipolar disorder? Compr Psychiatry. 1990;31:196-204
[238] Levitt AJ, Joffe RT, Brecher D, et al. Anxiety disorders and anxiety symptoms in a clinic sample of seasonal and non-seasonal depressives. J Affect Disord. 1993;28:51-56
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[240] Praschak-Rieder N, Willeit M, Neumeister A, et al. Prevalence of premenstrual dysphoric disorder in female patients with seasonal affective disorder. J Affect Disord. 2001;63:239-242
[241] Sher L. Alcoholism and seasonal affective disorder. Compr Psychiatry. 2004;45:51-56
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[244] Oren DA, Rosenthal NE. Seasonal affective disorders. In: Paykel ES, ed. Handbook of affective disorders, 2nd ed. London: Churchill Livingstone; 1992
[245] FDA Proposes New Warnings About Suicidal Thinking, Behavior in Young Adults Who Take Antidepressant Medications. FDA. 2007. http://www.fda.gov (last accessed 19 October 2010

17.6.5 ANXIETY

Anxiety conditions are varied and frequent.

At any time it is estimated that approximately 10% of the population suffer from symptoms due to anxiety. The life time prevalence is around 30% in modern epidemiological studies.

The international prevalence of anxiety disorders varies greatly between published epidemiologic reports. In a review study by Somers et al[246] the pooled 1-year and lifetime prevalence rates for total anxiety disorders were 10.6% and 16.6%. Women had generally higher prevalence rates across all anxiety disorder categories compared with men, but the magnitude of this difference varied.

Generalized anxiety disorder (GAD) is one of the most common mental disorders in primary care. A European study fond a 12-month prevalence of 1.7 to 3.4% [247].

A link between major depression and other anxiety disorders has been observerd in the majority of cases with GAD[248]. In a nationally representative survey of US adults, 66% of individuals with current GAD had at least one concurrent disorder[249]. Individual disorders found to co-occur in people with GAD (rates over the previous 30 days and lifetime) included[250] [251]:

- Social phobia – 23.2 and 34.4%
- Specific phobia – 24.5 and 35.1%
- Panic disorder – 22.6 and 23.5%.

GAD may also be associated with increased rates of substance abuse, post traumatic stress disorder and obsessive-compulsive disorder.

Patients with comorbid major depression and GAD tended to have a more severe and prolonged course of illness and greater functional impairment[252] with a poorer prognosis..

Longitudinal studies in treatment-seeking patients with GAD generally provide evidence for a prolonged and fluctuating course of illness. A prospective study of 179 patients with GAD (DSM-III-R) in the US found that approximately 60% of patients revcovered over 12 years (i.e. had no more than residual symptoms for eight consecutive weeks), but around 50% of recovered patients

[246] Somers J M, Goldner E M, Waraich P, Hsu L. Prevalence and Incidence Studies of Anxiety Disorders: A systematic Review of the Literature. Can J Psychiatry 2006;51:100–113
[247] Wittchen HU, Jacobi F, Rehm J, et al. The size and burden of mental disorders and other disorders of the brain in Europe 2010. Eur Neuropsychopharmacol 2011; 21:655.
[248] Wittchen HU, Jacobi F, Rehm J, et al. The size and burden of mental disorders and other disorders of the brain in Europe 2010. Eur Neuropsychopharmacol 2011; 21:655.
[249] Wittchen HU, Zhao S, Kessler RC, Eaton WW. DSM-III-R generalized anxiety disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1994; 51:355.
[250] Wittchen HU, Zhao S, Kessler RC, Eaton WW. DSM-III-R generalized anxiety disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1994; 51:355.
[251] Brawman-Mintzer O, Lydiard RB, Emmanuel N, et al. Psychiatric comorbidity in patients with generalized anxiety disorder. Am J Psychiatry 1993; 150:1216.
[252] Tyrer P, Seivewright H, Johnson T. The Nottingham Study of Neurotic Disorder: predictors of 12-year outcome of dysthymic, panic and generalized anxiety disorder. Psychol Med 2004; 34:1385.

subsequently relapsed during the 12 year period[253]. Those with comorbid major depression and GAD or panic disorder with or without agrophobia, were half as likely to recover, compared with either disorder alone[254].

Patients with an early age of onset tend to have a more protracted course and present with comorbid depression or other disorders[255]. Late onset GAD usually starts abruptly, and is associated with clearly identifiable stressors.

The symptoms of GAD include excessive worry, autonomic hyperactivity, exaggerated startle response, muscle tension, dysphoric mood, irritability, agitation or restlessness, concentration difficulties, insomnia and fatigue. The functional impairment is similar to that which is seen with major depression[256] [257] and there may be a safety threat in many positions on board ship, depending on the degree of symptoms and the extent of symptom control.

The variation from one individual to another is considerable. It is not possible to assess the safety risk based on general knowledg about the group with the same diagnosis. A personalized risk assessment must be carried out.

17.6.5.1 OBSESSIVE COMPULSIVE DISORDER

Obsessive-CompulsiveDisorder (OCD) is a condition which can lead to a safety threat on board.

The specific content of obsessions and compulsions varies widely among individuals; however, there are certain identifiable themes, also described as “symptom dimensions”. People with OCD often have symptoms in multiple dimensions, which include:

- Cleaning – fears of contamination and cleaning rituals
- Symmetry – symmetry obsessions and repeating ordering and counting compulsions
- Forbidden or taboo thoughts – ecamples include aggressive, sexual and religious obsessions and related compulsions
- Harm (eg thought or images about harm befalling oneself or others and checking compulsions)
- Hoarding (hoarding obsessions and compulsions)

The 12-month prevalence in the US is 1.2 % and estimated lifetime prevalence is 2-3 %[258] [259].

[253] Bruce SE, Yonkers KA, Otto MW, et al. Influence of psychiatric comorbidity on recovery and recurrence in generalized anxiety disorder, social phobia, and panic disorder: a 12-year prospective study. Am J Psychiatry 2005; 162:1179.
Am J Psychiatry 2005; 162:1179.
[255] Shores MM, Glubin T, Cowley DS, et al. The relationship between anxiety and depression: a clinical comparison of generalized anxiety disorder, dysthymic disorder, panic disorder, and major depressive disorder. Compr Psychiatry 1992; 33:237.
U. Impairment in pure and comorbid generalized anxiety disorder and major depression at 12 months in two national surveys. Am J Psychiatry 1999; 156:1915.
eralized anxiety disorder and major depression in a national survey. Int Clin Psychopharmacol 2000; 15:319.
[258] Kessler RC, Chiu WT, Demler O, et al. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005; 62:617.
[259] Ruscio AM, Stein DJ, Chiu WT, Kessler RC. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. Mol Psychiatry 2010; 15:53.

Comorbidity with other psychiatric conditions is common. 76% of patients with OCD have a lifetime history of another anxiety disorder (eg panic disorder, social anxiety disorder, generalized anxiety disorder or specific phobia), 63% have a lifetime history of a mood disorder, most commonly major depressive disorder (21%)[260] and 23-32% have comorbid obsessive-compulsive personality disorder[261]

Suicidal thoughts occur at some point in as many as 50% of individuals with OCD[262] and suicidal attempts are reported in up to 25% of individuals with OCD. The presence of comorbid depressive disorder increases the risk of suicide attempts. Some patients with OCD experience intrusive fears that they will harm others, but there is no data suggesting that they are more likely to do so at a rate higher than the general population.

Avoidance behaviour is common in OCD, and can be pervasive and severely restrict functioning. Once obsessions or compulsions are triggered, people with OCD may experience a range of affective responses, for example marked anxiety which can include recurrent panic attacks. Others report strong feeilings of disgust. Many individuals with OCD experience dysfunctional beliefs[263] including:

- Inflated responsibility and the tendency to overestimate threat
- Perfectionism and dthe intolerance of uncertainty
- Overvaluing the importance of thoughts (eg believing that having a forbidden thought is as bad as acting on it) and the need to control thoughts.

The mean age of onset of OCD is 19.5 years in the US, with 25% percent of cases beginning by the age of 14 years[264] [265]. If untreated the course of OCD is usually chronic, with fluctuating symptoms[266] [267]. Some have an episodic course and a minority has a deteriorating course.

Without treatment rates of remission of OCD in adults are low, eg. 20% in a 40 year follow-up study of 144 patients[268]. Even with treatment, only some adults will recover over time[269].

The condition is treated with SSRI preparations. Among those who do not get a satisfactory effect, second generation antipsychotic medication is often added, and exposure therapy with

[260] Ruscio AM, Stein DJ, Chiu WT, Kessler RC. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. Mol Psychiatry 2010; 15:53.
[261] Pinto A, Eisen JL. Personality features of OCD and spectrum conditions. In: The Oxford Handbook of Obsessive Compulsive and Spectrum Disorders, Steketee G (Ed), Oxford University Press, New York 2012.
[262] Torres AR, Ramos-Cerqueira AT, Ferrão YA, et al. Suicidality in obsessive-compulsive disorder: prevalence and relation to symptom dimensions and comorbid conditions. J Clin Psychiatry 2011; 72:17.
[263] Obsessive Compulsive Cognitions Working Group. Psychometric validation of the obsessive belief questionnaire and interpretation of intrusions inventory--Part 2: Factor analyses and testing of a brief version. Behav Res Ther 2005; 43:1527.
[264] Kessler RC, Chiu WT, Demler O, et al. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005; 62:617.
[265] Ruscio AM, Stein DJ, Chiu WT, Kessler RC. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. Mol Psychiatry 2010; 15:53.
[266] Skoog G, Skoog I. A 40-year follow-up of patients with obsessive-compulsive disorder [see commetns]. Arch Gen Psychiatry 1999; 56:121.
[267] Ravizza L, Maina G, Bogetto F. Episodic and chronic obsessive-compulsive disorder. Depress Anxiety 1997; 6:154.
[268] Skoog G, Skoog I. A 40-year follow-up of patients with obsessive-compulsive disorder [see commetns]. Arch Gen Psychiatry 1999; 56:121.
[269] Eisen JL, Pinto A, Mancebo MC, et al. A 2-year prospective follow-up study of the course of obsessive-compulsive disorder. J Clin Psychiatry 2010; 71:1033.

response prevention (ERP) in combination with cognitive therapy (CBT) is usual. ERP usually is more effective in patients with compulsions than obsessions.

A meta-analysis of eight randomized trials with 241 patients with OCD found that cognitive, behavioural and cognitive-behavioural psychotherapies led to greater reduction in symptoms than treatment as usual[270]. The therapies lead to average symptom reduction between 50 and 70%[271]

OCD is associated with a reduced quality of life as well as high levels of social and occupational impairment, due to time spent obsessing and acting on compulsions, avoidance of situations that can trigger obsessions and compulsions and specific symptoms that can create specific obstacles. There is a potential safety risk arising from the reduction of awareness and concentration when obsessions or compulsions occur and this is often worsened by exhaustion and fatigue. Therefore OCD is usually not compatible with work on board ships but a personalised risk assessment should be performed.

Reviewed 2015

17.6.6 PERSONALITY AND DEVELOPMENT DISORDERS

F 00-99	Other disorders, e.g. disorders of personality, attention (e.g. ADHD), development (e.g. autism) Impairment of performance and reliability and impact on relationships	P – If considered to have safety-critical consequences	R – As appropriate if capable of only limited duties	No anticipated adverse effects while at sea. No incidents during previous periods of sea service.

17.6.6.1 ADHD/ADD

ADHD (Attention Deficit Hyperactivity Disorder) or DAMP (Deficit in Attention Motor Control and Perception) can be a safety threat on board ship. ADD (Attention Deficit Disorder) is a variant of the disorder without dominating hyperactivity, although otherwise with the same symptoms. Symptoms which can be safety critical are a lack of :

- Working memory
- Task-shifting
- Self-monitoring
- Initiation
- Self-inhibiton

These symptoms may lead to the poor attention problems characteristic of adult ADHD eg.

[270] Gava I, Barbui C, Aguglia E, et al. Psychological treatments versus treatment as usual for obsessive compulsive disorder (OCD). Cochrane Database Syst Rev 2007; :CD005333.
[271] Abramowitz, JS. Understanding and treating obsessive-compulsive disorder: A cognitive-behavioral approach, Erlbaum, Mahwah NJ 2006.

- Difficulties in remaining focused in a task, especially for long periods
- Difficulties in organizing activities
- Difficulties in prioritizing tasks
- Difficulties in following through and completing tasks
- Forgetfulness
- Time managment difficulties (missing appointments or deadlines)

The condition is usually diagnosed in childhood and longitudinal studies of children with ADHD have documented the persistence of the disorder into adulthood in the majority of cases. Most studies report that 40-60% of patients go on to have significant ADHD-related problems in adulthood[272] [273] [274] [275]. Some adults present with impairment in a clinical setting only later in life when they confront new and increasingly complex tasks that characterize adulthood and that cannot be managed with their existing neuropsychological repertoire.Prevalence in adulthood is 1-2% of the population [276].

A systemic review of adverse occupational effects of ADHD found that adults with ADHD had higher levels of unemployment compared to control groups[277]. Adults with ADHD who are employed experience workplace impairment and reduced productivity; they are also at increased risk of accidents, trauma, and workplace injuries, particularly traffic accidents. Other problems associated with adult ADHD include reduced educational achievement and increased rates of substance abuse and criminality[278] [279] [280] [281].

The domains of emotional, educational, and social adjustment follow variable courses in individuals with ADHD, ranging from poor to good. The persistence of ADHD is not associated with a uniform functional outcome but leads instead to a wide range of emotional, educational, and social adjustment outcomes[282] [283]

There are many rating scales for screening and diagnosing ADHD in accordance with DSM-IV, but they need to be modified for DSM-V. 14 adult scales have been validated in 35 studies

[272] Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. J Abnorm Psychol 2002; 111:279.
[273] Mannuzza S, Klein RG, Bessler A, et al. Adult psychiatric status of hyperactive boys grown up. Am J Psychiatry 1998; 155:493.
[274] Weiss G, Hechtman L, Milroy T, Perlman T. Psychiatric status of hyperactives as adults: a controlled prospective 15-year follow-up of 63 hyperactive children. J Am Acad Child Psychiatry 1985; 24:211.
[275] Küpper T, Haavik J, Drexler H, et al. The negative impact of attention-deficit/hyperactivity disorder on occupational health in adults and adolescents. Int Arch Occup Environ Health 2012; 85:837.
[276] Thomsen PH, Damm D. ADHD hos voksne. Ugeskr Læger 2008; 170: 3395
[277] Biederman J, Mick E, Faraone SV. Normalized functioning in youths with persistent attention-deficit/hyperactivity disorder. J Pediatr 1998; 133:544.
[278] Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. J Abnorm Psychol 2002; 111:279.
[279] Mannuzza S, Klein RG, Bessler A, et al. Adult psychiatric status of hyperactive boys grown up. Am J Psychiatry 1998; 155:493.
[280] Weiss G, Hechtman L, Milroy T, Perlman T. Psychiatric status of hyperactives as adults: a controlled prospective 15-year follow-up of 63 hyperactive children. J Am Acad Child Psychiatry 1985; 24:211.
[281] Sobanski E, Brüggemann D, Alm B, et al. Psychiatric comorbidity and functional impairment in a clinically referred sample of adults with attention-deficit/hyperactivity disorder (ADHD). Eur Arch Psychiatry Clin Neurosci 2007; 257:371.
[282] Biederman J, Mick E, Faraone SV. Normalized functioning in youths with persistent attention-deficit/hyperactivity disorder. J Pediatr 1998; 133:544.
[283] Biederman J, Mick E, Faraone SV. Age-dependent decline of symptoms of attention deficit hyperactivity disorder: impact of remission definition and symptom type. Am J Psychiatry 2000; 157:816.

according to UpToDate[284], and Conners' Adult Rating Scale (CAARS) and the Wender Utah Rating Scale (short version) have more robust psychometric statistics and content validity[285].

The rate of comorbid psychiatric disorders in adults with ADHD tends to increase with age[286] [287] [288]. This includes anxiety, depression, substance-use disorder,and antisocial personality disorder.. The following co-occurrences have been demonstrated in US samples adults with ADHD compared with the general US population[289]:

- Mood disorders, OR = 2.7 to 7.5 (95% CI 3.0–8.2)
- Anxiety disorders, OR = 1.5 to 5.5 (95% CI 2.4–5.5)
- Substance use disorders (SUD), OR = 1.5 to 7.9 (95% CI 1.4–6.5)
- Intermittent explosive disorder, OR=3.7 (95% CI 2.2-6.2)

SIMILAR RESULTS HAVE BEEN REPORTED INTERNATIONALLY[290]

- Mood disorders, OR = 3.9 (95% CI 3.0–5.1)
- Anxiety disorders, OR = 4.0 (95% CI 3.0–5.2)
- SUD, OR = 4 (95% CI 2.8–5.8)

On asking for specialist advice it is important that specific questions are asked regarding the symptoms of ADHD as well as symptoms of comorbidity which could compromise safety.

As there are so many degrees of residual symptoms and dysfunction in adulthood and the effect of treatment is variable, it is not possible on a general basis to tell whether a specific individual will be a safety risk for ship or crew. An individual risk assessment must be carried out.

17.6.7 ASPERGER SYNDROME

Asperger syndrome is an autism spectrum disorder with the following DSM-IV criteria[291] [292]:

- Qualitative impairment in social interaction, as manifested by at least two of the following:
  - Marked impairments in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
  - Failure to develop peer relationships appropriate to developmental level
  - A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (eg, by a lack of showing, bringing, or pointing out objects of interest to other people)
  - Lack of social or emotional reciprocity

[284] Accessed 8 OCtober 2014
[285] Taylor A, Deb S, Unwin G. Scales for the identification of adults with attention deficit hyperactivity disorder (ADHD): a systematic review. Res Dev Disabil 2011; 32:924.
[286] Kessler RC, Adler L, Barkley R, et al. The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. Am J Psychiatry 2006; 163:716.
[287] Cumyn L, French L, Hechtman L. Comorbidity in adults with attention-deficit hyperactivity disorder. Can J Psychiatry 2009; 54:673.
[288] Biederman J. Impact of comorbidity in adults with attention-deficit/hyperactivity disorder. J Clin Psychiatry 2004; 65 Suppl 3:3.
[289] Kessler RC, Adler L, Barkley R, et al. The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. Am J Psychiatry 2006; 163:716.
[290] Fayyad J, De Graaf R, Kessler R, et al. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. Br J Psychiatry 2007; 190:402.
[291] UpToDate accessed 8 October 2014.
[292] Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (Copyright 2000). American Psychiatric Association.

- Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
    - Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal either in intensity or focus
    - Apparently inflexible adherence to specific, nonfunctional routines or rituals
    - Stereotyped and repetitive motor mannerisms (eg, hand or finger flapping or twisting, or complex whole-body movements)
    - Persistent preoccupation with parts of objects
- The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning
- There is no clinically significant general delay in language (eg, single words used by age two years, communicative phrases used by age three years)
- There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than social interaction), and curiosity about the environment in childhood
- Criteria are not met for another specific pervasive developmental disorder or schizophrenia

People with Asperger's can have difficulties in finding their place in and interacting with members of a group..

Individuals with Asperger's syndrome often behave as if they are alone even in a group of people, for example regarding their own interests as superior to the interst of other group members. They can appear as absent with improper eye contact.

Linguistic skills are usually normal, although sufferers may speak in a didactic voice without normal variation, in a theatrical way or with exaggerations which are out of context. They understand language literally, and may have difficulties in understanding how the meaning can change with the context, resulting in failure to understand metaphors, humor, sarcasms, teasing or cunning.

Disturbances of behaviour is often the first sign of Asperger's and may persist throughout life. Perseveration (continuously, involuntary repetitions of the same action) is conspicuous and more expressed and specific than is seen in other people of the same age. There may be a very narrow spectrum of interest, often for scientific or technical matters (eg ceiling fans or vacuum cleaners), and sufferers may have difficulties in changing their attention from their preferred objects, even after several reminders.

Cognitive rigidity is another symptom which can result in intolerance to changes in daily routines.

Individuals with Asperger's syndrome can be disorganised and have difficulties in carrying out daily duties, as overfocusing on one specific field of interest can take attention away from routine tasks. Problems can also be caused by lapse of memory and problems in planning.

Asperger's is associated with anxiety, ADHD, depression and other affective disorders, learning difficulties and tics. The frequency of comorbidity is uncertain, but several studies estimate that

most of the patients have at least one additional diagnosis in as many as 74% of cases[293], ADHD being most frequent in the young, depression in adults.

17.6.7.1 SAFETY CONSIDERATIONS

Several of the symptoms related to Asperger syndrome can be a safety threat. Communication can be misunderstood, the ability to plan and organise work, as well as prioritizing tasks can be endangered, and they may not fit well into the crew. They are sensitive to being critizised, but may critizise other brutally. Their perfectionism can be irritating to others, which can be dangerous in the 24 hour society of a crew, where people live and work together for prolonged time.

There are many different degrees of incapacity , from the mildest cases to the worst who are unable to function in any job. An individual risk assessment therefore must be carried out, looking specifically for the above mentioned symptoms which can threaten safety on board ship.

Reviewed 2014

[293] Mattila ML, Hurtig T, Haapsamo H, Jussila K, Kuusikko-Gauffin S, Kielinen M, Linna SL, Ebeling H, Bloigu R, Joskitt L, Pauls DL, Moilanen I: Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: a community- and clinic-based study. J Autism Dev Disord. 2010;40(9):1080

17.7 G 00-99 DISEASES OF THE NERVOUS SYSTEM

17.7.1 EPILEPSY

G 40-41	Single seizure Harm to ship, others and self from seizures	Single seizure T – While under investigation and for one year after seizure	R – One year after seizure and on stable medication. Non-watchkeeping duties; in nearcoastal waters.	One year after seizure and one year after end of treatment. If provoked, there should be no continuing exposure to the provoking agent.
	Epilepsy – No provoking factors (multiple seizures) Harm to ship, others and self from seizures	T – While under investigation and for two years after last seizure P – Recurrent seizures, not controlled by medication	R – Off medication or on stable medication with good compliance: case-by-case assessment of fitness, restricted to non-watchkeeping duties in near-coastal waters	Seizure-free for at least the last ten years, has not taken anti-epilepsy drugs during that ten-year period and does not have a continuing likelihood of seizures
	Epilepsy provoked by alcohol, medication, head injury (multiple seizures) Harm to ship, others and self from seizures	T – While under investigation and for two years after last seizure P – Recurrent seizures, not controlled by medication	R – Case-by-case assessment after two years' abstention from any known provoking factors, seizure-free and either off medication or on stable medication with good compliance; restricted to non-watchkeeping duties in nearcoastal waters	Seizure-free for at least the last five years, has not taken anti-epilepsy drugs during that five-year period, provided there is not continuing exposure to the provoking agent

17.7.1.1 EPILEPSY - INTRODUCTION

Epilepsy is a collective term for a wide spectrum of fits due to disturbances in cerebral function. They are divided into generalized seizures (approximately 40%) and partial (focal) (approximately 60%), which again can be divided in numerous subtypes. The prevalence in Norway is around 0,7%, whilst life time prevalence is around 3%, of which 70% go into remission. Each year we estimate 40-50 new cases per 100,000 inhabitants, i.e. around 2,300 new cases.

In 25-35% of the cases a specific cause is found. Of detectable causes brain abnormalities or metabolic disturbances are usual. In adults brain damages and brain tumors are frequent causes, although causes often are multifactorial.

17.7.1.2 EEG AND THE DIAGNOSIS OF EPILEPSY

One should not put too much emphasis on EEG in the diagnostics of epilepsy. EEG alone can neither be used alone to establish the diagnosis of epilepsy, nor to exclude such a diagnosis. The reason for this is the following:

- Most EEG changes can be causes by numerous neurological diseases
- Many diseases can cause different types of EEG changes
- Intermittent EEG changes (including intermittent epileptic discharges (IED) during seizure) can be infrequent, and may not be present during the registration of an EEG
- Eeg can be abnormal in individual without any sign of illness,
- Not all brain diseases are accompanied by EEG changes, especially if the pathological changes are smallm chronic or located deep in the brain tissue.

This means that when clinical observation indicates epilepsy and EEG is not pathological, clinical observation is regarded mor important than the laboratory findings (EEG).

EPILEPTIFORM ACTIVITY WITHOUT SEIZURE (INTERICTAL EPILEPTIFORM ACTIVITY = IED) ON EEG

The diagnostic criteria for IED are:

- Paroxystic findings on EEG, clearly different from the background activity of the individual
- They must include an abrupt change in polarity and last for several milliseconds
- Duration of each paroxysm can be up to 200 ms
- Spikes usually have a duration of less than 70 ms
- Sharp waves usually lasts for between 70 and 200 ms
- The discharge must represent a physiological area
- The paroxysms must not be one of the benign variants like «wicket spikes», «small sharp spikes» or «vertex waves».

SENSITIVITY

IED is found in 20-55% of individuals with epilepsy on first routine EEG[294] [295] [296] [297]. The number with IED increases to 80-90% if four or more consecutive EEGs are registered.[298] [299] [300] [301] [302].

In one study in an epilepsy monitoring unit, 43 percent of patients with epilepsy had an IED in the first hour of recording, and the number increased to 89 percent after 24 hours [303].

In another study of 100 adult patients with confirmed epileptic seizures, EEG monitoring for seven days revealed IEDs in 81 percent[304].

In another study in an epilepsy monitoring unit, 86 percent of 119 patients with definite epilepsy had IEDs in the first two days, 3 percent of patients developed IEDs after two days; only 12 percent never had IEDs [305].

SPECIFICITY

IEDs are rare in patients without a history of seizures. Studies in healthy flight personnel reveal IEDs in 0.5%[306] [307]. The prevalence of IEDs in hospitalized adults with neurologic or psychiatric illness is found to be 2.0-2.6%[308].

Some conditions are associated with the presence of IEDs on EEG, but do not imply epilepsy. These include occipital spikes seen in blind people (especially those who are congenitally blind)[309].

Withdrawal from short-acting barbiturates and benzodiazepines, certain metabolic derangements (eg, hypocalcemia, uremia, dialysis disequilibrium), as well as high drug levels of

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294 Glick TH. The sleep-deprived electroencephalogram: evidence and practice. Arch Neurol 2002; 59:1235.
295 King MA, Newton MR, Jackson GD, et al. Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet 1998; 352:1007.
296 Marsan CA, Zivin LS. Factors related to the occurrence of typical paroxysmal abnormalities in the EEG records of epileptic patients. Epilepsia 1970; 11:361.
297 van Donselaar CA, Schimsheimer RJ, Geerts AT, Declerck AC. Value of the electroencephalogram in adult patients with untreated idiopathic first seizures. Arch Neurol 1992; 49:231.
298 King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA, Silvapulle MJ, Berkovic SF: Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet. 1998;352(9133):1007
299 Marsan CA, Zivin LS. Factors related to the occurrence of typical paroxysmal abnormalities in the EEG records of epileptic patients. Epilepsia 1970; 11:361.
300 Doppelbauer A, Zeitlhofer J, Zifko U, et al. Occurrence of epileptiform activity in the routine EEG of epileptic patients. Acta Neurol Scand 1993; 87:345.
301 Goodin DS, Aminoff MJ. Does the interictal EEG have a role in the diagnosis of epilepsy? Lancet 1984; 1:837.
302 Salinsky M, Kanter R, Dasheiff RM. Effectiveness of multiple EEGs in supporting the diagnosis of epilepsy: an operational curve. Epilepsia 1987; 28:331.
303 Narayanan JT, Labar DR, Schaul N: Latency to first spike in the EEG of epilepsy patients. Seizure. 2008;17(1):34.
304 Walczak, T, Scheuer, M, Resor, S, Pedley, T: Prevalence and features of epilepsy without interictal epileptiform discharges, Neurology. 1993; 43:287.
305 Friedman DE, Hirsch LJ: How long does it take to make an accurate diagnosis in an epilepsy monitoring unit? J Clin Neurophysiol. 2009;26(4):213.
306 Bennett DR. Spike-wave complexes in "normal" flying personnel. Aerosp Med 1967; 38:1276.
307 Gregory RP, Oates T, Merry RT. Electroencephalogram epileptiform abnormalities in candidates for aircrew training. Electroencephalogr Clin Neurophysiol 1993; 86:75.
308 Zivin L et al, Brain 1968 / Cavazzuti GB et al, Epilepsia 1980 / Bricgers SL, Arch Neurol 1987.
309 Wong VC. Cortical blindness in children: a study of etiology and prognosis. Pediatr Neurol 1991; 7:178.

lithium, neuroleptics (especially clozapine), bupropion, and tricyclic antidepressants have been associated with IEDs even in the absence of accompanying seizures[310] [311] [312]

CONCLUSION

This means that EEG cannot exclude the diagnosis of epilepsy if the clinical suspicion is strong, and EEG cannot confirm the diagnosis of epilepsy if there are no seizures clinically suspect of epilepsy.

17.7.1.3 POST-TRAUMATIC EPILEPSY

All variants of secondary brain damage can lead to epilepsy, although it sometimes can take many years until epilepsy develops. While only 4% of all epilepsy cases are attributed to trauma, 13% of those cases that are of known cause are post-traumatic[313].

A distinct category of immediate seizures, those occurring upon or within seconds of impact, is controversial. Some feel that these are “convulsive concussions” and not epileptic events[314]; others include them in the category of early seizures because of their similar associated risk for post-traumatic epilepsy [315] [316].

Between 17 and 33% of patients with early seizures will develop epilepsy compared with a 2 percent overall incidence[317]. The 10-year incidence of epilepsy after traumatic brain injury (TBI) is estimated at about 2% [318]. The figures are disputed, and the results are different in different studies. In a context of risk assessment, one cannot, however, disregard these figures. There is a strong correlation between the egree of brain damage and the likelihood to develop posttraumatic epilepsy.

After commotion the RR (relative risk) was 2.2, after more serious damage RR was 7.4 for the development of epilepsy. After 10 years the likelihood is still increased: RR 1.51 and 4.29. The

310 Van Cott, AC, Brenner, RP. Drug Effects and Toxic Encephalopathy. In: Current practice of clinical electroencephalograhy, Ebersole, JS, Pedley, TA (Eds), Lippincott Williams and Wilkins, Philadephia 2003 p.463.
311 Malow BA, Reese KB, Sato S, et al. Spectrum of EEG abnormalities during clozapine treatment. Electroencephalogr Clin Neurophysiol 1994; 91:205.
312 Hughes JR, Schreeder MT. EEG in dialysis encephalopathy. Neurology 1980; 30:1148.
313 Annegers, JF. The epidemiology of epilepsy, In: The treatment of epilepsy: Principles and practice, 3rd ed, Wyllie, E (Ed), Lippincott Williams, Philadelphia 2001. p.135.
314 McCrory PR, Bladin PF, Berkovic SF: Retrospective study of concussive convulsions in elite Australian rules and rugby league footballers: phenomenology, aetiology, and outcome. BMJ. 1997;314(7075):171.
315 Barry E. Posttraumatic epilepsy, In: The treatment of epilepsy: Principles and practice, 3rd ed, Wyllie E (Ed), Lippincott Williams, Philadelphia 2001. p.609
316 Emanuelson I, Uvebrant P: Occurrence of epilepsy during the first 10 years after traumatic brain injury acquired in childhood up to the age of 18 years in the south western Swedish population-based series. Brain Inj. 2009;23(7):612.
317 Pagni CA, Zenga F: Posttraumatic epilepsy with special emphasis on prophylaxis and prevention. Acta Neurochir Suppl. 2005;93:27
318 Annegers JF, Grabow JD, Groover RV, Laws ER Jr, Elveback LR, Kurland LT: Seizures after head trauma: a population study. Neurology. 1980;30(7 Pt 1):683

incidence of post-traumatic seizures (early) can be as high as from 6-10% in some studies and 30% in others [319] [320].

In one population-based cohort, the cumulative five-year probability of seizures was 0.5% in patients with mild injury (those with loss of consciousness or amnesia <30 minutes); 1.2 percent for those with moderate injuries (loss of consciousness for 30 minutes to 24 hours or skull fracture); and 10.0% in those with severe injuries (loss of consciousness or amnesia for more than 24 hours or subdural hematoma or cerebral contusion[321] . Another study of 647 hospitalized patients categorized TBI severity more traditionally with the Glasgow Coma Scale (GCS). The two-year incidence of epilepsy was 8.0% for GCS 13 to 15 and 16.8 percent for GCS 3 to 8 [322].

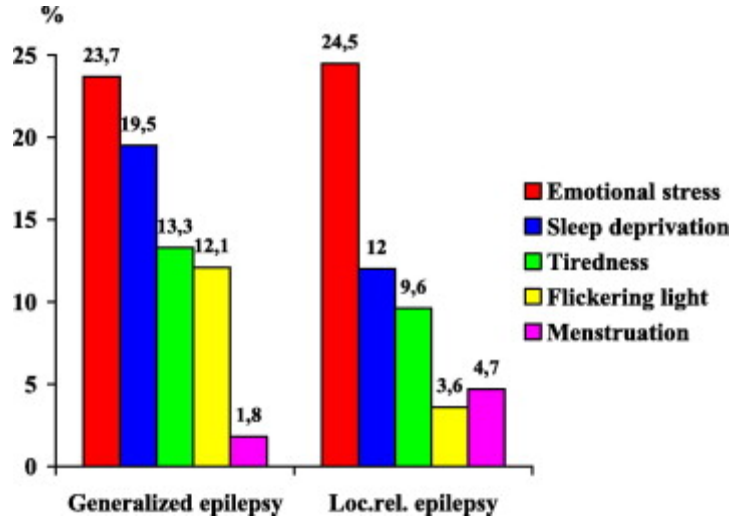
Other subsets of patients at much higher risk have been identified and include those with early seizures, intracranial hemorrhage or cerebral contusion, depressed skull fracture, and penetrating head injury[323] [324] [325] [326] [327]. Traumatic brain injury (TBI) associated with intracranial lesions on CT was associated with an 18% risk of late seizures in one series [328]. In penetrating missile combat injuries, the incidence is more than 50% [329] [330]. The requirement for neurosurgical procedure (hemorrhage evacuation, ventriculostomy) increased the risk, and multiple surgeries increased the risk over single surgeries [331].

17.7.1.4 EPILEPSY – SEIZURE PRECIPITATING FACTORS

Different factors can precipitate seizures in individuals who are predisposed. These include emotional stress, sleep deprivation, tiredness, flickering light and menstruation. A study of 1677 patients with epilepsy was carried out by Nakken et al [332] of twins and their family members ascertained from the Norwegian Twin Panel (NTP), the Danish Twin Registry (D>TR) and the Mid-Atlantic Twin Registry (MATR). Participants were asked about seizure precipitants using a closed-ended questionnaire. 53% reported at least one seizure-precipitating factor, while 30% claimed to have experienced two or more such factors. Emotional stress, sleep deprivation, and tiredness

319 Temkin NR: Risk factors for posttraumatic seizures in adults. Epilepsia. 2003;44 Suppl 10:18.
320 Frey LC: Epidemiology of posttraumatic epilepsy: a critical review. Epilepsia. 2003;44 Suppl 10:11
321 Annegers JF, Hauser WA, Coan SP, Rocca WA: A population-based study of seizures after traumatic brain injuries. N Engl J Med. 1998;338(1):20.
322 Analyzing risk factors for late posttraumatic seizures: a prospective, multicenter investigation. Englander J, Bushnik T, Duong TT, Cifu DX, Zafonte R, Wright J, Hughes R, Bergman W Arch Phys Med Rehabil. 2003;84(3):365.
323 Temkin NR. Risk factors for posttraumatic seizures in adults. Epilepsia 2003; 44 Suppl 10:18.
324 Frey LC. Epidemiology of posttraumatic epilepsy: a critical review. Epilepsia 2003; 44 Suppl 10:11.
325 Asikainen I, Kaste M, Sarna S. Early and late posttraumatic seizures in traumatic brain injury rehabilitation patients: brain injury factors causing late seizures and influence of seizures on long-term outcome. Epilepsia 1999; 40:584.
326 Raymont V, Salazar AM, Lipsky R, et al. Correlates of posttraumatic epilepsy 35 years following combat brain injury. Neurology 2010; 75:224.
327 Yeh CC, Chen TL, Hu CJ, et al. Risk of epilepsy after traumatic brain injury: a retrospective population-based cohort study. J Neurol Neurosurg Psychiatry 2013; 84:441.
328 Pagni CA, Zenga F: Posttraumatic epilepsy with special emphasis on prophylaxis and prevention. Acta Neurochir Suppl. 2005;93:27.
329 Pagni CA, Zenga F. Posttraumatic epilepsy with special emphasis on prophylaxis and prevention. Acta Neurochir Suppl 2005; 93:27.
330 Salazar AM, Jabbari B, Vance SC, Grafman J, Amin D, Dillon JD: Epilepsy after penetrating head injury. I. Clinical correlates: a report of the Vietnam Head Injury Study. Neurology. 1985;35(10):1406.
331 23.Englander J, Bushnik T, Duong TT, et al. Analyzing risk factors for late posttraumatic seizures: a prospective, multicenter investigation. Arch Phys Med Rehabil 2003; 84:365.
332 Nakken K O, Solaas M H, Kjeldsen M J, Friis M L, Pellock J M, Corey A L. Which seizure-precipitating factors do patients with epilepsy most frequently report? Epilepsy & Behaviour. 2005;6(1):85-89.

were the three most frequently reported precipitants. Patients with generalized seizures seemed to be more sensitive to sleep deprivation and flickering light than those with partial seizures, while women with partial seizures appeared to be more prone to seizures during menstruation than women with generalized seizures.



Distribution of five seizure-precipitating factors in those with generalized and localization-related epilepsy combined over populations, respectively. (Karl O. Nakken, Marit H. Solaas, Marianne J. Kjeldsen, Mogens L. Friis, John M. Pellock, Linda A. Corey)

Irregular diurnal rhythm and irregular meals can be a result of different shift regimes and overtime work in connection with port calls, loading and unloading. This can result in sleep deprivation and hypoglycaemia. Travel by helicopter can result in flickering light stimulation.

17.7.1.5 EPILEPSY – EFFECT OF TREATMENT AND PROGNOSIS

Approximately half of the patients who have recently been diagnosed with epilepsy are effectively treated by the first antiepileptic drug (AED) prescribed [333] [334]. If the first chosen medicine lacks efficacy, about 10-20% will have a successful second drug trial[335]. Around 2/3 of the patients will get seizure control on monotherapy. Up to 80% can be seizure free on treatment with antiepileptics [336] [337] [338].

Risk of seizure recurrence after a first seizure — the risk of recurrence after a single, unprovoked seizure was 14 percent at one year, 29 percent at three years, and 34 percent at five

[333] Kwan P, Brodie MJ: Effectiveness of first antiepileptic drug. Epilepsia. 2001;42(10):1255.
[334] Brodie MJ, Perucca E, Ryvlin P, et al. Comparison of levetiracetam and controlled-release carbamazepine in newly diagnosed epilepsy. Neurology 2007; 68:402.
[335] Bonnett LJ, Tudur Smith C, Donegan S, Marson AG. Treatment outcome after failure of a first antiepileptic drug. Neurology 2014; 83:552.
[336] Luciano AL, Shorvon SD Ann: Results of treatment changes in patients with apparently drug-resistant chronic epilepsy. Neurol. 2007;62(4):375.
[337] Callaghan BC, Anand K, Hesdorffer D, Hauser WA, French JA: Likelihood of seizure remission in an adult population with refractory epilepsy. Ann Neurol. 2007;62(4):382.
[338] Schiller Y, Najjar Y: Quantifying the response to antiepileptic drugs: effect of past treatment history. Neurology. 2008;70(1):54.

years in one prospective hospital-based study[339]. However, most individuals in this study were treated with AEDs. Prospective, randomized trials of individuals with a first unprovoked seizure estimate the two-year recurrence risk in untreated patients to be 40 to 50 percent[340] [341] [342]. The risk of recurrence is highest immediately after the first seizure and diminishes with time; 80 to 90 percent of patients who have recurrent seizures do so within two years[343] [344].

Most individuals with epilepsy will have good or complete seizure control on medicines, but some of them will never get seizure control. 20-40% of patients are likely to have refractory epilepsy (defined as therapeutic failure of three antiepileptic drugs)[345].

17.7.1.6 EPILEPSY – ADVERSE EFFECTS OF ANTIEPILEPTIC DRUG (AED) TREATMENT

Drowsiness, dizziness, visual disturbances, tiredness, headache, sleep disturbances and sometimes hyperactivity, ataxia, depression, sedation, irritability, agression, changes in mood and confusion can all be adverse effects of most AEDs. These adverse effects can represent a safety risk on board, dependent on the position and the job tasks the individual has. The seafarers' doctor must therefore assess the risk connected to the use of such medicines in each individual case.

EPILEPSY – RISK ASSESSMENT

There is a considerable safety risk associated with epilepsy in persons. The risk is greatest for individuals on bridge watch, or among those who have a safety-critical function. The risk can never be completely ignored, with or without treatment. The medical condition itself as well as the treatment may imply a safety risk which in many cases is unacceptable on board ship.

17.7.1.7 EPILEPSY – SERUM CONCENTRATION MEASUREMENT, DOSE CHANGES AND CESSATION OF MEDICAL TREATMENT

AED serum concentration is often measured to ensure that the dose taken gives a concentration within the therapeutic range for the medicine. It is important to emphasize that this does not guarantee effective treatment. Only observation over time can confirm that the treatment is effective.

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[339] Hauser WA, Rich SS, Annegers JF, Anderson VE. Seizure recurrence after a 1st unprovoked seizure: an extended follow-up. Neurology 1990; 40:1163.
[340] Kim LG, Johnson TL, Marson AG, et al. Prediction of risk of seizure recurrence after a single seizure and early epilepsy: further results from the MESS trial. Lancet Neurol 2006; 5:317.
[341] Marson A, Jacoby A, Johnson A, et al. Immediate versus deferred antiepileptic drug treatment for early epilepsy and single seizures: a randomised controlled trial. Lancet 2005; 365:2007.
[342] Randomized clinical trial on the efficacy of antiepileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure. First Seizure Trial Group (FIR.S.T. Group). Neurology 1993; 43:478.
[343] Hauser WA, Rich SS, Lee JR, et al. Risk of recurrent seizures after two unprovoked seizures. N Engl J Med 1998; 338:429.
[344] Berg AT. Risk of recurrence after a first unprovoked seizure. Epilepsia 2008; 49 Suppl 1:13.
[345] Sirven JI, Evaluation and management of drug-resistant epilepsy. UpToDate – last updated Aug 28, 2014. Accessed March 5, 2015.

In principle there is no difference between reduction of dose and cessation of treatment. Cessation of treatment is reduction of the dose to zero. If the dose of the AED is reduced from what has been effective, a new observation period is needed to ensure that the individual is still without fits.

Treatment with AEDs is usually not discontinued because the patient is «cured». Usually the reduction in dose or cessation of treatment is caused by adverse effects, because the patient has a desire to stop taking the medicines, or that it has been a long time since the last fit.

The below table from Specchio et al clearly demonstrates that cessation of treatment will lead to a higher rate of relapse.

Group of patients.	Patients without seizures on different points of time.				
	6 months	12 months	24 months	36 months	60 months
Patients who have ceased to take medication	88%	74%	57%	51%	48%
Patients on continuous medication	95%	91%	82%	80%	68%

Specchio LM, Tramacere L, La Neve A, Beghi E: Discontinuing antiepileptic drugs in patients who are seizure free on monotherapy. J Neurol Neurosurg Psychiatry. 2002;72(1):22.] The table shows that after 2 years 43% of those who have discontinued treatment have suffered new seizures, whilst 18% of those still on treatment have got further seizures. Even an 18% likelihood over a two year period is a moderate risk, and a 43% likelihood in two years is very high. This means that there is a need for sobriety in the assessment of risk connected to epilepsy in persons on ships.

Reviewed 2014

17.7.2 MIGRAINE

G 43	Migraine (frequent attacks causing incapacity) Likelihood of disabling recurrences	P – Frequent attacks leading to impairment	R – As appropriate if capable of only limited duties	No incapacitating adverse effects while at sea. No incidents during previous periods of sea service

Migraine is a common condition which affects up to 12% of the population[346]. It is more common in females than in males, affecting up to 17% of females and 6% of males. Migraine is

[346] Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M: Prevalence and burden of migraine in the United States: data from the American Migraine Study II. Headache. 2001;41(7):646

more common at an age of 30-39 and is more common in certain families[347]. Migraine without aura is the commonest type, accounting for approximately 75% of cases.

17.7.2.1 CLASSIFICATION OF MIGRAINE

Many conditions with unexplained neurologic symptoms have been called migraine variant or migraine equivalent, however most of these are probably not related to migraine. Some well-defined subtypes of migraine are agreed:

- Migraine with brainstem aura
- Hemiplegic migraine
- Retinal migraine
- Chronic migraine

17.7.2.2 PRECIPITATING FACTORS

- Most individuals suffering from migraine report that attacks are triggered - in a study from Kelman L of 1750 patients, approximately 75 % reported at least one trigger. The following factors were found to be migraine triggers [348]:
- Emotional stress (80%)
- Hormones in women (86%)
- Not eating (57%)
- Weather (53%)
- Sleep disturbances (50%)
- Odors (44%)
- Neck pain (38%)
- Lights (38%)
- Alcohol (38%)
- Smoke (36%)
- Sleeping late (32%)
- Heat (30%)
- Food (27%)
- Exercize (22%)
- Sexual activity (5%)

Obesity has been associated with an increased frequency and severity of migraine[349] [350] [351].

[347] Stewart WF, Schechter A, Rasmussen BK: Migraine Prevalence. Areview of population-based studies. Neurology. 1994;44(6 Suppl 4):S17 og Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF, AMPP Advisory Group: Migraine Prevalence, disease burden, and the need for preventive therapy. Neurology. 2007:68(5):343

[348] The triggers of precipitants of the acute migraine attack - Cephalalgia 2007;27(5):394

[349] Bigal ME, Liberman JN, Lipton RB: ”Obesity and migraine: a population study. Neurology. 2006;664(4):545

[350] Bigal ME, Lipton RB: ”Obesity is a risk factor for transformed migraine but not chronic tension-type headache.” Neurology. 2006;67(2):252

[351] Bigal ME, Tsang A, Loder E, Serrano D, Reed ML, Lipton RB: “Body mass index and episodic headaches: a population-based study”. Arch Intern Med. 2007;167(18):1964

17.7.2.3 MIGRAINE WITH BRAINSTEM AURA

Uncommon type with primary signs from the brainstem without weakness, also called basilar-type migraine. Aura consists of vertigo, dysarthria, tinnitus, diplopia, ataxia, devreased level of consciousness and hypacusis.

17.7.2.4 RETINAL MIGRAINE

Uncommon type characterized with repeated attacks of monocular scotomoata or blindness lasting less than one hour, associated with or followed by headache.

17.7.2.5 HEMIPLEGIC MIGRAINE

Hemiplegic migraine is characterized by attacks with motor weakness during the aura phase. Attacks include severe headache, scintillating scotoma, visual field defects, numbness, paresthesia, unilateral weakness, aphasia, fever, lethargy, coma and seizures. Symptoms can last for hours to days, rarely weeks, but usually resolve completely[352].

Most patients will be unable to carry out any duty on board during episodes. Some individuals must be taken care of by others. Some will need treatment which is not available on board ship.

17.7.2.6 CHRONIC MIGRAINE

Among patients with episodic migraine, transformation to chronic migraine occurs in approximately 3% per year[353]. Chronic migraine may revert to episodic migraine over time in 26-70% of patients[354] [355].

17.7.2.7 FREQUENCY OF ATTACKS

The mean frequency of attacks is three per year. However, the attack frequency is quite variable and ranges from a few per lifetime to 250 per year[356]. In many patients, the frequency of attacks falls after age 50 years, and hemiplegic attacks can evolve into more typical migraine attacks without hemiparesis[357] [358]. Even if most attacks occure without reported triggers, some attacks are precipitated by factors mentioned above.

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[352] Thomsen LL, Oleen J, Sporadic hemiplegic migraine. Cephalalgia 2004; 24:1016.
[353] Bigal, ME, Serrano D, Buse D, et al. Acute migraine medications and evolution from episodic to chronic migraine: a longitudinal populationa-based study. Headache 2008; 48:1157.
[354] Seok JI, Cho HI, Chung CS. From trhansformed migraine to episodic migraine: reversion factors. Headache 2006: 46:1186.
[355] Manack A, Buse DC, Serrano D, et al. Rates, predictors and consequences of remission from chronic migraine to episodic migraine. Neurology 2011; 76:711.
[356] Terwindt G, Kors E, Haan J, et al: Mutation analysis of the CACNA1A calcium channel subunit gene in 27 patients with sporadic hemiplegic migraine. Arch Neurol 2002; 59:1016.
[357] Ducros A, Denier C, Joutel A, et al. The clinical spectrum of familial hemiplecig migraine associated with mutations in a neuronal calcium channel. N Engl J Med 2001; 345:17.
[358] Bradshaw P, Parsons M. Hemiplegic Migraine, a clinical study. Q J Med 1965; 34:65.

17.7.2.8 ATTACKS

Prodromal phase is seen in 60% and consist of affective or vegetative symptoms occurring 24-48 hours prior to onset of headache. Frequent reported symptoms are euphoria, depression, irritability, food cravings, constipation, neck stiffness and increased yawning[359].

Aura: About 25% have one or more focal neurologic symptoms in the second phase, called migraine aura. Even if the traditional view is that aura precedes the headache, prospective data suggest that most patients with migraine experience headache during the aura phase[360]. Auras usually are a mixture of positive and negative features, usually visual, but can also be sensory, verbal or include motor disturbances. The development is quite typical and different from what is seen in stroke or TIA[361].

Migraine headache: usually unilateral and pulsating and frequently associated with nausea and sometimes vomiting. Photophobia and phonophobia is frequently seen, leading to a need to lying down in a darkened, quiet room[362] [363] [364] [365]. Untreated attacks lasts from four hours to several days. Many attacks resolves during sleep. An individual will not be able to carry out any duties during an attack.

Postdromal phase: During this phase sudden head movement transiently causes pain in the location of the antecedent headache. Individuals often feel drained and exhausted, although some report a feeling of mild elation of euophoria. Most individuals will not be able to work effectively and safely in this phase.

17.7.2.9 COMPLICATIONS

These are characterized by attacks associated with prolonged symptoms or, rarely, with infarction or seizures. Prolonged symptoms may last for the entire headache, for several days or weeks, or in some cases leave a permanent neurologic deficit. Status migrainosus, persistent aura without infarction, migrainous infarction and migraine aura-triggered seizures are the more important complications. Complications are more often seen in hemiplegic migraine (and basilar and ophtalmoplegic migraine) than in other types.

17.7.2.10TREATMENT

Many different drugs are used in the treatment of migraine. The efficacy varies from individual to individual and with the migraine subtype. Observed effect is the only way to conclude that the

[359] Kelman L: The premonitory symptoms (prodrome): a tertiary care study of 893 migraineurs. Headache. 2004;44(9):865.
[360] Hansen JM, Lipton RB, Dodick DW, et al. Migraine headache is present in the aura phase: a prospective study. Neurology 2012; 79:2044.
[361] Cutrer FM, Huerter K: Migraine aura. Neurologist. 2007;13(3):118
[362] Charles A. The evolution of a migraine attack - a review of recent evidence. Headache 2013; 53:413.
[363] Silberstein SD. Migraine symptoms: results of a survey of self-reported migraineurs. Headache 1995; 35:387.
[364] Kelman L, Tanis D. The relationship between migraine pain and other associated symptoms. Cephalalgia 2006; 26:548.
[365] Wang YF, Fuh JL, Chen SP, et al. Clinical correlates and diagnostic utility of osmophobia in migraine. Cephalalgia 2012; 32:1180.

treatment is sufficient. Regular daily lifestyle, regular meals, regular sleep, avoidance of dehydration and avoidance of precipitating factors are important parts of the treatment. Shipboard rhythm sometimes can make this difficult, and precipitate attacks.

17.7.2.11 SAFETY RISK ASSESSMENT

A migraine attack usually make the person incapable of carrying out his/her duties. This can be a serious threat, on the bridge watch or when having safety-critical duties and may be critical in cases of low manning, or lone watch-keeping. Attacks usually resolve on treatment or spontaneously even without treatment, but this can take hours or days. This may lead to an unacceptable burden on others, or be critical if the person is the only one with his professional competence on board. The individual risk assessment must take into account the manning of the ship, the position, the job tasks etc.

Reviewed 2014

17.7.3 SLEEP-APNOEA

G 47	Sleep apnoea Fatigue and episodes of sleep while working.	T – Until treatment started and succesful for three months. P – Treatment unsuccessful or not being complied with	L – Once treatment demonstrably working effectively for three months, including compliance with CPAP (continuous positive airway pressure) machine use confirned. Six-monthly assessments of compliance based on CPAP machine recording	Case-by-case assessment based on job and emergency requirements, informed by specialist advice.

Sleep apnoea is a common, chronic condition with a prevalance estimated at 15 – 30% in males and 5 – 15% in females in North America, depending on the criteria used for diagnosis[366]. Common risk factors include obesity, male gender, advancing age and upper airway soft tissue abnormalities. Other factors include smoking, medical conditions eg congestive cardiac failure, chronic lung disease, acromegaly and hypothyroidism.The prevalence also varies with race and ethnicity with African Americans and Asian populations having higher rates than the Caucasians of the same age group and BMI. Patients most commonly complain of snoring and daytime sleepiness although these are non specific for diagnosis. In a systematic review it was found that the most useful finding for diagnosis was nocturnal choking or gasping.[367]. Additional symptoms may include restless sleep, periods of silence terminated by loud snoring, fatigue, poor concentration, nocturnhal angina, nocturia and morning headaches. Common findings on

[366] Young T, Palta M, Dempsey J, Peppard PE, Nieto FJ, Hla KM, Burden of sleep apnea: rationale, design, and major findings of the Wisconsin Sleep Cohort study. WMJ. 2009;108(5):246

[367] Myers KA, Mrkobrada M, Simel DL; Does this patient have obstructive sleep apnea?: The Rational Clinical Examination systematic review. JAMA. 2013 Aug;310(7):731-41

examination include obesity, a crowded oropharyngeal airway, large neck circumference and hypertension.

The severity of sleep apnoea can be graded using the Apnoea-Hypopnoea Index (AHI) into mild (5 – 15 respiratory events per hour of sleep), moderate (15 – 30 respiratory events per hour of sleep) and severe (AHI greater than 30 respiratory events per hour of sleep).

The risks of sleep apnoea increase with the severity of disease and range from decreased daytime alertness to cardiovascular morbidities and mortality eg hypertension, coronary artery disease, cardiac arrhythmias, heart failure and stroke. Daily function may be impaired by excessive daytime sleepiness, inattention and fatigue which can induce or exacerbate cognitive deficits and increase the likelihood of errors and accidents. In particular motor vehicle crashes are two to three times more common in people with sleep apnoea and this represents and an impact on morbidity and mortality similar to the cardiovascular sequelae[368]. Successful treatment improves driving simulator performance and decreases motor vehicle crashes.

Weight loss and continuous positive airway pressure (CPAP) therapy are the main stays of treatment. Patients should also avoid alcohol, even during the daytime and avoid certain medications such as benzodiazepines. Whilst rarely leading to complete resolution of sleep apneoa, weight loss has been shown to improve overall health, decrease the AHI and probably decrease daytime sleepiness[369]. There is evidence that CPAP has beneficial effects across a range of symptoms and severity of disease. In a meta-analysis of 22 randomized trials (1160 patients) that compared nocturnal CPAP with a control, nocturnal CPAP significantly improved both subjective and objective sleepiness, quality of life, cognitive function, and depression[370]

A person with sleep apnoea must demonstrate a subjective and objective response to treatment, be compliant with treatment and subject to regular specialist review before being considered as fit to go to sea. Certificates will need to be restricted or time limited as appropriate and after discussion with the specialist and additional consideration must be given to the safety function of the person's role.

17.7.4 NARCOLEPSY

G 47	Narcolepsy Fatigue and episodes of sleep whilst working	T – Until controlled by treatment for at least two years P – Treatment unsuccessful or not being complied with	R, L – Near coastal waters and no watchkeeping duties, if specialist confirms full control of symptoms for at least two years. Annual review	Not applicable

[368] George CF; Sleep apnea, alertness, and motor vehicle crashes. Am J Respir Crit Care Med. 2007;176(10):954
[369] Smith PL, Gold AR, Meyers DA, Haponik EF, Bleecker ER; Weight loss in mildly to moderately obese patients with obstructive sleep apnea: Ann Intern Med. 1985;103(6 (Pt 1)):850
[370] Giles TL, Lasserson TJ, Smith BJ, White J, Wright J, Cates CJ; Continuous positive airways pressure for obstructive sleep apnoea in adults: Cochrane Database Syst Rev. 2006

Narcolepsy is a clinical syndrome of daytime sleepiness with cataplexy, hypnagogic hallucinations and sleep paralysis although only one third of patients will have all four symptoms. Narcolepsy type 1 (narcolepsy with cataplexy) is estimated to have a prevalence of 25 to 50 per 100,000 people and an incidence of 0.74 per 100,000 person-years[371] It is equally common amongst men and women and classically begins in the teens and early twenties but can present as early as five and after forty years old. Other features include chronic fatigue or tiredness, poor performance at work, poor memory and concentration, car accidents, slurred speech, blurred vision, irregular breathing pattern and sleep attacks. People with narcolepsy are managed with non pharmacological approaches including general lifestyle measures and sleep hygiene. Pharmocological treatment is aimed to reduce excessive daytime sleepiness and cataplexy[372]. Psycho social support is essential and patients are followed up every 6 to 12 monts. The residual sleepiness on treatment is monitored using the multiple sleep latency test[373] or maintenance of wakefulness test but despite treatment daytime performance rarely normalises[374]. In most patients symptoms remain stable. Consideration of a fitness certificate can only be considered with restrictions and time limitations under specialist follow up.

Reviewed 2015

17.7.5 OTHER ORGANIC NEUROLOGICAL DISEASE

G 00-99	Other organic nervous disease, e.g. multiple sclerosis, Parkinson's disease. Recurrence/progression. Limitations on muscular power, balance, coordination and mobility T	T – Until diagnosed and stable P – If limitations affect ability to reliably perform work safely and effectively or unable to meet physical capability requirements (C – Physical capability requirements)	R, L – Case-by-case assessment based on job and emergency requirements, informed by specialist advice	Case-by-case assessment based on job and emergency requirements, informed by specialist advice

17.7.5.1 MULTIPLE SCLEROSIS

DISEASE PATTERNS

No clinical findings are unique to MS, but many may be highly characteristic of the disease. There are several disease patterns.

[371] Longstreth WT Jr, Koepsell TD, Ton TG, Hendrickson AF, van Belle G; The epidemiology of narcolepsy. Sleep. 2007;30(1):13
[372] Wise MS,Arand DL, Auger RR et el; American Academy of Sleep Medicine:Treatment of narcolepsy and other hypersomnias of central origin:Sleep 2007;30:172-1727.
[373] Arnand D, Bonnet M, Hurwitz T et al; The clinical use of the MSLT and MWT:Sleep. 2005;28:123-144
[374] Miller MM, Hajdukovic R, Erman MK; Treatment of narcolepsy with methamphetamine: Sleep.1993;16:306-317.

Clinically isolated syndrome (CIS)

This is the first attack of a disease compatible with MS (eg. Optic neuritis, brainstem syndromes, transvere myelitis) that exhibits characteristics of inflammatory demyelination but has yet to fulfill MS diagnostic critieria[375]. For patients with CIS who have MRI lesions at baseline, the long-term (ie ≥ 10 year) likelihood of developing MS is ≥ 60%. In CIS-patients who have a normal baseline MRI, the long-term likelihood of developing MS is approximately 20%[376].

Relapsing-Remitting (RRMS)

This type is characterized by cleraly defined relapses with full recovery or with sequelae and residual deficit upon recovery. There is no disease progression during the periods between disease relapses. This type of MS accounts for approximately 85-90% of cases at onset[377]. The most common clinical presentation is a spinal cord syndrome with spastic paraparesis and no clear sensory level. Most patients with RRMS will eventually enter a secondary progressive phase as discussed below.

Secondary progressive (SPMS)

Secondary progressive multiple sclerosis is characterized by an initial RRMS disease course followed by gradual worsening with or without occasional relapses, minor remissions and plateaus. The transition from RRMS to SPMS usually occurs 10-20 years after disease onset. However, there are no established criteria to determine when RRMS converts to SPMS and the diagnosis of SPMS is made retrospectively[378]. In one report, the median time from the first symptoms of MS (a clinically isolated syndrom or CIS) to the development of SPMS was 19 years, while the median time from MS diagnosis to SPMS was 12 years[379].

Primary progressive (PPMS)

Primary progressive multiple sclerosis is characterized by progressive accumulation of disability from disease onset with occasional plateaus, temporary minor improvements or acute relapses still consistent with the definition. PPMS represents about 10% of MS cases at disease onset[380]. The most common clinical presentation is a spinal cord syndrome with spastic paraparesis and no clear sensory level[381]. These patients have a more even sex distribution than RRMS, tend to have a later age of onset, and may have a worse prognosis for ultimate disability in comparison with patients who have RRMS.

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[375] Lublin FD, Reingold SC, Cohen JA, et al. Defining the clinical course of multiple sclerosis: the 2013 revisions. Neurology 2014; 83:278.
[376] Olek MJ. Clinically isolated syndromes suggestive of multiple sclerosis. UpToDate, last updated Dec 11, 2014. Accessed March 6 2015.
[377] Weinshenker BG. Natural history of multiple sclerosis. Ann Neurol 1994; 36 Suppl:S6.
[378] Lublin FD, Reingold SC, Cohen JA, et al. Defining the clinical course of multiple sclerosis: the 2013 revisions. Neurology 2014; 83:278.
[379] Eriksson M, Andersen O, Runmarker B. Long-term follow up of patients with clinically isolated syndromes, relapsing-remitting and secondary progressive multiple sclerosis. Mult Scler 2003; 9:260.
[380] Koch M, Kingwell E, Rieckmann P, Tremlett H. The natural history of primary progressive multiple sclerosis. Neurology 2009; 73:1996.
[381] Rice CM, Cottrell D, Wilkins A, Scolding NJ. Primary progressive multiple sclerosis: progress and challenges. J Neurol Neurosurg Psychiatry 2013; 84:1100.

RATE OF WORSENING

Worsening of disability due to MS is highly variable[382], but accumulating evidence suggests that worsening in most patients with MS is slow[383] [384] [385] [386] [387] [388]. One of the largest longitudinal studies followed 2319 patients from British Columbia for 22,723 patient years[389]. Disability scores were prospectively assigned in greater than 95 percent of the patients.

The following observations were reported[390]:

- The median time from disease onset to EDSS 6 (cane needed for walking) was 27.9 years; the median age from birth to EDSS 6 was 59 years
- A primary progressive course was associated with more rapid disease progression than a relapsing course, and was a risk factor in multivariate analysis for time to use of a cane (EDSS 6) from both MS onset (hazard ratio [HR] 2.90, 95% CI 2.39-3.52) and from birth (HR 2.68, 95% CI 2.20-3.26)
- Although men progressed more quickly than women from onset, both men and women required a cane at similar ages (58.8 and 60.1 years), and male sex was not associated with a worse outcome after controlling for other factors
- The type of onset symptoms (eg, motor, sensory, optic neuritis, cerebellar, ataxia, or brainstem) did not predict disease progression after controlling for other factors
- A younger age at onset was associated with slower progression, but patients older at onset were consistently older when they progressed to EDSS 6 than patients younger at onset (figure 1). Similar results were found in a large epidemiology study from France[391].

Some earlier studies suggested that MS was more rapidly progressive. As an example, a 25-year follow-up study of 308 patients with MS found that 50 percent of the patients reached EDSS 6 within 16 years of onset[392].

FREQUENCY OF RELAPSES

Frequency of relapses — The frequency of relapses is highly variable. Summaries of many studies provide an average figure of 0.4 to 0.6 relapses per year. Relapses tend to be more

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[382] Scalfari A, Neuhaus A, Daumer M, et al. Early relapses, onset of progression, and late outcome in multiple sclerosis. JAMA Neurol 2013; 70:214.
[383] Koch M, Kingwell E, Rieckmann P, Tremlett H. The natural history of primary progressive multiple sclerosis. Neurology 2009; 73:1996.
[384] Confavreux C, Vukusic S, Moreau T, Adeleine P. Relapses and progression of disability in multiple sclerosis. N Engl J Med 2000; 343:1430.
[385] Pittock SJ, McClelland RL, Mayr WT, et al. Clinical implications of benign multiple sclerosis: a 20-year population-based follow-up study. Ann Neurol 2004; 56:303.
[386] Tremlett H, Paty D, Devonshire V. Disability progression in multiple sclerosis is slower than previously reported. Neurology 2006; 66:172.
[387] Tremlett H, Zhao Y, Rieckmann P, Hutchinson M. New perspectives in the natural history of multiple sclerosis. Neurology 2010; 74:2004.
[388] Koch M, Kingwell E, Rieckmann P, et al. The natural history of secondary progressive multiple sclerosis. J Neurol Neurosurg Psychiatry 2010; 81:1039.
[389] Tremlett H, Paty D, Devonshire V. Disability progression in multiple sclerosis is slower than previously reported. Neurology 2006; 66:172.
[390] Tremlett H, Paty D, Devonshire V. Disability progression in multiple sclerosis is slower than previously reported. Neurology 2006; 66:172.
[391] Confavreux C, Vukusic S. Age at disability milestones in multiple sclerosis. Brain 2006; 129:595.
[392] Runmarker B, Andersen O. Prognostic factors in a multiple sclerosis incidence cohort with twenty-five years of follow-up. Brain 1993; 116 (Pt 1):117.

frequent during the first years of the disease and wane in later years[393]. Some data suggest that a high relapse frequency in the first two to five years following the diagnosis of MS is associated with increased risk of secondary progression and disability[394] [395] [396].

In a single center study that analyzed data from 2587 relapses occurring in 1078 patients during an average follow-up of 7.4 years, relapses causing permanent disability were rare[397]. Relapses were not associated with starting or stopping interferon treatment.

Relapses of MS may be more common after stressful life events[398] [399] [400]. Perhaps the strongest evidence comes from a meta-analysis of 14 observational studies that found a significant association between stress and MS exacerbations[401]. The authors cautioned that the study does not offer absolute evidence of a causal association.

PROGNOSTIC FACTORS

A variety of factors have been identified as possible prognostic indicators in MS that may modify the disease course or predict exacerbations. However, none are established as reliable, and our ability to accurately predict outcome for individual patients with MS is quite limited[402].

IMPACT OF TREATMENT

In general terms, treatment of acute MS relapses with glucocorticoids improves short-term outcomes but has no known effect on disease activity or long-term disability. Disease modifying drugs are effective for reducing the frequency of relapses in patients with relapsing-remitting MS. However, their benefit for reducing long-term disability is uncertain, as discussed separately. No disease modifying treatments are proven effective for the progressive forms of MS. However, there is evidence that some treatments for SPMS are associated with modest benefit.

SAFETY CONSIDERATIONS

A specialist advice should always be obtained. Questions regarding likelihood for relapse within the timeframe of the validity period should be answered. A unrestricted certificate should

[393] Vollmer T. The natural history of relapses in multiple sclerosis. J Neurol Sci 2007; 256 Suppl 1:S5.
[394] Eriksson M, Andersen O, Runmarker B. Long-term follow up of patients with clinically isolated syndromes, relapsing-remitting and secondary progressive multiple sclerosis. Mult Scler 2003; 9:260.
[395] Ebers GC. Prognostic factors for multiple sclerosis: the importance of natural history studies. J Neurol 2005; 252 Suppl 3:iii15.
[396] Scalfari A, Neuhaus A, Degenhardt A, et al. The natural history of multiple sclerosis: a geographically based study 10: relapses and long-term disability. Brain 2010; 133:1914.
[397] Bejaoui K, Rolak LA. What is the risk of permanent disability from a multiple sclerosis relapse? Neurology 2010; 74:900.
[398] Mohr DC, Hart SL, Julian L, et al. Association between stressful life events and exacerbation in multiple sclerosis: a meta-analysis. BMJ 2004; 328:731.
[399] Goodin DS, Ebers GC, Johnson KP, et al. The relationship of MS to physical trauma and psychological stress: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 1999; 52:1737.
[400] Buljevac D, Hop WC, Reedeker W, et al. Self reported stressful life events and exacerbations in multiple sclerosis: prospective study. BMJ 2003; 327:646.
[401] Mohr DC, Hart SL, Julian L, et al. Association between stressful life events and exacerbation in multiple sclerosis: a meta-analysis. BMJ 2004; 328:731.
[402] Swanton J, Fernando K, Miller D. Early prognosis of multiple sclerosis. Handb Clin Neurol 2014; 122:371.

not be issued. Restricted and limited certificates could be considered in some cases. Other cases will be found to be unfit. The uncertainty regarding the prognosis and likelihood for relapse incidate a «worst-case» way of assessment. Lone watch-keeping should not be allowed.

Reviewed 2015

17.7.6 SYNCOPE AND OTHER DISTURBANCES OF CONCSIOUSNESS

R 55	Syncope and other disturbances of consciousness Recurrence causing injury or loss of control	T – Until investigated to determine cause and to demonstrate control of any underlying condition		
	a) simple faint;	P – If recurrent incidents persist despite full investigation and appropriate treatment		Simple faint; if no new events
	b) not a simple faint, unexplained disturbance; not recurrent and without any detected underlying cardiac, metabolic or neurological cause	T – Four weeks P – If recurrent incidents persist despite full investigation and appropriate treatment	R, L – Case-by-case decision, near-coastal waters with no lone watchkeeping	Three months after event if no recurrences
	c) syncope with recurrent or with possible underlying cardiac, metabolic or neurological cause	T – With possible underlying cause that is not identified or treatable; for six months after event if no recurrences T – With possible underlying cause or cause found and successfully treated; for one month after successful treatment P – For all of above if recurrent incidents persist despite full investigation and appropriate treatment	R, L – Case-by-case assessment, near-coastal waters with no lone watchkeeping	With possible underlying cause but no treatable cause found; one year after event if no recurrences With possible underlying cause found and treated; three months after successful treatment
	d) disturbance of consciousness with features indicating an epileptic seizure. Go to G40-41	P – For all of above if recurrent incidents persist despite full investigation and appropriate treatment		With seizure markers – not applicable

17.7.6.1 ACUTE LOSS OF CONSCIOUSNESS

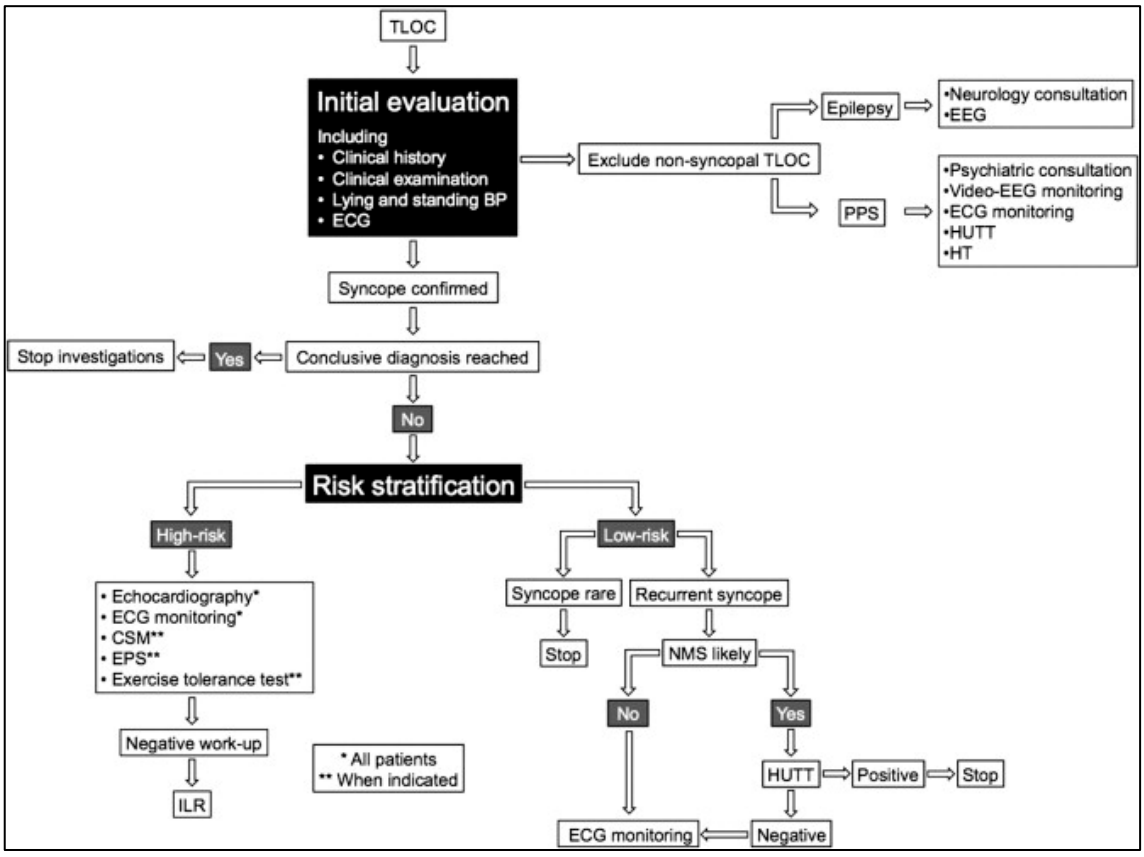
Syncope is an abrupt and transcient loss of consciousness associated with absence of muscular tonus. It usually is a benign and self-limiting condition, even if there may be more serious underlying conditions. In about one third of the cases the syncope results in trauma and relapsing episodes can be a psychological burden.

In a prospective study of 341 patients the different types of syncope showed the following distribution[403]:

- Reflex (neurally-mediated; this includes vasovagal) – 58%
- Cardiac disease, most often a bradyarrhythmia or tachyarrhythmia – 23%
- Neurologic or psychiatric disease – 1%
- Unexplained syncope – 18%; a higher value (41%) was noted in another large series[404]

DIAGNOSIS

The diagnostic process after syncope involves many possible causes and hence, examinations and tests. The following algorithm from Mereu, Sau & Lim, published in Autonomic Neuroscience in 2014, may be useful[405].



Algorithm for the diagnostic management of syncope.
TLOC: transient loss of consciousness, HT: hyperventilation test, HUTT: head-up tilt test, EPS: electrophysiological study, CSM: carotid sinus massage, NMS: neurally mediated syncope, ILR: implanted loop recorder, EEG: electroencephalography.

[403] Alboni P, Brignole M, Menozzi C, Raviele A, Del Rosso A, Dinelli M, Solano A, Bottoni N J Diagnostic value of history in patients with syncope with or without heart disease. Am Coll Cardiol. 2001;37(7):1921.
[404] Kapoor WN. Evaluation and outcome of patients with syncope. Medicine (Baltimore) 1990; 69:160.
[405] Mereu R, Sau A, Lim PB. Diagnostic algorithm for syncope. Aut Neurosc 2014; 184:10-16

Mereu R, Sau A, Lim PB. Diagnostic algorithm for syncope. Aut Neurosc 2014; 184:10-16:

In approximately half of the cases it is possible to find an underlying condition which caused the acute loss of consciousness. Sometimes the underlying condition can be life threatening, eg some arrhythmias, ischemic conditions, cardiac valve disease, pacemaker errors, blood loss, lung embolism or subarachnoid bleeds.

Usual causes include vasovagal syncope (25-65% of all cases[406]), sinus carotid syndrome, orthostasis or medication.

A thorough investigation of the case is necessary to diagnose an underlying condition, if possible. The risk assessment should include the likelihood for a new episode. Cardiological and neurological investigation should usually be undertaken to establish – if possible – the underlying cause.

In the absence of an established underlying disease or when the cause is unknown, «worst-case» assessment should apply[407].

When an underlying cause is known, the risk assessment will be based on this knowledge.

Reviewed 2014

17.7.7 INTRACRANIAL SURGERY/INJURY

T 90	Intracranial surgery/injury including treatment of vascular anomalies or serious head injury with brain damage. Harm to ship, others and self from seizures. Defects in cognitive, sensory or motor function. Recurrence or complication of underlying condition.	T – For one year or longer unttil seizure likelihood low base don advice from specialist P – Continuing impairment from underlying condition or injury or recurrent seizures	R – After at least one year, near coastal, no lone watchkeeping if seizure likelihoods low and no impairment from underlying condition or injury Conditional on continued compliance with any treatment and on periodic review, as recommended by specialist	No impairment from underlying condition or injury, not on any anti-epilepsy medications. Seizure likelihood very low. Condtional on continued compliance with any treatment and on periodic review, as recommended by specialist.

Intracranial surgery and injury can result in a wide spectrum of sequelae and each case must be assessed carefully with due attention to any underlying disease, the surgery/injury sustained, medications required and the ongoing symptoms including but not limited to: seizure activitiy, cognitive status, physical capabilities and emotional sequelae. Despite an extensive search of the

[406] Brignole M, Menozzi C, Bartoletti A, Giada F, Lagi A, Ungar A, Ponassi I, Mussi C, Maggi R, Re G, Furlan R, Rovelli G, Ponzi P, Scivales A: A new management of syncope: prospective systematic guideline-based evaluation of patients referred urgently to general hospitals. Eur Heart J. 2006;27(1):76.

[407] Soteriades ES, Evans JC, Larson MG, Chen MH, Chen L, Benjamin EJ, Levy D: Incidence and prognosis of syncope. N Engl J Med. 2002;347(12):878.

literature we have been unable to quantify the risk and hence individual specialist input is essential.

Reviewed 2015

17.8 H 00-99 DISEASES OF THE EYE AND EAR

17.8.1 EYE DISEASES

H 00-59	Eye disorders – Progressive or recurrent (eg glaucoma, maculopathy, diabetic retinopathy, retinitis pigmentosa, keratoconus, diplopia, blepharospasm, uveiis, corneal ulceration and retinal detachment) Future inability to meet vision standards, risk of recurrence.	T – Temporary inability to meet relevant vision standards (Ch 12) and low likelihood of subsequent deterioration or impairing recurrence once treated or recovered P – Inability to meet relevant vision standards (Ch 12) or, if treated, increased likelihood of subsequent deterioration or impairing recurrence	R – Near coastal waters if recurrence unlikely but forseeable and treatable with earl y medical intervention. L – If risk of progression foresseable but unlikely and can be detected by regular monitoring.	Very low likelihood of recurrence. Progression to a level where visual standard (Ch 12) are not met during period of certificate is very unlikely

All of the information given here must be used in conjunction with the Vision Standards outlined in Chapter 12.

17.8.1.1 GLAUCOMA

Glaucoma is a group of eye diseases traditionally characterized by elevated intraocular pressure (IOP). However it is more accurately defined as an optic neuropathy. It is the second leading cause of blindness world wide (after cataracts).Glaucoma in adults is often generally categorised into open angle or closed angle glaucoma.

OPEN ANGLE GLAUCOMA

This is the most common type of glaucoma amongst people of European or African descent. It is an optic neuropathy characterized by progressive peripheral visual field loss followed by central field loss, in a characteristic pattern. Risk factors include age, race, family history and raised IOP Individuals rarely experience symptoms and the disease is often only diagnosed on visual field testing. The mean progression rate from a full field of vision to blindness takes approximately 25 years in untreated patients[408] and fitness will depend upon full specialist evaluation of visual fields and visual acuity.Time limitation or restriction in trade area may be appropriate to allow

[408] Heijl A, Bengtsson B, Hyman L, Leske MC, Early Manifest Glaucoma Trial Group; Natural history of open-angle glaucoma. Ophthalmology. 2009;116(12):2271

necessary follow-up by ophthalmologist. Visual fields must be checked thoroughly on all medical examinations by the seafarer's doctor.

CLOSED ANGLE GLAUCOMA

Angle-closure glaucoma is characterized by narrowing or closure of the anterior chamber angle. The normal anterior chamber angle provides drainage for the aqueous humor and when this drainage pathway is narrowed or closed, inadequate drainage leads to elevated intraocular pressure and damage to the optic nerve. Acute angle-closure glaucoma occurs in eyes with a certain anatomical predisposition and presents as a painful red eye that must be treated within 24 hours to prevent permanent blindness. All patients with an acute episode require referral to an Opthalmologist and empirical treatment in a primary care setting if the diagnosis is likely and specialist assessment will not be available for over one hour.

Angle closure glaucoma may be primary or secondary. Individuals are anatomically predisposed to primary angle closure and risk factors for developing the disease include family history, age (over 40 to 50 years), female sex, hyperopia, over the counter medications and race[409]. In chronic angle closure glaucoma the rapidity and degree of the intraocular pressure elevation from angle closure determines whether symptoms occur. The patient may not notice damage to the peripheral vision, which generally precedes decrease in central vision and may only be noted at visual field testing. Laser peripheral iridotomy is the first step in treatment of patients with chronic angle-closure glaucoma, to relieve any pupillary block component. The intraocular pressure may remain elevated, however, if scarring has already damaged the drainage angle. In this case, the remaining glaucoma is treated medically and surgically much as in open-angle glaucoma. In cases of secondary angle closure glaucoma treatment is aimed at the cause.

Patients with already diagnosed closed angle glaucoma have a higher risk of further acute episodes, and should not serve on vessels far out at sea.

Patients who have undergone surgical treatment will need ongoing specialist review of their visual acuity, visual fields and IOP. The other eye should also be examined and if a narrow angle is found, prophylactic laser peripheral iridotomy should be performed to prevent future attacks of angle closure.

Untreated, approximately 50% of fellow eyes in acute angle-closure patients will have an angle-closure attack within five years[410].

[409] Traverso CE, Bagnis A, Bricola G. Angle-closure glaucoma. In: Ophthalmology, 2nd ed, Yanoff (Ed), Mosby, 2004. p.1491
[410] Saw SM, Gazzard G, Friedman DS; Interventions for angle-closure glaucoma: an evidence-based update. Ophthalmology. 2003;110(10):1869

17.8.1.2 AGE RELATED MACULAR DEGENERATION

Age-related macular degeneration (AMD) is a degenerative disease of the central portion of the retina (the macula) that results primarily in the loss of central vision. Central vision is required for activities such as driving, reading, watching television or monitors, and performing activities of daily living.

Risk factors for AMD include age (very rare under 55 years[411]), smoking, family history, cardiovascular disease and cataracts. AMD is classified as dry (atrophic) or wet (neovascular or exudative) for clinical purposes. Dry AMD progresses to wet AMD in some patients - the risk of developing wet AMD in people with bilateral early dry AMD (bilateral soft drusen) was estimated at 1.0 to 4.7 percent at one year and 13 to 18 percent at three years[412].

Wet AMD is more common than dry AMD among patients with advanced AMD. Although wet AMD is found in only 10 to 15% of patients with AMD, wet AMD accounts for more than 80% of cases with severe visual loss or legal blindness. In contrast to dry AMD, in which vision loss is slow and gradual, wet AMD is characterized by rapid distortion and loss of central vision over a period of weeks to months. The contralateral eye is at high risk of developing neovascularization, with a cumulative incidence estimated at 10, 28, and 42% at one, three, and five years, respectively[413] . Most patients with advanced AMD do not become completely blind, though significant visual loss results in disability and clinical depression in over one third of patients[414].

17.8.1.3 DIABETIC RETINOPATHY

Diabetic retinopathy (DR) is one of the most important causes of visual loss worldwide, and is the principal cause of impaired vision in patients between 25 and 74 years of age. It is divided into two main forms: non proliferative (NPDR) and proliferative retinopathy (PDR). Visual loss in NPDR is usually progressive due to macular oedema and NPDR can be classified into mild, moderate, severe and very severe categories, primarily relating to the risk of progression to proliferative retinopathy.

The one year risk of progression is 5% for mild disease, 15% for moderate, 52% for severe and 75% for very severe disease .

Prolifereative retinopathy may develop in the setting of prior or coexisting non proliferative changes or may arise without substantial NPDR. It is characterised by the presence of neovascularization arising from the disc and/or retinal vessels and the consequences of this

[411] Smith W, Assink J, Klein R, Mitchell P, Klaver CC, Klein BE, Hofman A, Jensen S, Wang JJ, de Jong PT; Risk factors for age-related macular degeneration: Pooled findings from three continents. Ophthalmology. 2001;108(4):697.
[412] Bressler NM; Age-related macular degeneration is the leading cause of blindness... JAMA. 2004;291(15):1900
[413] Risk factors for choroidal neovascularization in the second eye of patients with juxtafoveal or subfoveal choroidal neovascularization secondary to age-related macular degeneration. Macular Photocoagulation Study Group. Arch Ophthalmol. 1997;115(6):741
[414] Jager RD, Mieler WF, Miller JW; Age-related macular degeneration. N Engl J Med. 2008;358(24):2606

neovascularization, including preretinal and vitreous hemorrhage, subsequent fibrosis, and traction retinal detachment. All of these can lead to a deterioration in vision but this may be acute or chronic and fluctuant or permanent. The severity of proliferative retinopathy can be classified as early, high risk, and severe. In early PDR, new vessels are present as fine loops or networks, but they do not meet the criteria for the high risk category.

There is a 75% five-year risk of progression from early to high risk stages. High risk PDR is defined by moderate to severe neovascularization of the optic disc (greater than one-third to one-half disc area), any neovascularization of the optic disc if vitreous or preretinal hemorrhage is present, or moderate to severe neovascularization elsewhere on the retina (at least one-half disc area) if vitreous or preretinal hemorrhage is present. Untreated high risk proliferative retinopathy results in a 60% risk of severe vision loss at five years[415]. Macular oedema can be present with any degree of proliferative retinopathy and should be addressed as part of the overall treatment strategy.

When assessing the fitness of a person with diabetic retinopathy to serve on board a ship it is important to carry out a full risk assessment of their disease, not just the eye manifestations.

17.8.1.4 RETINITIS PIGMENTOSA

Retinitis pigmentosa (RP) comprises a complex group of inherited dystrophies characterized by progressive degeneration and dysfunction of the retina. It may occur as part of a syndrome or sporadically. A family history of RP is present in about 70 % of patients and the worldwide prevalence is estimated at 1 in 4000 to 5000. Night and peripheral vision are lost progressively, leading to a constricted visual field and markedly diminished vision in some patients. Presentation is variable with some experiencing significant visual loss in childhood whilst others remain asymptomatic will into adulthood. However most patients reach the criteria for legal blindness by the age of 40 due to restrictions in the visual field. [416]

Night blindness (nyctalopia) is one of the earliest symptoms and patients may notice that they become disoriented in dim light, or that adaptation to dim light is slow. However, night blindness may go unrecognized until the disease is advanced and many do not complain of this symptom at all [417]. Progressive constriction of the visual field is another common feature and patients may be considered “clumsy” before the diagnosis is made[418]. In two longitudinal studies of patients with RP, followed for three and nine years, the visual field diminished at a rate of 4.6 to 12 % per

[415] Aiello LM; Perspectives on diabetic retinopathy. Am J Ophthalmol. 2003;136(1):122
[416] Hartong DT, Berson EL, Dryja TP; Retinitis pigmentosa. Lancet. 2006;368(9549):1795.
[417] Heckenlively JR, Yoser SL, Friedman LH, Oversier JJ; Clinical findings and common symptoms in retinitis pigmentosa. Am J Ophthalmol. 1988;105(5):504.
[418] Pagon RA; Retinitis pigmentosa. Surv Ophthalmol. 1988;33(3):137

year[419] [420].Visual acuity is variably affected although eventually most patients experience some loss.

Treatment options are limited and all patients with RP should be under the care of an ophthalmologist who specializes in hereditary eye disease. An individualised risk assessment must be carried on in close cooperation with the treating specialist.

17.8.1.5 KERATOCONUS

Keratoconus is an eye condition in which the normally round dome-shaped cornea progressively thins causing a cone-shaped bulge to develop. This impairs the ability of the eye to focus and causes a loss of visual acuity although the changes may take many years to develop. Exactly why this happens is unknown, but genetic factors play a role and it is more common in people with allergic diseases such as asthma, in Down's syndrome and in some disorders of connective tissue such as Marfan's disease. It affects up to one in 1,000 people and is more common in people of Asian heritage. It is usually diagnosed in teenagers and young people. The condition may be managed with contact lenses although a corneal transplant may be required[421]. An individualise risk assessment with specialist input must be completed.

17.8.1.6 DIPLOPIA

Binocular diplopia (double vision with both eyes open and absent when either eye is closed) often results from dysfunction of one or more of the extraocular muscles. In contrast monocular diplopia, which persists when one eye is closed, suggests local eye disease or a refractive problem. There are a wide range of aetilogies of both and treatment depends on the cause. A full, expert Opthalmology assessment is required and the results should form the basis of a thorough individualized risk assessment.

17.8.1.7 BLEPHAROSPASM

Blepharospasm is a focal dystonia involving the orbicularis oculi muscles and other periocular muscles. Clinical manifestations include increased blinking and spasms of involuntary eye closure. Symptoms are usually bilateral, synchronous, and symmetric, but may be asymmetric. Blepharospasm may be mild and nondisabling, or it may cause significant disability through interference with vision as a result of the eye closure. Patients with blepharospasm typically complain of increased spasms under conditions of bright light or stress, such as driving a car in traffic. The impact on a person's ability to perform their regular and emergency duties must be

[419] Berson EL, Sandberg MA, Rosner B, Birch DG, Hanson AH; Natural course of retinitis pigmentosa over a three-year interval. Am J Ophthalmol. 1985;99(3):240.
[420] Holopigian K, Greenstein V, Seiple W, Carr RE; Rates of change differ among measures of visual function in patients with retinitis pigmentosa.
[421] http://www.moorfields.nhs.uk/condition/keratoconus

considered and supported by a speciailist opinion an individual risk assessment must be performed.

17.8.1.8 UVEITIS

Uveitis, the process of ocular inflammation, can be classified into anterior uveitis (affecting the anterior uveal tract and synonymous with iriitis) or inflammation affecting structures within the posterior uveal tract eg retinitis, vitiritis, choriditis. It may be divided into four different subsets based on the aetiology: infections, systemic immune-mediated disease, syndromes confined primarily to the eye and masquerade syndromes. The symptoms of uveitis depend upon the portion of the uveal tract that is involved. Anterior uveitis may produce pain and redness, although these symptoms are minimal if inflammation begins insidiously (eg, in juvenile idiopathic arthritis [JIA]) and the degree of visual loss associated with anterior uveitis is variable. By contrast, posterior or intermediate uveitis is more likely to be painless, but may result in visual changes such as floaters or reduced visual acuity. Redness of the eye is not a prominent feature of posterior inflammation unless there is an accompanying anterior uveitis. The clinical course, severity and prognosis for uveitis is related to the underlying cause and an individualized risk assessment must be performed.

17.8.1.9 RETINAL DETACHMENT

Retinal detachments can be rhegmatogenous, caused by a break in the retina or nonrhegmatogenous, caused by leakage or exudation from beneath the retina (exudative RD) or vitreous traction pulling on the retina (traction RD). Nontraumatic rhegmatogenous retinal detachment occurs in approximately 1 in 10,000 people per year[422] and myopia is a major risk factor. Posterior vitreous detachment (PVD) is the most common cause of retinal tears which often lead to rhegmatogenous retinal detachment and this is most common between the ages of 50 to 75 years. Patients with a unilateral PVD are very likely to develop PVD in the other eye; in one series of 51 patients, 90 % developed PVD in the contralateral eye within three years[423] . Patients diagnosed with an uncomplicated PVD have a 3.4 % chance of developing a retinal tear within six weeks[424]. Retinal detachments most commonly present with a sudden increase in floaters which can range from being an inconvenience to a major visual disturbance. The rate of progression of retinal detachment varies depending upon the size of the retinal break, location of the break, and movements of the eye. Large horseshoe retinal tears or giant retinal tears that have persistent vitreoretinal traction will usually allow a retinal detachment to progress over the period of hours to days. In contrast, small horseshoe retinal tears or operculated holes often result in more slowly progressive retinal detachment that can take one to four weeks to

[422] Haimann MH, Burton TC, Brown CK; Epidemiology of retinal detachment. Am J Ophthalmol. 1982;94(5):670.
[423] Hikichi T, Yoshida A; Time course of development of posterior vitreous detachment in the fellow eye after development in the first eye. Ophthalmology. 2004;111(9):1705.
[424] Hollands H, Johnson D, Brox AC, Almeida D, Simel DL, Sharma S; Acute-onset floaters and flashes: is this patient at risk for retinal detachment? JAMA. 2009;302(20):2243

develop[425]. As the retinal detachment progresses from the full-thickness retinal breaks posteriorly towards the macula, the size of the visual field defect will enlarge in a corresponding fashion. Patients will lose the ability to read once the retinal detachment involves the macula or the central area of the retina responsible for reading vision. Without treatment, most symptomatic retinal detachments progress to involve the entire retina and lead to loss of vision.The treatment options, prognosis and risk of recurrence vary with the underlying aetiology so specialist assessment and an individual risk assessment is vital.

Reviewed 2015

17.8.2 OTITIS

H65-67	Otitis external; otitis media Recurrence, risk as infection source in food handlers, problems using hearing protection	T – Until treated P – If chronic discharge from ear in food handler	Case-by-case assessment. Consider effects of heat, humidity and hearing protection use in otitis externa.	Effective treatment and no excess likelihood of recurrence

17.8.2.1 OTITIS EXTERNA

Otitis externa (external otitis, swimmer's ear) is an inflammation of the external auditory canal and may be secondary to infectious, allergic or dermatological disease. Acute bacterial infection is the most common cause[426]. It is estimated to have a lifetime incidence of 10%[427] and is known to affect people of all ages. It is found to peak in the 7 – 12 year old age range and to decline in incidence in people over 50 years of age[428]. In a study done in the UK, the 12-month prevalence of otitis externa was >1% and its prevalence was higher for females than for males up to the age of 65 years[429]. In the same study, the incidence of otitis externa increased towards the end of the summer, especially in the youngest age group (5-19 years old). It is common in warmer temperatures, high-humidity conditions and after swimming. The causes are often multifactorial with intact skin in the ear canal and cerumen production being protective against infections due to the fact that cerumen produces a slightly acidic pH[430]. However breakdown of skin integrity, insufficient cerumen production, or blockage of the ear canal with cerumen (which promotes water retention) can predispose to infection. Skin integrity can be injured by direct trauma (including excessive or aggressive scratching/cleaning), heat and moisture or persistent water in the ear canal – conditions not uncommon in persons in certain roles.

[425] Byer NE; Natural history of posterior vitreous detachment with early management as the premier line of defense against retinal detachment. Ophthalmology. 1994;101(9):1503.
[426] Stone KE. Otitis externa. Pediatr Rev. 2007;28(2):77.
[427] Rosenfeld RM, Schwartz SR, Cannon CR, et al. American Academy of Otolaryngology-Head and Neck Surgery Foundation. Clinical practice guideline: acute otitis externa. Otolaryngol Head Neck Surg. 2014;150(suppl 1):S1-S24.
[428] Roland PS, Stroman DW. Microbiology of acute otitis externa. Laryngoscope. 2002;112:1166-1177.
[429] Rowlands S, Devalia H, Smith C, et al. Otitis externa in UK general practice: a survey using the UK General Practice Research Database. Br J Gen Pract.
[430] Osguthorpe JD, Nielsen DR. Otitis externa: review and clinical update. Am Fam Physician. 2006;74:1510-1516.

Patients with uncomplicated diffuse otitis externa usually respond to treatment. Between 65% and 90% of patients have clinical resolution within 7 - 10 days, regardless of agent used[431]. Complications include periauricular cellulitis and malignant exernal otitis/necrotizing external otitis. This latter complication, more common in patients with diabetes[432] or who are immunocompromised, is severe and potentially life threatening. It occurs when the infection spreads from the skin to bone and marrow spaces of the skull base and can itself lead to further intracranial complications including meningitis, brain abscess and dural sinus thrombophlebitis[433].

Person's with an otitis externa that does not settle within 7 – 10 days should have a specialist report outlining the presence or absence of any complications and the recommended treatment and follow up plan. A declaration of full fitness is probably not possible until the condition is fully resolved.

Acute otitis media is largely a childhood disease and data regarding it's incidence and prevalence in adult populations is unavailable.

17.8.2.2 OTITIS MEDIA

ACUTE OTITIS MEDIA (AOM)

Infection or inflammation of the middle ear is one of he most common infections although it primarily occurs in childhood. It is largely a self limiting illness that responds well to antibiotic therapy. Because of the risk of complications in adults immediate antibiotic treatment is recommended in older patients. In the days before antibioltics, acute coalescent mastoiditis complicated AOM in approximately 20% of cases[434], however current studies indicate that mastoiditis and other infectious complications develop in adults in less than 0.5% of cases of AOM[435] [436]. Complications may be more common in patients with an altered immune status, abnormal anatomy and/or incomplete treatment. Infection can also result in perforation of the tympanic membrane. This serves to drain a middle ear abscess and relieve middle ear pressure but does require a course of oral and topical antibiotics. Most perforations heal spontaneously in a matter of days although persisitent subjective hearing loss may continue.

[431] Rosenfeld RM, Schwartz SR, Cannon CR, et al. American Academy of Otolaryngology-Head and Neck Surgery Foundation. Clinical practice guideline: acute otitis externa. Otolaryngol Head Neck Surg. 2014;150(suppl 1):S1-S24.
432 Rubin Grandis J, Branstetter BF 4th, Yu VL. The changing face of malignant (necrotising) external otitis: clinical, radiological, and anatomic correlations. Lancet Infect Dis. 2004;4(1):34.
[433] Schwarz GA, Blumenkrantz MJ, Sundmäker WL. Neurologic complications of malignant external otitis. Neurology. 1971;21(11):1077.
[434] HOUSE HP. Otitis media; a comparative study of the results obtained in therapy before and after the introduction of the sulfonamide compounds. Arch Otolaryngol. 1946;43:371.
[435] Hafidh MA, Keogh I, Walsh RM, Walsh M, Rawluk D. Otogenic intracranial complications. a 7-year retrospective review. Am J Otolaryngol. 2006;27(6):390.
[436] Leskinen K, Jero J. Acute complications of otitis media in adults. Clin Otolaryngol. 2005;30(6):511.

A person with an acute otitis media should be declared temporarily unfit until all symptoms have settled and a course of treatment has been completed. If symptoms are slow to settle specialist referral should be sought to exclude any underlying disease or complication.

OTITIS MEDIA WITH EFFUSION (OME)

OME is defined by the presence of middle ear fluid but without signs of acute inflammation or infection. The tympanic membrane is usually not bulging which distinguishes it from an AOM. OME usually follows AOM but may also be secondary to barotrauma, allergy or Eustachian tube dysfunction. Rarely it may be due to obstruction of the nasopharynx by a mass. The presence of OME should prompt specialist referral and assessment before a valid certificate is given. Restrictions or time limitations may be needed if ongoing treatment and follow up is required and if the person is to fly to join the ship he must be deemed fit to fly.

CHRONIC OTITIS MEDIA (COM)

COM is a recurrent ear infection of the middle ear/mastoid air tract in the presence of a tympanic membrane perforation. It can be classified to:

- Benign/inactive – a dry tympanic membrane perforation unassociated with active infection
- Chronic serous otitis media – continuous serous drainage, typically straw coloured
- Chronic suppurative otitis media (CSOM) – persistent purulent discharge through a perforated tympanic membrane.

In children chronic ear disease often follows AOM although the point in time when an AOM becomes a CSOM is debated with ranges from 2 weeks to 3 months . CSOM in adults occurs in patients with a perforated TM that will not heal as a result of Eustachian tube dysfunction (secondary to upper respiratory tract infection or allergic rhinitis) or abnormal patency with either reflux of contents of the nasopharynx or obstruction of drainage from the middle ear.

Investigation of CSOM may involve specialist referral as a cholesteotoma (primary or secondary) should be excluded and any complications identified and treated. These include:

- Mastoiditis: this occurs more commonly in children than adults and has declined rapidly with the use of antibiotic therapy. However it can complicate CSOM with or without cholesteotoma. A study from Turkey reported 25 cases of mastoid abscess in almost 3000 cases of CSOM ie a risk of 0.86% over 9 years .
- Facial nerve palsy: this is usually gradual and in one case series of 709 patients with complicated CSOM it occurred in 14% .
- Intracranial complications: these are the most serious and require immediate intervention. Again they are rare with effective antibiotic treatment (one large study gives an overall rate of 0.1 – 2%) but can include lateral and /or cavernous sinus thrombosis, meningitis and intracranial

abscess. In a retrospective study from Brazil over a 15 year period meningitis and intracranial abscess were the most common .

A person with CSOM should have specialist assessment with the necessary treatment and a specialist report is vital in assessing fitness. A restricted and/or time limited certificate may be warranted to enable approporiate follow up. A declaration of permanent unfitness may be necessary depending upon the role of the person.

Reviewed 2015

17.8.3 EAR DISORDERS

H68-95	Ear disorders: Progressive (e.g. otosclerosis)	T – Temporary inability to meet relevant hearing standards (B – Hearing requirements) and lowiii likelihood of subsequent deterioration or impairing recurrence once treated or recovered P – Inability to meet relevant hearing standards (B – Hearing requirements) or, if treated, increased likelihood or subsequent deterioration or impairing recurrence	L – If recurrence foreseeable but unlikely and it can be detected by regular monitoring	Very low[iii] likelihood of recurrence. Progression to a level where hearing standards (B – Hearing requirement) are not met during period of certificate is very unlikely.

17.8.3.1 OTOSCLEROSIS

Otosclerosis is a bony overgrowth that involves the footplate of the stapes. As the overgrowth develops, the stapes can no longer function as a piston, but rather rocks back and forth and eventually becomes totally fixed. Conduction gradually becomes worse until a maximal conductive hearing loss of 60 dB is reached.

Treatment for otosclerosis and the accompanying hearing loss involves either hearing amplification or surgical stapedectomy.

Any person with a diagnosis of otosclerosis should be assessed thoroughly and a specialist report obtained to document current hearing capability with and without amplification and the likely progression of any hearing loss over the validity period of the certificate. A restricted or time limited certificate or a declaration of unfitness may be necessary.

17.8.3.2 TINNITUS

Tinnitus is a perception of sound in proximity to the head in the absence of an external source. It can be perceived as being within one or both ears, within or around the head, or as an outside distant noise. The sound is often a buzzing, ringing, or hissing, although it can also sound like other noises. It may be continuous or intermittent.

About one third of all people experience tinnitus at least once in their lifetime. The prevalence of tinnitus in adults worldwide has been estimated to be between 10.1% and 14.5%[437], and the condition is more common in people aged between 40 and 70 years old. The incidence of tinnitus is estimated to be 5.7% to 7% per year worldwide[438]. The prevalence of tinnitus in people with noise exposure is higher than in the general population.

The impact of tinnitus on an individual can be significant. Some individuals "experience" tinnitus, while others "suffer from it." Overall, about 25% of tinnitus sufferers report an increase in tinnitus severity over time and it is usually not an occasional phenomenon – 74% of patients in this study reported that their tinnitus was present for more than 26 days per month[439]. Chronic tinnitus is unlikely to remit completely, but often becomes less bothersome over time, especially in the setting of hearing loss.

Tinnitus is a symptom, not a diagnosis and a wide range of diseases can cause tinnitus:

- Otological
- Neurological, such as multiple sclerosis, head trauma
- Metabolic, such as hyperlipidaemia, vitamin B12 deficiency, diabetes mellitus, hyperthyroidism, hypothyroidism
- Psychogenic
- Vascular disorders, such as arterial bruits, venous hums
- Ototoxic medicine, such as aspirin, NSAIDs, aminoglycosides, certain narcotics, phosphodiesterase type 5 inhibitors

Subjective tinnitus is more likely to occur due to otological problems[440] whereas objective tinnitus usually occurs due to the perception of sounds produced by neighbouring structures, such as muscular contraction and vascular noise[441]. Tinnitus symptoms are exacerbated by insomnia and depression and these associated factors should be addressed alongside treatment of the tinnitus itself. Any documented hearing loss should also be addressed.

[437] Tyler RS. Tinnitus hand book of medicine. San Diego, CA: Singular Publishing Group; 2000.
[438] Sanchez L. The epidemiology of tinnitus. Audiological Medicine. 2004;2:8-17.
[439] Stouffer JL, Tyler RS. Characterization of tinnitus by tinnitus patients. J Speech Hear Disord. 1990;55(3):439.
[440] Crummer RW, Hassan GA. Diagnostic approach to tinnitus. Am Fam Physician. 2004;69:120-126.
[441] Seidman MD, Arenberg AG, Shirwany NA. Palatal myoclonus as a cause of objective tinnitus: a report of six cases and a review of the literature. Ear Nose Throat J. 1999;78:292-297.

Once any identified underlying cause has been identified and treated the treatment of tinnitus itself focuses on cognitive behaviour therapy and patient education.

A person with tinnitus must have been assessed for underlying causes and have satisfactory hearing to perform his/her routine and emergency duties. A specialist report may be useful in assessing hearing, impact and current treatment options.

17.8.3.3 PRESBYCUSIS

Presbycusis, or age-related hearing loss, is a common cause of hearing loss in adults worldwide and is characterized by symmetrical progressive loss of hearing over many years. It usually affects the high frequencies of hearing, although its presentation and clinical course can be variable. Presbycusis has a tremendous impact on the quality of life of millions of older individuals and is increasingly prevalent as the population ages .

The prevalence of hearing loss increases with age with up to 80% of functionally significant hearing loss occurring in older adults . In one population cohort study in the US the prevalence of hearing loss as defined by audiometry increased steadily with age :

- •11% at 44 – 54 years
- •25% at 55 – 64 years
- •43% at 65 – 84 years

It is more common in men than women but this may be related to higher levels of noise exposure.

Multiple factors can influence the onset and severity of presbycusis and these include :

- Low socioeconomic status
- Noise exposure
- Ototoxins eg aminoglycosides, chemotherapeutic agents, heavy metals
- Infections
- Smoking
- Hypertension
- Diabetes
- Vascular disease
- Immunologic disorders
- Hormonal factors eg oestrogens

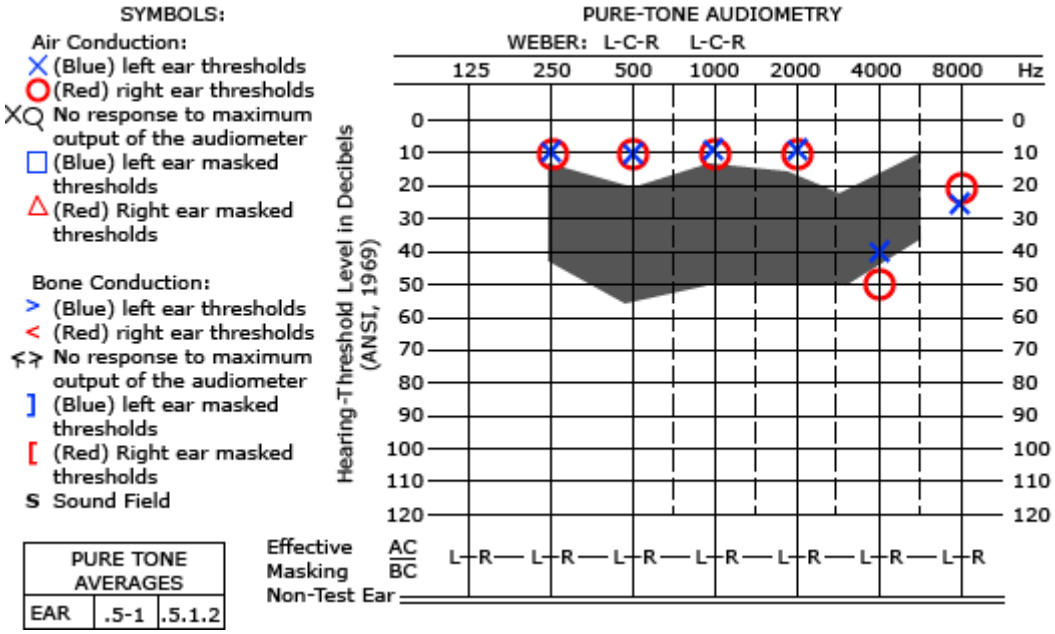
A genetic component also predisposes individuals to age-related hearing loss .

The hallmark of presbycusis is the progressive, symmetric loss of high-frequency hearing over many years and significantly asymmetric hearing loss should be appropriately investigated for other causes. Common complaints associated with presbycusis include the inability to hear or understand speech in a crowded or noisy environment, difficulty understanding consonants, and the inability to hear high pitched voices or noises. Hearing loss can also be accompanied by tinnitus (described as a roaring sound, crickets, or bells in the ear), vertigo, and disequilibrium

leading to falls. Presbycusis can greatly impact quality of life, causing low self-esteem, isolation, and depression and may also be associated with dementia .

NOISE EXPOSURE

Everyday noise exposure, compounded over time, has an impact upon our ability to hear. Excessive noise can ultimately affect the degree of the presbycusis that develops. Constant exposure to loud noises can cause high frequency sensorineural hearing loss.



Given that some persons are exposed to significant noise exposure in certain roles it is wise to bear this in mind in the case of a person with hearing loss.

Any assessment of a person suffering with presbycusis should include hearing and the ability to perform routine and emergency duties alongside the impact of other associated factors such as those mentioned above. A specialist report may be useful in assessing the likely progress of the hearing loss and other symptoms during the validity period.

Reviewed 2015

17.8.4 MÉNIÈRE'S DISEASE AND OTHER FORMS OF CHRONIC OR RECURRENT IMPAIRING VERTIGO

H 81	Ménière's disease and other forms of chronic or recurrent impairing vertigo. Inability to balance, causing loss of mobility and nausea (C – Physical capability requirements)	T – During acute phase P – Frequent attacks leading to impairment	R – If not capable of performing all tasks, but can perform safety-critical duties or compensating measures have been implemented R, L – If frequent specialist surveillance required	Low[iii] likelihood of impairing effects while at sea

Vertigo is a symptom of illusory movement. Some people perceive themselves to be moving whereas others perceive motion of the environment. Vertigo is a symptom, not a diagnosis. It arises because of asymmetry in the vestibular system due to damage to or dysfunction of the labyrinth, vestibular nerve, or central vestibular structures in the brainstem. The causes of vertigo are often classified into central and peripheral causes and these have distinctive clinical features, but with some overlap. Peripheral causes of vertigo generally comprise 80% of cases; of these, benign paroxysmal positional vertigo, vestibular neuritis, and Meniere's disease are the most common[442]. When assessing a person currently suffering from the symtoms of vertigo or with a diagnosis of vertigo due care and attention must be paid as to whether or not the person poses a safety risk to himself, to the vessel or to others and whether he is physically capable of performing his routine or emergency duties.

17.8.4.1 MENIERE'S DISEASE

Meniere disease is a condition that is thought to arise from abnormal fluid and ion homeostasis in the inner ear and manifests as episodic vertigo, tinnitus and hearing loss. It can begin at any age but patients typically present between the ages of 20 – 40 years. It affects both ears and both sexes equally[443]. Reported incidences vary from 4.3 – 100 people per 100 000 with a prevalence of 218 per 100 000 [444] [445]. The incidence of bilateral disease varies in the literature from 2 – 73%[446]. The course of the disease varies greatly between individuals and affected people tend to cycle between active symptoms and periods of remission. Vertigo characteristically persists from 20 minutes to 24 hours and approximately two thirds of patients experience attacks in clusters with the remainder experiencing sporadic attacks. The frequency of attacks may decline over time[447]. The vertigo may lead to an increased risk of falls and decline in the physical capability of the person and this must be considered in the overall risk assessment and decision of fitness. Hearing loss is sensorineural, usually fluctuating and often initially affects the lower frequencies. It progresses over time and often results in permanent hearing loss at all frequencies over an 8 – 10 year period. Tinnitus is characteristically low pitched and may be associated with auditory disturbance. In addition to the impact on physical capability mentioned above the hearing loss and tinnitus associated with the disease must also be thoroughly assessed from a safety perspective.

17.8.4.2 BENIGN PAROXYSMAL POSITIONAL VERTIGO

BPPV is the most common form of positional vertigo and accounts for almost 50% of patients with peripheral vestibular dysfunction. It has a peak incidence between 50 – 70 years of age but

[442] Kroenke K, Hoffman RM, Einstadter D: How common are various causes of dizziness? A critical review. South Med J. 2000;93(2):160.
[443] Perez-Garrigues H, Lopez-Escamez JA, Perez P et al. Time course of episodes of definitive vertigo in Meniere's disease: Arch Otolaryngol Head Neck Surg. 2008;134(11):1149.
[444] Wladislavosky-Waserman P, Facer GW, Mokri B, et al. Meniere's disease: a 30-year epidemiologic and clinical study in Rochester, MN, 1951-1980. Laryngoscope. 1984;94:1098-1102.
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can occur in any age group . A retrospective US study showed an incidence of 64 per 100 000 per year increasing by 38% per decade of life whilst a cross-sectional European study showed a lifetime prevalence amongst the general adult population of 2.4%. This study also showed that BPPV is more common in females (lifetime prevalence 3.2%) than males (1.6%). The overall one year prevalence was 1.6% and the one year incidence was 0.6% . Patients complain of recurrent episodes of vertigo lasting one minute or less recurring periodically for weeks or months without treatment. One third of patients remit at three weeks and the majority by six months after onset . However the recurrence of symptoms is fairly common. One study of 50 patients found a recurrence rate of 18% and 30 % at one and three years respectively whilst another of 103 patients found that 35% had a recurrence by 5 years with recurrent more likely in patients over 40 years or who had suffered symptoms for more than three years prior to treatment.

17.8.4.3 VESTIBULAR NEURITIS

This is a benign disorder, self-limiting and associated with a complete recovery in most cases. However it's symptoms of vertigo, nausea, vomiting and gait impairment may cause significant short term disability. Severe symptoms are likely to resolve in one to two days followed by a more gradual reduction in symptoms and the return of equilibrium – while the acute illness rarely lasts more than a few days problems with imbalance and nonspecific dizziness may persist for months . A person suffering an acute attack should be declared temporarily unfit until the episode has resolved. Usually the condition does not recur, in one study of 103 patients followed over 10 years only 2 cases of recurrence were observed . However there have been studies showing a 15% risk of development of BPPV and a 10% risk of developing panic attacks over 2 years .

Reviewed 2015

17.9 I 00-99 DISEASES OF THE CIRCULATORY SYSTEM

17.9.1 CONGENITAL AND VALVE DISEASE OF HEART

I 05-08	Congenital and valve disease of heart (including surgery for these conditions) Heart murmurs not previously investigated Likelihood of progression, limitations on exercise capacity	T – Until investigated and, if required, treated P – If exercise tolerance reduced or episodes of incapacity occur or if on anticoagulants or if permanent high likelihood of impairing event	R – Near-coastal waters if case-by-case assessment indicates either likelihood of acute complications or rapid progression L – If frequent surveillance required	Heart murmurs – where unaccompanied by other heart abnormalities and considered benign by a specialist cardiologist following examination Other conditions – case-by-case assessment based on cardiologist advice

17.9.1.1 AORTIC STENOSIS

Aortic valve sclerosis is defined as aortic valve thickening and calcification without a significant gradient (defined as an aortic jet velocity <2 m/sec). Aortic stenosis (AS) is present when the antegrade velocity across an abnormal valve is at least 2 m/sec.

The stages of AS are defined by symptoms, valve anatomy, valve hemodynamics, and left ventricular function, see table[448].

Stages of aortic stenosis
Mild stenosis
Aortic valve area > 1.5 cm^2
Mean pressure gradient < 25 mmHg
Aortic V_{max} < 3 m/sec
Moderate stenosis
Aortic valve area 1.0-1.5 cm^2
Mean pressure gradient 25-40 mmHg
Aortic V_{max} 3-4 m/sec
Severe stenosis
Aortic valve area < 1.0 cm^2
Mean pressure gradient > 40 mmHg
Aortic V_{max} > 4 m/sec

The natural history of AS begins with a prolonged asymptomatic period. In general, symptoms in patients with AS and normal left ventricular systolic function rarely occur until the stenosis is severe (valve area is <1.0 cm^2, the jet velocity is over 4.0 m/sec, and/or the mean transvalvular gradient exceeds 40 mmHg). However, many patients do not develop symptoms until critical valve obstruction is present, whilst some patients become symptomatic when the stenosis is less severe, particularly if there is coexisting aortic regurgitation. Thus, serial hemodynamic measurements alone do not identify the time of symptom onset.

Most patients with AS develop symptoms before the onset of left ventricular systolic dysfunction. However, in some patients, there is a reduction in systolic myocardial function and a decrease in the ability of the left ventricle to develop pressure and shorten against a load before the onset of symptoms. At this point, the left ventricle fails, resulting in reductions in stroke

[448] N M Rajamannan, B Gersh, R O Bonow. Calcific aortic stenosis: from bench to the bedside-emerging clinical and cellular concepts. Heart 2003;89:801-805

volume and cardiac output, and eventual heart failure. In addition, the marked elevation in left ventricular pressure can produce or exacerbate mitral regurgitation.

Dyspnoea occurs in 60%, chest pain in 50% and can be impossible to distinguish from coronary heart disease. Syncope is a classical symptom which can be caused by arrhythmia and hypotension. It occurs in 40%[449].

RISK FACTORS FOR PROGRESSION

The rate of progression of the stenotic lesion and the time to onset of symptoms varies significantly among patients. Whether patients at high risk for rapid progression can be successfully identified remains controversial[450]. Several prospective series have attempted to identify risk factors for progression in asymptomatic patients (with symptomatic patients being treated surgically)[451] [452] [453] [454] [455] [456] [457] [458] [459]. Among the factors that may be important are:

- Aortic jet velocity and valve area
- Degree of valve calcification
- Older age
- Male gender
- Cause of AS
- Hypercholesterolemia
- Renal insufficiency
- Hypercalcemia
- Smoking
- Metabolic syndrome
- Diabetes mellitus

[449] Lombard TJ, Selzer A. Valvular aortic stenosis. A clinical and hemodynamic profile of patients. Ann Intern Med. 1987;106:292-298
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[453] Palta S, Pai AM, Gill KS, Pai RG. New insights into the progression of aortic stenosis: implications for secondary prevention. Circulation 2000; 101:2497.
[454] Pellikka PA, Sarano ME, Nishimura RA, et al. Outcome of 622 adults with asymptomatic, hemodynamically significant aortic stenosis during prolonged follow-up. Circulation 2005; 111:3290.
[455] Freeman RV, Otto CM. Spectrum of calcific aortic valve disease: pathogenesis, disease progression, and treatment strategies. Circulation 2005; 111:3316.
[456] Briand M, Lemieux I, Dumesnil JG, et al. Metabolic syndrome negatively influences disease progression and prognosis in aortic stenosis. J Am Coll Cardiol 2006; 47:2229.
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[458] Pohle K, Mäffert R, Ropers D, et al. Progression of aortic valve calcification: association with coronary atherosclerosis and cardiovascular risk factors. Circulation 2001; 104:1927.
[459] Kamalesh M, Ng C, El Masry H, et al. Does diabetes accelerate progression of calcific aortic stenosis? Eur J Echocardiogr 2009; 10:723.

PROGNOSIS OF SYMPTOMATIC AORTIC STENOSIS (AS)

Patients with symptomatic severe AS who do not undergo valve replacement have a poor prognosis[460] [461] [462] [463] [464] [465]. Mortality in patients with AS dramatically increases after the development of the cardiac symptoms. This observation, along with improved survival rates following valve replacement, is the basis for the recommendation for prompt valve replacement in such patients. Poor clinical outcomes in symptomatic AS patients were documented in early studies[466] [467] [468] [469], and have continued to be observed in later series of medically treated patients[470] [471] [472] [473]. In later series, some medically treated symptomatic patients underwent balloon aortic valvuloplasty for palliation, but their clinical outcomes were likely not substantially changed by this procedure, which has been shown not to improve prognosis in adults with severe AS. The high mortality rates observed in symptomatic patients who do not undergo valve replacement may be in part due to comorbidities that preclude surgery.

- A review of studies performed during 1913 to 1970 found that mean survival after onset of heart failure ranged from 0.5 to 2.8 years, after onset of syncope ranged from 0.8 to 3.8 years, and after onset of angina ranged from 2 to 4.7 years[474]. Studies performed during 1967 to 1982 reported two-year actuarial mortality rates of 24 to 69 percent in patients with New York Heart Association functional class III to IV symptoms.
- In the PARTNER trial, 179 patients with AS with heart failure symptoms were assigned to the standard therapy arm[475]. The majority of these patients received balloon aortic valvuloplasty (64 percent during the first 30 days and 20 percent later). The mortality rate at one year was 51 percent in this group.
- In an observational study of symptomatic AS patients not eligible for a transcatheter aortic valve implantation trial, 274 patients received medical treatment (including balloon aortic valvuloplasty in 65 percent)[476]. Mortality was 32 percent during median follow-up of one year.

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[460] Chizner MA, Pearle DL, deLeon AC Jr. The natural history of aortic stenosis in adults. Am Heart J 1980; 99:419.
[461] Ross J Jr, Braunwald E. Aortic stenosis. Circulation 1968; 38:61.
[462] Schwarz F, Baumann P, Manthey J, et al. The effect of aortic valve replacement on survival. Circulation 1982; 66:1105.
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[464] Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010; 363:1597.
[465] Ben-Dor I, Pichard AD, Gonzalez MA, et al. Correlates and causes of death in patients with severe symptomatic aortic stenosis who are not eligible to participate in a clinical trial of transcatheter aortic valve implantation. Circulation 2010; 122:S37.
[466] Turina J, Hess O, Sepulcri F, Krayenbuehl HP. Spontaneous course of aortic valve disease. Eur Heart J 1987; 8:471.
[467] Chizner MA, Pearle DL, deLeon AC Jr. The natural history of aortic stenosis in adults. Am Heart J 1980; 99:419.
[468] Ross J Jr, Braunwald E. Aortic stenosis. Circulation 1968; 38:61.
[469] Schwarz F, Baumann P, Manthey J, et al. The effect of aortic valve replacement on survival. Circulation 1982; 66:1105.
[470] Chizner MA, Pearle DL, deLeon AC Jr. The natural history of aortic stenosis in adults. Am Heart J 1980; 99:419.
[471] Kitai T, Honda S, Okada Y, et al. Clinical outcomes in non-surgically managed patients with very severe versus severe aortic stenosis. Heart 2011; 97:2029.
[472] Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010; 363:1597.
[473] Ben-Dor I, Pichard AD, Gonzalez MA, et al. Correlates and causes of death in patients with severe symptomatic aortic stenosis who are not eligible to participate in a clinical trial of transcatheter aortic valve implantation. Circulation 2010; 122:S37.
[474] Turina J, Hess O, Sepulcri F, Krayenbuehl HP. Spontaneous course of aortic valve disease. Eur Heart J 1987; 8:471.
[475] Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010; 363:1597.
[476] Ben-Dor I, Pichard AD, Gonzalez MA, et al. Correlates and causes of death in patients with severe symptomatic aortic stenosis who are not eligible to participate in a clinical trial of transcatheter aortic valve implantation. Circulation 2010; 122:S37.

MANAGEMENT OF AORTIC STENOSIS

Aortic valve replacement (AVR) for symptomatic aortic stenosis (AS) effectively treats symptoms and prolongs life and should be considered in all patients. Medical management is indicated when valve replacement is not possible or is refused by the patient. No endocarditis prophylaxis is indicated[477].

Adults with severe symptomatic AS should only engage in mild physical activity, as symptoms will be precipitated by even moderate physical exertion.

There is a high risk of sudden death in symptomatic patients who are followed conservatively.

Occurrence of symptoms is important in the prognostic assessment. Mean survival without surgery is 2-3 years. Between 8% and 34 % die abruptly[478].

After successful aortic valve replacement life expectancy is almost normal. Relative survival rate at 5, 10 and 15 years are 99%, 85% and 82%[479] [480].

Although randomized trials comparing surgery to continued medical therapy have not been performed, observational studies have found that corrective surgery in this setting is followed by symptomatic improvement and a substantial increase in survival[481] [482] [483] [484] [485] [486] [487] [488].

The magnitude of benefit from aortic valve replacement in patients with symptomatic AS is illustrated by the following observations:

[477] Bonow RO, Carabello BA, Chatterjee K, et al. 2008 Focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Circulation 2008; 118:e523.

[478] Sorgato A, Faggiano P, Aurigemma GP, et al. Ventricular arrhythmias in adult aortic stenosis: prevalence, mechanisms, and clinical relevance. Chest. 1998;113:482-491

[479] Kvidal P, Bergstrom R, Horte LG, et al. Observed and relative survival after aortic valve replacement. J Am Coll Cardiol. 2000;35:747-756.

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[481] Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014; 63:e57.

[482] Freeman RV, Otto CM. Spectrum of calcific aortic valve disease: pathogenesis, disease progression, and treatment strategies. Circulation 2005; 111:3316.

[483] Smith N, McAnulty JH, Rahimtoola SH. Severe aortic stenosis with impaired left ventricular function and clinical heart failure: results of valve replacement. Circulation 1978; 58:255.

[484] Murphy ES, Lawson RM, Starr A, Rahimtoola SH. Severe aortic stenosis in patients 60 years of age or older: left ventricular function and 10-year survival after valve replacement. Circulation 1981; 64:II184.

[485] Schwarz F, Baumann P, Manthey J, et al. The effect of aortic valve replacement on survival. Circulation 1982; 66:1105.

[486] Lund O. Preoperative risk evaluation and stratification of long-term survival after valve replacement for aortic stenosis. Reasons for earlier operative intervention. Circulation 1990; 82:124.

[487] Kouchoukos NT, Dávila-Román VG, Spray TL, et al. Replacement of the aortic root with a pulmonary autograft in children and young adults with aortic-valve disease. N Engl J Med 1994; 330:1.

[488] Horstkotte D, Loogen F. The natural history of aortic valve stenosis. Eur Heart J 1988; 9 Suppl E:57.

- In a retrospective review of 99 elderly patients with AS and, in almost all, New York Heart Association (NYHA) class III or IV, follow-up at 55 months revealed that 91 percent of survivors were in NYHA class I or II[489].
- In a retrospective study of 144 symptomatic patients, survival at three years was 87 percent in 125 who underwent valve replacement compared to 21 percent in 19 nonoperated patients[490].

COMPLICATIONS

Replacement of a diseased heart valve with a prosthetic valve exchanges the native disease for prosthesis-related complications[491] [492] [493] . The incidence of serious complications in appropriately managed patients is approximately 3% per year. The frequency of various complications depends upon the valve type and position, and multiple clinical risk factors including the adequacy of anticoagulation and the patient's life expectancy.

Prosthetic heart valves are associated with a variety of complications:

- Systemic embolization
- Bleeding
- Valve obstruction due to thrombosis or pannus formation
- Endocarditis
- Structural deterioration, particularly with bioprosthetic valves
- Paravalvular regurgitation
- Hemolytic anemia
- Patient-prosthesis mismatch

FOLLOW-UP

Asymptomatic patients with an aortic V_{max} > 4 m/sec should be seen by a cardiologist every 6 months, and earlier if symptoms occur.

After implantation of mechanic or biological valves, echocardiography is necessary at 2-3 months and 1 year, for biological valves it should also be repeated at 5 years. In addition all individuals who experience symptoms or who develop a murmur should have an echocardiogram. Clinical assessment by a specialist is recommended on an annual basis for asymptomatic patients, and always when symptoms or murmurs arise.

The five main issues in follow-up and management of patients with a prosthetic heart valve:

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[489] Murphy ES, Lawson RM, Starr A, Rahimtoola SH. Severe aortic stenosis in patients 60 years of age or older: left ventricular function and 10-year survival after valve replacement. Circulation 1981; 64:II184.
[490] Schwarz F, Baumann P, Manthey J, et al. The effect of aortic valve replacement on survival. Circulation 1982; 66:1105.
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- Antithrombotic therapy to prevent valve thrombosis and thromboembolism
- Evaluation of valve function and durability
- Endocarditis prophylaxis
- Safety of exercise
- Pregnancy

Thromboembolic and anticoagulation-related problems are by far the most frequent complications of mechanical valves. In contrast, structural failure is relatively rare with these prostheses compared to biprosthetic valves. The long-term likelihood of thromboembolism is generally lower with bioprosthetic valves, though there is an increased likelihood of thromboembolism for mechanical as well as bioprosthetic valves after valve implantation. Other major complications of prosthetic heart valves include endocarditis, paravalvular leak and hemolysis.

SAFETY RISK ASSESSMENT

The risk assessment must take into account the likelihood for sudden deterioration and incapacitation of an individual with diagnosed aortic valve stenosis, even if they are asymptomatic. The restrictions on physical exercise, the need for follow-up, not only at regular intervals, but in case of deterioration should form part of the assessment. In patients with prosthetic valves, there will always be a risk for complications, related to the valves themselves, or to the anticoagulation therapy or possible endocarditis. An unrestricted and unlimited health certificate usually is not applicable.

Reviewed 2014

17.9.2 HYPERTENSION

I 10-15	Hypertension. Increased likelihood of ischaemic heart disease, eye and kidney damage and stroke. Possibility of acute hypertensive episode.	T – normally if > 160 systolic or > 100 diastolic mmHg until investigated and t reate accordance with national international guidelines for hypertension management. P – if persistently > 160 systolic or > 100 diastolic mmHg with or without treatment	L – If additional surveillance needed to ensure levl remains within national guideline limits	If treated in accordance with national guidelines and free from impairing effects from condition or medication

A normal blood pressure is generally accepted to be 120/80mmHg and a measurement above this on two separate occasions in a patient who is not acutely unwell is indicative of hypertension. There are different national and international guidelines in place that give definitions of the degree of hypertension and recommended therapy but these are beyond the scope of this guidance. Hypertension is common with one study estimating that between 29 and 31% of adults

in the US are have high blood pressure[494] however control remains poor with studies estimating that only 46 to 51% of persons with hypertension have their blood pressure under control, defined as a level below 140/90 mmHg[495].

The aetiology of primary and secondary (or identifiable) hypertension vary but there are specific risk factors for primary hypertension that include:

- Race: hypertension is more common in people of Afro-Caribbean origin[496]
- Hypertension in one or both parents
- High or excessive salt intake
- High or excessive alcohol intake
- Physical inactivity, obesity and weight gain
- Dyslipidaemia independent of obesity
- Certain personality traits
- Vitamin D deficiency[497]

A number of conditions may lead to secondary hypertension:

- Renal diseaseand renovascular disease
- Drugs eg oral contraceptive pill, long term non steroidal anti-inflammatories, many anti depressants
- •Cushings syndrome and other endocrine disorders
- Primary aldosteronism
- Coarctation of the aorta

It is the recognized complications of hypertension that pose the biggest risk to the person and to the vessel. The likelihood of developing these complications varies with the blood pressure and the increase in risk begins as the blood pressure rises above 115/75 mmHg in all age groups[498]. However, this relationship does not prove causality, which can only be demonstrated by randomized trials showing benefit from blood pressure reduction.

Risks include:

- Cardiovascular disease – hypertension accounts for 54% of all cerebrovascular accidents and 47% of all ischaemic heart disease[499] and the increase in cardiovascular risk associated with hypertension is importantly affected by the presence or absence of other risk factors[500]

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[496] Carson AP, Howard G, Burke GL, Shea S, Levitan EB, Muntner P; Ethnic differences in hypertension incidence among middle-aged and older adults: the multi-ethnic study of atherosclerosis: Hypertension. 2011 Jun;57(6):1101-7. Epub 2011 Apr 18.

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[499] Lawes CM, Vander Hoorn S, Rodgers A, International Society of Hypertension; Global burden of blood-pressure-related disease, 200: Lancet. 2008;371(9623):1513.

[500] Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK; The progression from hypertension to congestive heart failure: JAMA. 1996;275(20):1557

- Heart failure - the risk of heart failure increases with the degree of blood pressure elevation[501]
- Left ventricular hypertrophy is a common finding in patients with hypertension and is important clinically because it is associated with increases in the incidence of heart failure, ventricular arrhythmias, death following myocardial infarction, decreased LV ejection fraction, sudden cardiac death, aortic root dilation, and a cerebrovascular event. Lowering the blood pressure with anti hypertensive agents or other means decreases the cardiac mass in left ventricular hypertrophy, related both to the anti hypertensive response and in some cases to the type of therapy[502]
- Chronic renal failure and end stage renal disease[503]
- Acute hypertensive emergencies – malignant hypertension and hypertensive encephalopathy. These are acute, life threatening events generally associated with a blood pressure greater than 180/120mmHg[504]

If a person has documented hypertension at the time of the medical examination he/she should be referred back to their own doctor for repeat measurements and appropriate investigation and treatment before a certificate, restricted or not, is issued.

Reviewed 2015

17.9.3 CARDIAC EVENT EG CORONARY HEART DISEASE, MYOCARDIAL INFARCTION ETC

17.9.3.1 ISCHAEMIC HEART DISEASE

I 20-25	Cardiac event, e.g. myocardial infarction, ECG evidence of past myocardial infarction or newly recognized left bundle-branch block, angina, cardiac arrest, coronary artery bypass grafting, coronary angioplasty. Acute impairment or exercise limitation. Problems of managing repeat cardiac event at sea.	T – For three months after initial investigation and treatment, longer if symptoms not resolved P – If criteria for issue of medical certificate not met and further reduction of likelihood of recurrence improbable	L – If excess likelihood of recurrence is very lowiii and fully compliant with risk reduction recommendations and no relevant co-morbidity: Issue six-month medical certificate initially and then annual medical certificate R, L – If likelihood of recurrence is lowiii, restricted to: – no lone working or solo watchkeeping; and – operations in near-coastal waters, unless working on vessel with ship's doctor:	Not applicable.

[501] Levy D, Larson MG, Vasan RS, et al. The progression from hypertension to congestive heart failure. JAMA 1996; 275:1557.
[502] Ruilope LM, Schmieder RE; Left ventricular hypertrophy and clinical outcomes in hypertensive patients: Am J Hypertens. 2008;21(5):500
[503] Hsu CY, McCulloch CE, Darbinian J, Go AS, Iribarren C; Elevated blood pressure and risk of end-stage renal disease in subjects without baseline kidney disease: Arch Intern Med. 2005;165(8):923.

[504] Marik PE, Varon J; Hypertensive crises: challenges and management: Chest. 2007;131(6):1949

			issue six-month medical certificate initially and then annual medical certificate. R, L – If likelihood of recurrence is moderateiii and asymptomatic. Able to meet the physical requirements of their normal and emergency duties: – no lone working or solo watchkeeping; and – operating within one hour of port, unless working on vessel with ship's doctor. Case-by-case assessment Annual review.	

CORONARY RISK FACTORS

The relative importance of risk factors for the development of Ischaemic Heart Disease (IHD) according to age was evaluated in a report in which 11,016 men aged 18 to 39 years were followed for 20 years[505]. The relative risks associated with the traditional risk factors were of similar magnitude as in a group of 8955 men aged 40 to 59 years. These included:

- Age — relative risk 1.63 per six year increase
- Serum cholesterol — relative risk 1.92 per 40 mg/dL [1.04 mmol/L] increase
- Systolic blood pressure — relative risk 1.32 per 20 mmHg increase
- Cigarette smoking — relative risk 1.36 per 10 cigarette/day increase

Smoking — Cigarette smoking is the most common and most modifiable risk factor in young patients. It has been noted in 65 – 92% of young patients suffering a myocardial infarction (MI), compared to 24 – 56% of patients older than 45 years of age[506] [507] [508] [509] [510] [511] [512].

[505] Navas-Nacher EL, Colangelo L, Beam C, Greenland P. Risk factors for coronary heart disease in men 18 to 39 years of age. Ann Intern Med 2001; 134:433.
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When risk factors are identified, the 10-year likelihood for an event should be calculated by one of the generally accepted risk calculators, like the Framingham calculator[513] or similar. The European Society of Cardiology has developed several charts for high- and low-risk countries for age ≥40 years[514].

ISCHAEMIC HEART DISEASE IN THE YOUNG

IHD in individuals below the age of 40 years is frequently a silent process. One study of autopsies in 760 victims aged 15 – 34 years of accidents, suicides and homicides showed advanced athreomata in 2% of men and no women aged 15-19. Avanced lesions were founds in 20% of males and 8% of females aged 30-34, while 19 and 8% respectively had ≥40% stenosis of the left anterior descending artery[515].

The clinical presentation of IHD in younger patients is different from that in older patients. A high proportion of young patients do not experience angina[516], and, in the majority of cases, an acute coronary syndrome that progresses rapidly to MI (most often an ST elevation MI) if left untreated is the first manifestation of IHD[517] [518] [519].

SCREENING FOR SILENT MYOCARDIAL ISCHEMIA

This is the most common form of ischemia, accounting for more than 75% of ischemic episodes[520]. Screening for this can be done in various ways as shown below. Exercise testing appears to be the most readily available laboratory diagnostic test in asymptomatic individuals and those with a history of IHD or exertional angina[521].

- Continuous ECG (Holter) monitoring
- Exercise myocardial perfusion scintigraphy
- Radionuclide angiocardiography
- Pharmacologic stress scintigraphy
- Hemodynamic monitoring

[513] D'Agostino RB Sr, Vasan RS, Pencina MJ, et. al. General Cardiovascular Risk Profile for Use in Primary Care. The Framingham Heart Study. Circulation. 2008 Jan 22.
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Conventional ST segment analysis during exercise treadmill test is moderately sensitive in detecting IHD. However, it has low specificity because of an unacceptably high rate (10- 35%) of false positive responses, particularly in asymptomatic persons and especially in women[522].

Sensitivity is about 60% and with optimal techniques a theoretical specificity could reach 90% although it rarely reaches more than 80%. This means that if the prevalence of IHD in the tested population is 1%, 94% of all "positive tests" will be false positive. If the prevalence is 5% in the tested population, 76% are false positive, and even with a prevalence of 10%, as many as 60% of the "positive" tests are false positive. This means that if all the persons tested have known IHD, the value of testing increases considerably (which is why this test is useful in follow-up of already diagnoses IHD-patients). However, on the other hand, the younger the person being tested and the more likely he or she is to be healthy, the less reliable a positive test will be[523], which makes this test almost useless as a screening test for IHD on apparently healthy individuals without known risk factors.

PCI - RESTENOSIS AND THROMBOSIS

After a successful procedure, coronary stents can fail to maintain vessel patency due to either restenosis or stent thrombosis. Restenosis is a gradual re-narrowing of the stented segment that occurs mostly 3 to 12 months after stent placement. It usually presents as recurrent angina but can present as an acute myocardial infarction in approximately 10% of patients.

FIRST GENERATION STENTS: Angiographic restenosis rate at 6 months after successful stent placement was 32% in the STRESS study, and 22% in the Benestent study. PTCA alone had a restenosis frequency of 42% in the STRESS study and 32% in the Benestent study.[524] [525].

SECOND GENERATION BARE-METAL STENTS: A pooled analysis of 6186 patients from six major clinical trials assessing second generation bare-metal stents was carried out by Cutlip et al. The frequency of clinical restenosis was defined as target lesion or target vessel revascularization beyond 30 days, death, or myocardial infarction in the target vessel territory[526]. At one year, target lesion revascularization (TLR) was performed in 12% and target vessel revascularization in 14.1%. Angiographic restenosis was not equivalent to clinical restenosis. Clinically relevant restenosis occurred in only about half of patients with angiographic restenosis.

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DRUG-ELUTING STENTS: The rate of in-stent restenosis (ISR) has been reported between 3% and 20% , depending on which drug-eluting stent (DES) is evaluated, the duration of follow-up, and the complexity of the lesions in which the stents were placed[527].

- In the j-Cypher registry of nearly 13,000 patients who received a sirolimus-eluting stent (SES), the cumulative incidence of target lesion revascularisation (TLR) within the first year was 7.3%[528]. TLR continued to occur at a rate of 2.2% per year, such that the rate was 15.9% at five years.
- In the Endeavor IV trial, 1548 patients were randomly assigned to either a zotarolimus eluting stent or a paclitaxel-eluting stent (PES). At three years, the rates of TLR were 6.5 and 6.1%, respectively[529].
- In the SIRTAX LATE study, 1012 patients were randomly assigned to either SES or PES[530]. In an analysis of the 444 patients who underwent repeat angiography, the cumulative five-year rates of TLR were 13.1 and 15.1% respectively.

In a review of 1084 patients who underwent follow-up angiography six months after bare-metal stent placement, the incidence of restenosis was as low as 16% in the absence of any risk factors (diabetes, multiple stents, and minimal luminal diameter after stenting <3 mm)[531], and as high as 59% when at least three risk factors were present[532].

CORONARY ARTERY BYPASS GRAFT (CABG)

SAPHENOUS VEIN GRAFT: In the PREVENT IV trial[533] 1828 patients from 100 centres were followed up at 12 and 18 months by angiography. Vein graft failure was seen in 43% of patients and about 25% of the grafts had failed. Late occlusion some time after the first 12 to 18 months occurs when the areas of intimal hyperplasia develop lipid deposition and finally an atherosclerotic-like plaque[534]. From the end of year one to year six, SVGs obstruct at the rate of approximately 2% per year; the subsequent closure rate rises to 4-5% per year.

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INTERNAL THORACIC ARTERY GRAFT: Long-term graft patency is much higher with ITA than venous grafts[535] [536] [537] [538] [539]. ITA graft patency is over 95% at five years and slightly lower at 10 years, particularly if the graft is placed to the LAD[540] [541] [542]. Right ITA graft patency is similar at five years, but falls below 90% at 10 years, particularly if it is placed to the right coronary artery.

TOTAL ARTERIAL CORONARY REVASCULARIZATION: In a review of 3220 patients undergoing total arterial coronary revascularization at the Royal Melbourne Hospital, the operative mortality was 0.7%, and angiographic graft patency was 97% and 89% at five years for left and right ITA grafts and 91% at one year for radial artery grafts (only 65 patients)[543].

PCI BELOW THE AGE OF 40

The long-term outcome outcome of young patients undergoing percutaneous coronary intervention (PCI) is good[544] [545] [546]. In a study that assessed the outcome of PCI in 140 consecutive patients ≤40 years of age, the acute success rate was 93% with a 28% rate of angiographic restenosis[547]. Ten-year overall survival following PCI was 96% and ten-year event-free survival (without MI, elective CABG, or repeat PCI) was 58%.

RISK ASSESSMENT

This means that the likelihood for an incident to occur in the validity period of the medical certificate after PTCA, PCI or CABG is moderate to high in most cases – in some cases very high.

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17.9.3.2 ANTITHROMBOTIC MEDICATION AFTER PCI

STENT THROMBOSIS

Stents are thrombogenic. Older studies demonstrated a thrombosis frequency of 18% (acute or sub-acute).

Stent thrombosis is defined as acute (within 24 hours), subacute (within 30 days) or late (up to 1 year) or very late (more than 1 year).

Stent thrombosis is most frequently seen shortly after the surgical intervention. The incidence within 30 days is 0.5-1.5% for both bare-metal stents and drug-eluting stents (DES)[548] [549] [550]. Late thrombosis (>30 days) is seen more often with DES, where stent thrombosis can occur after several years[551]. In non-selected patients with DES some reports demonstrates stent thrombosis at a yearly rate of 0.6% during three year follow-up[552]. Late stent thrombosis usually present a clinical picture similar to acute myocardial infarction, often with ST-elevations, and mortality is high (16-45%) in several studies[553] [554] [555] [556].

ANTITHROMBOTIC THERAPY

All patients who undergo percutaneous coronary intervention (PCI), including those treated with balloon angioplasty without stenting, receive dual antiplatelet therapy (DAPT), which is the combination of aspirin and a P2Y12 receptor blocker. The rationale for the use of DAPT, as opposed to antiplatelet monotherapy, is derived from the known tendency of circulating blood to

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clot in the presence of many metals. This period of risk decreases after the metal portion of the stent is endothelialized[557] [558] [559] [560].

In early studies of patients who received bare metal stents (BMS), the rate of stent thrombosis was significantly lower with aspirin plus ticlopidine than with aspirin alone (or aspirin plus warfarin)[561] [562] [563] [564] [565].

Anticoagulation with warfarin alone does not provide sufficient protection against stent thrombosis compared to acetylsalicylic acid and tienpyridines, and is not an alternative to antiplatelet treatment[566].

It is now recommended to use DAPT (acetylsalicylic acid + clopidogrel) for 12 months after PCI, and continue acetylsalicylic acid throughout life[567].

In about 10% of the cases of PCI, the individual also needs anticoagulation (warfarin) for various reasons (earlier thrombosis, embolism, valve prosthesis etc.)[568]. In these cases a delicate balance between the likelihood of thrombosis and the likelihood of bleeding must be taken into account.

Reviewed 2014

[557] Airoldi F, Colombo A, Morici N, et al. Incidence and predictors of drug-eluting stent thrombosis during and after discontinuation of thienopyridine treatment. Circulation 2007; 116:745.
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17.9.4 CARDIAC ARRYTHMIAS, PACEMAKER AND ICD

I 44-49	Cardiac arrhythmias and conduction defects (including those with pacemakers and implanted cardioverter defibrillators (ICD)). Likelihood of impairment from recurrence, exercise limitation. Pacemaker/ICD activity may be affected by strong electric fields.	T – Until investigated, treated and adequacy of treatment confirmed P – If disabling symptoms present or excess likelihood of impairment from recurrence, including ICD implant	L – If surveillance needed at shorter intervals and no impairing symptoms present and very lowiii excess likelihood of impairment from recurrence, based on specialist report R – Restrictions on solo duties or for distant waters if lowi likelihood of acute impairment from recurrence or foreseeable requirement for access to specialist care Surveillance and treatment regime to be specified. If pacemaker fitted, duration of medical certificate to coincide with pacemaker surveillance.	Surveillance not needed or needed at intervals of more than two years; no impairing symptoms present; and very low likelihood of impairment from recurrence, based on specialist report

A cardiac arrhythmia is a disturbance in the rate of cardiac muscle contractions, or any variation from the normal rhythm or rate of heart beat. Cardiac arrhythmias may be acute or chronic and are found in a vast range of medical conditions. They may be defined in a number of ways by:

- Site of origin eg supraventricular, atrial, ventricular
- Mechanism of disturbance eg fibrillation, automaticity, re-entry or triggered activity
- Rate of disturbance eg tachycardia, bradycardia
- Electrocardiogram experience eg long QT syndrome

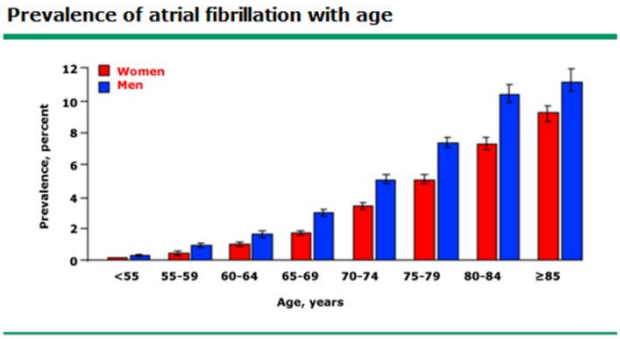
Here we will consider a few of the many arrhythmias which may be seen in clinical practice. A review of the emergency assessment and management of cardiac arrhythmias is beyond the scope of these guidelines and we will focus on the long term management and the impact that such a condition may have on a person's fitness to work at sea.

When assessing a person with a known history of a cardiac arrhythmia the seafarer's doctor should perform a full, individualised risk assessment including requesting specialist input where appropriate. Considerations must include but are not limited to the type of arrhythmia, presenting signs and symptoms, risk of recurrence within the validity period of the certificate, the impact on the person's physical ability to perfom his/her regular or emergency tasks, the acute treatment required in the event of a recurrence, the risk of complications and the treatment required, ongoing medication and any potential side effects, the need for specialist follow up and any other comorbidities.

17.9.4.1 ATRIAL FIBRILLATION

Atrial fibrillation is the most common sustained cardiac arrhythmia in clinical practice.

A systematic review of 184 population based studies from across the world has estimated that in 2010 the number of individuals with AF was 33.5 million and evidence suggests that its incidence and prevalence are increasing[569] [570].



The prevalence of AF depends upon population characteristics and varies with age, sex, race, geography and time period.

AF is uncommon in infants and children and healthy young adults are also at low risk[571]. The prevalence of AF increases with age as demonstrated in the ATRIA study which looked at almost 1.9 million subjects in the US[572]. Overall the prevalence of AF was 1% but this ranged from 0.1% in adults less than 55 years of age to 9% in those greater than 80 years of age. Of all those suffering with AF, 70% were at least 65 years old and 45 % were over 75 years old. Similar results were seen in a European based cohort study of 6808 subjects of 55 years of age or above[573]. The prevalence of AF was 5.5% ranging from 0.7% in those aged 55 – 59 years and 17.8% in those over 85 years. At every age group the prevalence was higher in men than women and both studies os observed that AF was more frequent in whites than those of Afro-Caribbean origin(2.2% vs 1.5% and 6.0% vs 5.1%). Other studies have also observed a lower rate of AF in Afro-Caribbeans, Hispanics and Asians[574]. When looking at geography the age adjusted prevalence rate (per 100 000 populatio) was highest in North America (700 – 775) and lowest in Japan and South Korea (250 – 325) with similar low rates also observed in China (325 – 400).

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The incidence of AF also increases with age[575] [576] and the lifetime risk was studied in the Framingham Heart Study[577]. 8725 patients were followed from 1968 – 1999 and of these 936 developed AF. The risk of developing AF from age 40 to age 95 was 26% in men and 23% in women. Lifetime risk did not change substantially with increasing age because AF incidence also rose with age – the risk of developing AF from age 80 – 95 years was 23% for men and 22% for women.

As most information is derived from clinical visits it is likely that the prevalence of paroxysmal AF is even higher than these figures suggest. Subclinical AF refers to asymptomatic episodes in a patient without a history of prior AF and are also only detected by monitoring techniques. The ASSERT study[578] monitored 2580 patients pover the age of 65 years with a dual chamber pace maker or implantable cardioverter defibrillator) and a history of hypertension but no previous AF, for the development of AF defined as rates >190 bpm for over 6 minutes. It also looked at the relationship between subclinical AF and stroke.

They noted that:

- At three months subclinical AF was noted in about 10% of patients – the medial number of episodes was two and the median time for the event to occure was 36 days.
- At 2.5 years subclinical AF had been noted in 35% of individuals with 16% developing clinical AF.

Hypertensive heart disease and coronary heart disease are the most common underlying disorders in patients with AF in developed countries[579] whilst Rheumatic Heart Disease, although now rare in developed countries, is also associated with a much higher incidence of AF[580].

The most serious complication of AF is aterial thromboembolism and the most clinically evident thromboembolic event is ischemic stroke. Peripheral embolization acconts for less than 10% of all such events and many are subclinical[581]. Antithrombotic therapy has been shown to lower the risk of clinical thromboembolic events in virtually all patients with AF including all levels of risk and type of AF. However for a person this comes with its own issues with regards to fitness and these need to be taken into consideration as part of an individualised risk assessment.

[575] Heeringa J, van der Kuip DA, Hofman A, Kors JA, van Herpen G, Stricker BH, Stijnen T, Lip GY, Witteman JC. Prevalence, incidence and lifetime risk of atrial fibrillation: the Rotterdam study. Eur Heart J. 2006;27(8):949.
[576] Psaty BM, Manolio TA, Kuller LH, Kronmal RA, Cushman M, Fried LP, White R, Furberg CD, Rautaharju PM. Incidence of and risk factors for atrial fibrillation in older adults. Circulation. 1997;96(7):2455.
[577] Lloyd-Jones DM, Wang TJ, Leip EP, Larson MG, Levy D, Vasan RS, D'Agostino RB, Massaro JM, Beiser A, Wolf PA, Benjamin EJ. Lifetime risk for development of atrial fibrillation: the Framingham Heart Study. Circulation. 2004;110(9):1042.
[578] Healey JS, Connolly SJ, Gold MR, Israel CW, Van Gelder IC, Capucci A, Lau CP, Fain E, Yang S, Bailleul C, Morillo CA, Carlson M, Themeles E, Kaufman ES, Hohnloser SH, ASSERT Investigators. Subclinical atrial fibrillation and the risk of stroke. N Engl J Med. 2012;366(2):120.
[579] Krahn AD, Manfreda J, Tate RB, Mathewson FA, Cuddy TE. The natural history of atrial fibrillation: incidence, risk factors, and prognosis in the Manitoba Follow-Up Study. Am J Med. 1995;98(5):476.
[580] Diker E, Aydogdu S, Ozdemir M, Kural T, Polat K, Cehreli S, Erdogan A, Göksel S. Prevalence and predictors of atrial fibrillation in rheumatic valvular heart disease. Am J Cardiol. 1996;77(1):96.
[581] Go AS, Hylek EM, Chang Y, Phillips KA, Henault LE, Capra AM, Jensvold NG, Selby JV, Singer DE.

Comparison of the CHADS2 and CHA2DS2-VASc risk stratification scores for subjects with nonvalvular AF

Definition and scores for $CHADS_2$ and CHA_2DS_2-VASc	
$CHADS_2$ acronym	**Score**
Congestive HF	1
Hypertension	1
Age ≥75 years	1
Diabetes mellitus	1
Stroke/TIA/TE	2
Maximum score	6
CHA_2DS_2-VASc acronym	**Score**
Congestive HF	1
Hypertension	1
Age ≥75 years	2
Diabetes mellitus	1
Stroke/TIA/TE	2
Vascular disease (prior MI, PAD, or aortic plaque)	1
Age 65 to 74 years	1
Sex category (ie, female sex)	1
Maximum score	9

Stroke risk stratification with the $CHADS_2$ and CHA_2DS_2-VASc scores	
$CHADS_2$ acronym	**Unadjusted ischemic stroke rate (% per year)***
0	0.6%
1	3.0%
2	4.2%
3	7.1%
4	11.1%
5	12.5%
6	13.0%
CHA_2DS_2-VASc acronym	**Unadjusted ischemic stroke rate (% per year)***
0	0.2%
1	0.6%
2	2.2%
3	3.2%
4	4.8%
5	7.2%
6	9.7%
7	11.2%
8	10.8%
9	12.2%

AF: atrial fibrillation; $CHADS_2$: Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Prior Stroke or TIA or Thromboembolism (doubled); CHA_2DS_2-VASc: Congestive heart failure, Hypertension, Age ≥75 years (doubled), Diabetes mellitus, Prior Stroke or TIA or thromboembolism (doubled), Vascular disease, Age 65-74 years, Sex category; HF: heart failure; LV: left ventricular; MI: myocardial infarction; PAD: peripheral artery disease; TE: thromboembolic; TIA: transient ischemic attack.
* These unadjusted (not adjusted for possible use of aspirin) stroke rates were published in 2012[1]. Actual rates of stroke in contemporary cohorts might vary from these estimates.

Reference:

1. *Friberg L, Rosenqvist M, Lip GY. Evaluation of risk stratification schemes for ischaemic stroke and bleeding in 182 678 patients with atrial fibrillation: the Swedish Atrial Fibrillation cohort study. Eur Heart J 2012; 33:1500.*

Original figure modified for this publication. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol 2014. DOI: 10.1016/j.jacc.2014.03.022. Table used with the permission of Elsevier Inc. All rights reserved.

The incidence of thromboembolism varies between studies depending upon the population studied, how event rates are ascertained and the definition of a thromboembolic event that is used. Some studies only count stroke events whilst others include transient ischaemic events, pulmonary emboli and peripheral embolisation[582]. Multiple, large studies of patients who have not been anticoagulated have given rates of thromboembolism as anywhere between 2.1 and 5%[583] [584] [585]. Two major risk (of embolization) prediction models have been developed in clinical practice, CHA2DS2-VASc and CHADS2. They are used to assess the likelihood of a thromboembolic event and therefore whether or not a patient should be started on anti coagulation. Whilst they are not perfect, they are also useful tools to use in assessing the risk of a person suffering a thromboembolic event during the validity period of a certificate and along side a detailed specialist report can give valuable information. A more detailed discussion as to the advantages and disadvantages of each system is beyond the scope of this guidance but further information is available in the literature referenced within this section.

The other aspect to treatment of AF is symptom control through the control of rate or rhythm. Unfortunately the return to and maintenance of sinus rhythm (SR) does not reduce the frequency of clinical thromboembolic events. The two largest trials, AFFIRM[586] and RACE[587] demonstrated that these events occurred with equal frequency whether a rate control or

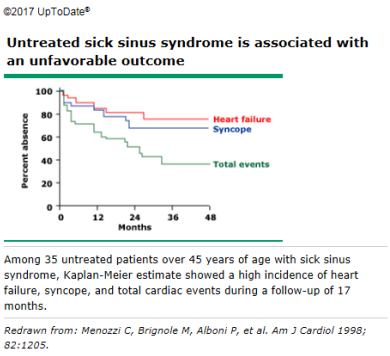
[582] Friberg L, Skeppholm M, Terént A. Benefit of anticoagulation unlikely in patients with atrial fibrillation and a CHA2DS2-VASc score of 1. J Am Coll Cardiol. 2015;65(3):225.
[583] Gage BF, Waterman AD, Shannon W, Boechler M, Rich MW, Radford MJ. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. JAMA. 2001;285(22):2864.
[584] Friberg L, Rosenqvist M, Lip GY. Net clinical benefit of warfarin in patients with atrial fibrillation: a report from the Swedish atrial fibrillation cohort study. Circulation. 2012 May;125(19):2298-307. Epub 2012 Apr 18.
[585] Friberg L, Skeppholm M, Terént A. Benefit of anticoagulation unlikely in patients with atrial fibrillation and a CHA2DS2-VASc score of 1. J Am Coll Cardiol. 2015;65(3):225.
[586] Wyse DG, Waldo AL, DiMarco JP, Domanski MJ, Rosenberg Y, Schron EB, Kellen JC, Greene HL, Mickel MC, Dalquist JE, Corley SD, Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) Investigators. A comparison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med. 2002;347(23):1825.
[587] Van Gelder IC, Hagens VE, Bosker HA, Kingma JH, Kamp O, Kingma T, Said SA, Darmanata JI, Timmermans AJ, Tijssen JG, Crijns HJ, Rate Control versus Electrical Cardioversion for Persistent Atrial Fibrillation Study Group. A comparison of rate control and rhythm control in patients with recurrent persistent atrial fibrillation. N Engl J Med. 2002;347(23):1834.

rhythm control strategy was initiated. Most events occurred when anti coagulation was stopped or the INR was sub therapeutic (less than 2.0). Hence long term anticoagulation is required even in those patients in whom sinus rhythm is restored and maintained. The choice of which treatment strategy to pursue is again outside of the scope of this guidance.

17.9.4.2 SICK SINUS SYNDROME

Sick sinus syndrome is characterised by dysfunction of the sinoatrial (SA) node and surrounding atrial myocardium. Patients usually complain of fatigue, light headedness, palpitations and presyncope or syncope. The syndrome is more common in those aged 70 years and older[588]and occurs when the sinus node tissue is replaced by fibrous tissue. Alternatively this can be due to a number of medications and toxins including beta blockers[589], calcium channel blockers[590], digoxin[591] and other antiarrythmic medications or other causes eg infiltrative diseases, inflammatory diseases or trauma.

Many patients with sick sinus syndrome will have periods of normal sinus node function[592], however once present sinus node dysfunction will progress im most patients and gives a greater likelihood of tachyarrythmias. It is very difficult to predict the time span of disease progression and specialist advice with regard to treatment and prognosis is required. These patients, particularly those with a history of alternating tachycardia and bradycardia also have an increased risk of thromboembolic events. In one small prospectively-followed cohort of 35 patients[593] aged ≥45 years with symptomatic sick sinus syndrome manifested by a mean sinus rate at rest ≤50 beats/minute and/or intermittent SA block, who did not undergo immediate treatment but were followed for an average of 17 months, a cardiovascular event requiring treatment occurred in 57% of patients and included syncope (23%), overt heart failure (17%), chronic atrial fibrillation (11%), or poorly tolerated atrial arrhythmias (6%). Treatment of sick sinus syndrome is most usually with a pacemaker after any reversible cause has been resolved.



Among 35 untreated patients over 45 years of age with sick sinus syndrome, Kaplan-Meier estimate showed a high incidence of heart failure, syncope, and total cardiac events during a follow-up of 17 months.

Redrawn from: Menozzi C, Brignole M, Alboni P, et al. Am J Cardiol 1998; 82:1205.

[588] Andersen HR, Thuesen L, Bagger JP, Vesterlund T, Thomsen PE. Prospective randomised trial of atrial versus ventricular pacing in sick-sinus syndrome. Lancet. 1994;344(8936):1523.
[589] Strauss HC, Gilbert M, Svenson RH, Miller HC, Wallace AG. Electrophysiologic effects of propranolol on sinus node function in patients with sinus node dysfunction. Circulation. 1976;54(3):452.
[590] Breithardt G, Seipel L, Wiebringhaus E, Loogen F. Effects of verapamil on sinus node function in man. Eur J Cardiol. 1978;8(3):379.
[591] Margolis JR, Strauss HC, Miller HC, Gilbert M, Wallace AG. Digitalis and the sick sinus syndrome. Clinical and electrophysiologic documentation of severe toxic effect on sinus node function. Circulation. 1975;52(1):162.
[592] Lien WP, Lee YS, Chang FZ, Lee SY, Chen CM, Tsai HC. The sick sinus syndrome: natural history of dysfunction of the sinoatrial node. Chest. 1977;72(5):628
[593] Menozzi C, Brignole M, Alboni P, Boni L, Paparella N, Gaggioli G, Lolli G. The natural course of untreated sick sinus syndrome and identification of the variables predictive of unfavorable outcome. Am J Cardiol. 1998;82(10):1205

17.9.4.3 ATRIOVENTRICULAR BLOCK

Atrioventricular block is a cardiac electrical disorder defined as impaired (delayed/absent) conduction from the atria to the ventricles.

It is described in degrees

- First degree: delayed conduction from atria to ventricles (PR interval >200 milliseconds) without interruption in atrial to ventricular conduction
- Second degree: intermittent atrial conduction to the ventricle, often in a regular pattern eg 2:1 or 3:1 or higher degrees of block further classified into Mobitz type I or II.
- Third degree: complete AV block with no atrial impulses conducted to the ventricles.

Major causes of atrioventricular (AV) block

Physiologic and pathophysiologic
Increased vagal tone
Fibrosis and sclerosis of the conduction system
Ischemic heart disease
Cardiomyopathy and myocarditis
Congenital heart disease
Familial AV block
Progressive cardiac conduction system disease
Other
Hyperkalemia, infiltrative malignancies, neonatal lupus syndrome, severe hypo- or hyperthyroidism, trauma, degenerative neuromuscular diseases
Iatrogenic
Drugs
Beta blockers, calcium channel blockers, digoxin, adenosine, antiarrhythmic drugs
Cardiac surgery
Transcatheter aortic valve implantation
Catheter ablation of arrhythmias
Transcatheter closure of VSD
Alcohol septal ablation for HCM

VSD: ventricular septal defect; HCM: hypertrophic cardiomyopathy

There are a wide range of a causes of AV block and it is difficult to estimate it's incidence or prevalence. One study examined first degree AV block in 21213 patients and found it to be more prevalent in African-Americans and in patients over 50 years in all races[594]. Another study examined the 24 hour monitors of 625 asymptomatic patients aged 15 to 83 years. Type I second degree AV block was found in 2,2% of patients, more frequently with a resting heart rate less than 60bpm[595].

Symptoms associated with AV block are fatigue, dyspnoea, chest pain, palpitations and nausea and vomiting… Syncope and pre syncope are less commonly associated symptoms and are most commonly encountered in the emergency setting.

Management of patients with AV block is largely based on the relief of symptoms and, for more advanced block, the prevention of syncope and sudden cardiac death. For first degree and type I second degree block no specific treatment is required unless symptoms are present. Occasionally these patients develop symptoms and management should include cessation of all AV nodal blocking medication. Patients may need to be considered for permanent pacemaker if their symptoms are severe enough. The prognosis related to first degree heart block remains

[594] Upshaw CB Jr. Comparison of the prevalence of first-degree atrioventricular block in African-American and in Caucasian patients: an electrocardiographic study III. J Natl Med Assoc. 2004;96:756-760.
[595] DePaula RS, Antelmi I, Vincenzi MA, et al. Cardiac arrhythmias and atrioventricular block in a cohort of asymptomatic individuals without heart disease. Cardiology. 2007;108:111-116.

uncertain. A 2016 meta analysis[596] showed that patients with first degree heart block have a hightre risk of mortality, heart failure or left ventricular function and atrial fibrillation. However first degree block was not associated with a higher risk of cardiovascular mortality, coronary heart disease, myocardial infarction or stroke.

In patients with type II second or third degree block again all medications blocking the AV node should be stopped and specific management of any underlying condition must be otimised. A pacemaker +/- ICD may well be required if symptoms persist/worsen. The type of pacemaker used with or without an implantable cardiac defibrillator is beyond the scope of these guidelines. Once treated these patients have an excellent prognosis with a low rate of complications related to the pacemaker (see below).

17.9.4.4 VENTRICULAR TACHYCARDIA

NON SUSTAINED VENTRICULAR TACHYCARDIA (MSVT)

NSVT is a common but little understood arrhythmia which is usually asymptomatic and often diagnosed on ambulatory or exercise testing performed for other reasons. Definitions vary but the most commonly used is

- Three or more consecutive ventricular beats
- Rate of >120 bpm
- Duration of less than 30 seconds

It may occur in the absence of any heart disease but is more commonly associated with ischaemic and non ischaemic heart disease, known genetic disorders eg long QT syndrome or Brugada's syndrome, infectious diseases eg Chagas' disease in Central America, congenital heart disease, metabolic problems including drug toxicity or electrolyte imbalance[597].

The estimated prevalence of NSVT in the general population is as high as 4% although this is probably an under estimate. Prevalence increases with increasing age although there do not appear to be any sex specific differences[598]. Prevalence increases in patients post MI (5-9%) and especially with an ejection fraction less than 35% (12% compared to 6% in patients with an EF >

[596] Kwok CS, Rashid M, Beynon R, Barker D, Patwala A, Morley-Davies A, Satchithananda D, Nolan J, Myint PK, Buchan I, Loke YK, Mamas MA. Prolonged PR interval, first-degree heart block and adverse cardiovascular outcomes: a systematic review and meta-analysis. Heart. 2016;102(9):672.
[597] Nathani P, Shetty S, Lokhandwala Y. Ventricular tachycardia in structurally normal hearts: recognition and management. J Assoc Physicians India. 2007;55(suppl):33-38.
[598] Kostis JB, McCrone K, Moreya AE, et al. Premature ventricular complexes in the absence of identifiable heart disease. Circulation. 1981;63:1351-1356.

35%). NSVT has been observed in 25% of patients with hypertrophic obstructive cardiomyopathy and up to 80% of patients with idiopathic dilated cardiomyopathy[599] [600].

Patients with NSVT and no identified symptoms do not require any specific therapy of the NSVT. However any underlying cardiac comorbidity should be optimally treated as appropriate for that condition. Some patients may develop symptoms such as palpitations, chest pain, shortness of breath and syncope or presyncope. These patients should be treated with medication eg beta blockers although other anti arrythmics may be preferred in different clinical settings[601]. Implantable cardiac defibrillators are not usually indicated for the treatment of NSVT as ut us self limiting and self terminating. However those who are found to have a cardiomyopathy may be a candidate for ICD placement for prevention of sudden cardiac death due to sustained ventricular tachyarrythmias.

SUSTAINED VENTRICULAR TACHYCARDIA (VT)

VT is a ventricular rhythm faster than 100 bpm lasting at least 30 seconds or requiring termination earlier due to heamodynamic instability where the beats have a uniform and stable QRS morphology. It may be idiopathic but most often occurs in people with underlying heart disease of various types including:

- Coronary artery disease – responsible for up to 70% of cases in the US
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Infiltrative cardiomyopathy
- Chagas heart disease
- Complex congenital heart disease
- Cardiac sarcoidosis
- Arryhthmogenic right ventricular cardiomyopathy
- Left ventricular noncompaction

The clinical presentation can be hugely variable ranging from sudden cardiac arrest to mild symptoms including shortness of breath, chest pain, palpitations, syncope and general malaise. Population studies have estimated the incidence of fatal ventricular arrhythmias in the general population to be 54 per 100 000 people[602] although this increases with age, the presence of risk factors for CAD and the presence of structural heart disease.

[599] Maggioni AP, Zuanetti G, Franzosi MG, et al. Prevalence and prognostic significance of ventricular arrhythmias after acute myocardial infarction in the fibrinolytic era. Circulation. 1993;87:312-322.
[600] Bigger JT Jr, Fleiss JL, Kleiger R, et al. The relationships among ventricular arrhythmias, left ventricular dysfunction, and mortality in the 2 years after myocardial infarction. Circulation. 1984;69:250-258.
[601] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, Dorian P, Huikuri H, Kim YH et al. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Europace. 2014 Sep;16(9):1257-83.
[602] Stecker E, Vickers C, Waltz J, et al. Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction: two-year findings from the Oregon Sudden Unexpected Death Study. J Am Coll Cardiol. 2006;47:1161-1166.

Idiopathic VYT generally carries a favourable prognosis with appropriate treatment[603] . In contrast, patients who develop sustained VT in the context of left ventricular dysfunction often have re-entrant rhythms which can degenerate to VF and are associated with a high mortality rate. Prophylactic use of an implantable cardioverter defibrillator has become the most important treatment to reduce mortality among high risk patients[604].

All persons with a history of a cardiac arrythmia with or without symptoms should be evaluated by a specialist and a thorough risk assessment should be conducted by the seafarer's doctor. Factors to consider include but are not limited to the type of rhythm disturbance, symptoms experienced, the need for treatment and monitoring, likely progression over the validity period and the possibility of any complications arising that may require medical care acutely.

17.9.4.5 USE OF CARDIAC IMPLANTABLE DEVICES: PACEMAKERS AND IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS (ICD)

There are a variety of potential complications associated with the use of cardiac implantable devices. Major complications in first time ICD patients requiring reoperation or hospitalization were analysed in a cohort of over 114000 patients aged 65 years and a medial follow up of 2,7 years. The rate of ICD complications was found to be 6,1 per 100 patient years ie a rate of 0,061 per year[605].

Lead malfunctions are more common in ICD leads with significant variability in the rates of malfunction in certain leads. Reported lead failure rates range from 1-9% at 2 years, 2-15% at 5 years and 5-40% at 8-10 years[606]. Pulse generator malfunctions are rare but significant and in a 2006 meta analysis were 1,3 per 1000 patient years for permanent pacemakers and 26,5 per 1000 patient years for ICDs although the rates fell significantly over time[607]. The true incidence of cardiac device infection is difficult to assess because of a lack of mandatory reporting but it is estimated at 0,8-5,7% with most occurring in the first 12 months[608].

Other complications include:

- Tricuspid regurgitation (TR) – TR can result from the placement of leads causing damage to the tricuspid valve or impeding the appropriate closure of the valve during systole. The frequencdy of developing

[603] Yarlagadda RK, Iwai S, Stein KM, et al. Reversal of cardiomyopathy in patients with repetitive monomorphic ventricular ectopy originating from the right ventricular outflow tract. Circulation. 2005;112:1092-1097.
[604] Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Circulation. 2006;114:e385-e484.
[605] Ranasinghe I, Parzynski CS, Freeman JV, Dreyer RP, Ross JS, Akar JG, Krumholz HM, Curtis JP. Long-Term Risk for Device-Related Complications and Reoperations After Implantable Cardioverter-Defibrillator Implantation: An Observational Cohort Study. Ann Intern Med. 2016 May
[606] Eckstein J, Koller MT, Zabel M, Kalusche D, Schaer BA, Osswald S, Sticherling C. Necessity for surgical revision of defibrillator leads implanted long-term: causes and management. Circulation. 2008;117(21):2727.
[607] Maisel WH. Pacemaker and ICD generator reliability: meta-analysis of device registries. JAMA. 2006;295(16):1929.
[608] Eggimann P, Waldvogel F. Pacemaker and defibrillator infections. In: Infections Associated with Indwelling Medical Devices, Waldvogel FA, Bisno AL (Eds), American Society for Microbiology Press, Washington, DC 2000. p.247.

significant TR is estimated at 10-20% of persons with a device with transvenous leads, ultimately leading to heart failure symptoms in 50% of those with severe TR[609].

- Increased defibrillation threshold – the safety threshold values for pacing and defibrillation may change over time due to lead displacement, inflammation at the tip, exit block, lead failure, progressive left ventricular disease and the effects of certain drugs. The need for regular threshold testing is an area of debate[610] and advice on the need for checks should be taken from the specialist unit overseeing care.

- Inappropriate shocks – these occur in up to 25% of patients[611], most commonly due to supraventricular tachycardias including a sinus tachycardia, electrical noise, inappropriate sensing and device failure, usually lead fracture. Patients who experience such shocks may become nervous and uncomfortable and there is some evidence that they may have an increase in mortality[612]

NEED FOR FOLLOW UP

Follow up can take place in person or remotely with the approach taken tailored to the individual patient and according to local protocol. It is usually recommended that the patient is seen in person initially and at least every 12 months[613] and a specialist report should include the planned follow up required for each patient, including any events which, should they occur would necessitate more urgent specialist review.

INTERACTIONS WITH ELECTROMAGNETIC FIELDS

There has always been concern regarding the potential for lectromagnetic interference with cardiac implantable devices but the risk is quite low[614] [615].

Electromagnetic interference can occur in a hospital setting or non hospital environment. There have been occasional reports of cardiac implantable devices being impacted by sources such as slot machines and laptop computers and are disclaimers relating to keyless entry systems and hybrid engines although no evidence exists. However there are some sources in the non hospital environment that are concerning[616]. Whilst hospital sources are outside of the scope of this document a few potential sources of interest include:

[609] Lin G, Nishimura RA, Connolly HM, Dearani JA, Sundt TM 3rd, Hayes DL. Severe symptomatic tricuspid valve regurgitation due to permanent pacemaker or implantable cardioverter-defibrillator leads. J Am Coll Cardiol. 2005;45(10):1672.
[610] Wilkoff BL, Fauchier L, Stiles MK, Morillo CA, Al-Khatib SM, Almendral J, Aguinaga L et al. 2015 HRS/EHRA/APHRS/SOLAECE expert consensus statement on optimal implantable cardioverter-defibrillator programming and testing. Heart Rhythm. 2016 Feb;13(2):e50-86. Epub 2015 Dec 1.
[611] Dichtl W, Wolber T, Paoli U, Brüllmann S, Stühlinger M, Berger T, Spuller K, Strasak A, Pachinger O, Haegeli LM, Duru F, Hintringer F. Appropriate therapy but not inappropriate shocks predict survival in implantable cardioverter defibrillator patients. Clin Cardiol. 2011;34(7):433
[612] Powell BD, Saxon LA, Boehmer JP, Day JD, Gilliam FR 3rd, Heidenreich PA, Jones PW, Rousseau MJ, Hayes DL. Survival after shock therapy in implantable cardioverter-defibrillator and cardiac resynchronization therapy-defibrillator recipients according to rhythm shocked. The ALTITUDE survival by rhythm study. J Am Coll Cardiol. 2013;62(18):1674.
[613] Slotwiner D, Varma N, Akar JG, Annas G, Beardsall M, Fogel RI, Galizio NO, Glotzer TV, Leahy RA, Love CJ, McLean RC, Mittal S. HRS Expert Consensus Statement on remote interrogation and monitoring for cardiovascular implantable electronic devices. Heart Rhythm. 2015;12(7):e69.
[614] Pinski SL, Trohman RG. Interference in implanted cardiac devices, Part I. Pacing Clin Electrophysiol. 2002;25(9):1367.
[615] Kolb C, Zrenner B, Schmitt C. Incidence of electromagnetic interference in implantable cardioverter defibrillators. Pacing Clin Electrophysiol. 2001;24(4 Pt 1):465.
[616] Misiri J, Kusumoto F, Goldschlager N. Electromagnetic interference and implanted cardiac devices: the nonmedical environment (part I). Clin Cardiol. 2012 May;35(5):276-80. Epub 2012 Apr 26.

1. Household appliances.

Although there are no specific studies it is commonly accepted that contemporary pacemakers and ICDs are shielded from the microwave energy produced by modern appliances[617] and no special precautions are recommended when using such tgings as televisions, radios, toasters, microwave ovens and electric blankets. However there is some evidence that a device may be affected in very narrow circumstances eg induction cooker tops if the pot was not placed centrally on the coil and the patient stood as close as possible to the cooker[618].

2. Cellular telephones.

Cellular telephones are unlikely to cause clinically significant interference with cardiac devices however the advice offered is that patients should not carry or place a cellular telephone within 15cm of the device[619].

3. Portable media players

Small studies have found that portable media players are unlikely to interfere with the intrinsic function of pacemakers or ICDs they are capable of causing programmer interference when

Documented sources of electromagnetic interference (EMI) in patients with implanted cardiac devices

Source	Examples
Electromagnetic fields	
Daily life*	Faulty home appliances
	Metal detectors
	Anti-theft equipment
	Slot machines
Work and industrial environment	High voltage power lines¶
	Welding equipmentΔ
	Electronic motors while "on"
	Induction furnaces
	Degaussing coils
Medical/hospital environment	Magnetic resonance imaging
	Defibrillation or cardioversion
	Device-device interaction (eg, pacemaker and neural stimulator)
	Radiofrequency ablation
	Electrocautery
	Transcutaneous nerve stimulation
	Therapeutic diathermy
	Lithotripsy
	Radiation therapy◇

* There are many potential sources of single-beat inhibition. However, single-beat inhibition is not clinically significant and does not merit specific mention.
¶ If working at or near the level of the power line. There is no convincing evidence that being under the power lines at ground level will cause interference.
Δ Although all welding equipment is capable of causing interference, it most commonly occurs with equipment that operates at ≥150 amps.
◇ Radiation therapy may cause electromagnetic interference but may also result in direct damage to the pulse generator resulting in sudden no output or "runaway".

[617] Goldschlager N, Epstein A, Friedman P, Gang E, Krol R, Olshansky B, North American Society of Pacing and Electrophysiology (NASPE) Practice Guideline Committee. Environmental and drug effects on patients with pacemakers and implantable cardioverter/defibrillators: a practical guide to patient treatment. Arch Intern Med. 2001;161(5):649.
[618] Irnich W, Bernstein AD. Do induction cooktops interfere with cardiac pacemakers? Europace. 2006;8(5):377.
[619] Irnich W, Batz L, Müller R, Tobisch R. Electromagnetic interference of pacemakers by mobile phones. Pacing Clin Electrophysiol. 1996;19(10):1431.

placed within 2 inches of the device, but not if 6 inches from the device[620]. Hence the advice is that all portable media players are kept at least 6 inches or 15 cm from the device and that portable headphones are kept at least 3 cm from the device[621]. CB radios or large speakers may potentially cause interference and patients should be made aware of this possibility.

4. Security systems

Systems such as antishoplifiting gates and metal detectors are in wide spread use. Although device interference is possible and has been reported in case studies[622] there is no evidence that any clinically significant intereference woulkd occur with the transient exposure of walking through such a field. The recommendation is 'don't linger, don't lean' [623].

5. External electrical activity

Potential causes of concern in the workplace and relevant to persons are welders, industrial welding machines, electric motors and degaussing coils. The functional evaluation of pacemakers and ICDs in the workplace has rarely demonstrated any interference however given the concern it is recommended that sources of electromagnetic field in the work place are assessed before the person returns to work. This is particularly the case in persons who are pacemaker dependent or have an ICD[624]. This recommendation may be restrictive to many persons and may mean that they need to be considered permanently unfit for work at sea. As with all cases a specialist report, probably including input from a cardiac physiologist is essential as part of the individual risk assessment that must be carried out by the seafarer's doctor. Such an assessment should also include the underlying clinical condition and any other comorbidities.

Reviewed January 2017

17.9.5 ISCHAEMIC CEREBROVASCULAR DISEASE (STROKE OR TRANSIENT ISCHAEMIC ATTACK)

I 61-69 G 46	Ischaemic cerebrovascular disease (stroke or transient ischaemic attack). Increased likelihood of recurrence, sudden loss of capability, mobility limitation. Liable to develop other circulatory	T – Until treated and any residual impairment stabilised and for three months after event P – If residual symptoms interfere with duties or there is significant likelihood of recurrence.	R, L – Case-by-case assessment of fitness for duties; exclude from lone watchkeeping. Assessment should include likelihood of future cardiac events. General standards of physical fitness should be met (C – Physical	Not applicable

[620] Webster G, Jordao L, Martuscello M, Mahajan T, Alexander ME, Cecchin F, Triedman JK, Walsh EP, Berul CI. Digital music players cause interference with interrogation telemetry for pacemakers and implantable cardioverter-defibrillators without affecting device function. Heart Rhythm. 2008;5(4):545.
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[622] Gimbel JR, Cox JW Jr. Electronic article surveillance systems and interactions with implantable cardiac devices: risk of adverse interactions in public and commercial spaces. Mayo Clin Proc. 2007;82(3):318.
[623] Pinski SL, Trohman RG. Interference in implanted cardiac devices, Part I. Pacing Clin Electrophysiol. 2002;25(9):1367.
[624] Fetter JG, Benditt DG, Stanton MS. Electromagnetic interference from welding and motors on implantable cardioverter-defibrillators as tested in the electrically hostile work site. J Am Coll Cardiol. 1996;28(2):423.

	disease causing sudden loss of capability		capability requirements). Annual assessment.	

Cerebrovascular disease is the third leading cause of death in developed countries after heart disease and cancer and it's overall prevalence is estimated at 794 per 100 000 with a huge economic impact due to loss of these people from the workplace and the extended recovery time they require.

17.9.5.1 TRANSIENT ISCHAEMIC ATTACK (TIA)

A TIA is defined as a transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischaemia, without acute infarction[625]. This has replaced the former definition of focal neurological impairment lasting less than 24 hours. The incidence and prevalence of TIA are difficult to determine, largely due to the varying criteria used to identify a TIA. Each year in England 2000 people have a first TIA and the age adjusted annual incidence rate for TIA in the UK is estimated at 190 cases per 100 000 population[626] [627]. The population wide prevalence in the US is approximately 2.3% but this varies according to age with a 2% prevalence in patients aged 55 to 64 years, 3.5% in patients aged 65 – 74 years, 4.3% in patients aged 75 – 84 years and rising to over 5% for people aged over 85 years[628]. TIAs are more common in males and non Hispanic black people within the US but is believed that up to 50% of TIAs go unrecognised and never come to medical attention[629].

Causes of TIA include:

- In situ thrombosis of an intracranial artery or artery to artery embolization of thrombus – 16%
- Cardioembolic events – 29% - these are secondary to intracardiac thrombus formation eg related to atrial fibriallation or an impaired ejection fraction or rarely due to embolization from a venous thrombus across a cardiac shunt
- Small vessel occlusion – 16% - hypertension and diabetes predispose to small ischaemic lesions. These may occur in the brain stem or internal capsule where a small lesion can result in significant disability.
- Occlusion due to hypercoagulability, vasculitis, vasospasm or sickle cell disease – 3%
- Uncertain – 36%

All patients who have suffered a possible TIA require rapid investigation and intitiation of secondary prevention therapy. New guidelines suggest that this should occur within the first

[625] Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack. Stroke. 2009;40:2276-2293.
[626] Gibbs RGJ, Newson R, Lawrenson R, et al. Diagnosis and initial management of stroke and transient ischemic attack across UK health regions from 1992 to 1996. Experience from primary care database. Stroke. 2001;32:1085-1090.
[627] van Rees JB, van Welsenes GH, Borleffs CJ, Thijssen J, van der Velde ET, van der Wall EE, van Erven L, Schalij MJ. Update on small-diameter implantable cardioverter-defibrillator leads performance. Pacing Clin Electrophysiol. 2012;35(6):652.
[628] Bots ML, Van der Wilk EC, Koudstaal PJ, et al. Transient neurological attacks in the general population: prevalence, risk factors, and clinical relevance. Stroke. 1997;28:768-773.
[629] Go AS, Mozaffarian D, Roger VL, et al. Heart disease and stroke statistics - 2014 update: a report from the American Heart Association. Circulation. 2014;129:e28-e292.

one to two days after a TIA[630]. The urgency in assessment and treatment is largely due to the fact that a TIA is a well recognised risk to precede a disabling stroke in the same way that unstable angina can occur shortly before a fatal MI. The risk of a stroke in the first the months after a TIA is 10.5% but half of these occur within the first two days[631]. Detailed risk assessment eg ABCD2[632] score, used alongside clinical judgement, can predict those at highest risk of a an early stroke:

Risk level	ABCD2 score	Risk of stroke within 2 days
High	6 or 7	8.1%
Intermediate	4 or 5	4.1%
Low	0 - 3	1%

As a TIA is often indicative of underlying cardiac or atherosclerotic disease 17% of patients with a TIA will be dependent and 5% will be dead 6 months after the event, despite full resolution of symptoms[633].

17.9.5.2 STROKE

Stroke is defined as an acute neurological deficit and caused by cerebrovascular aetiology. It is divided into ischaemic stroke caused by vascular occlusion or stenosis (approximately 85% of cases) and haemorrhagic stroke caused by vascular rupture (approximately 15% of cases)[634].

Stroke is the third leading cause of death and a major cause of disability in the US, England, Wales and Canada[635] [636]. In Scotland in 2006 the incident rate, standardised by age and sex was 166 per 100 000[637] and there are approximately 700 000 new strokes per year[638]. Ischemic stroke prevalence can be further divided to[639]

[630] Johnston SC, Albers GW, Gorelick PB, et al. National Stroke Association recommendations for systems of care for transient ischemic attack. Ann Neurol. 2011;69:872-877.
[631] Johnston SC, Gress DR, Browner WS, et al. Short-term prognosis after emergency department diagnosis of TIA. JAMA. 2000;284:2901-2906.
[632] http://www.stroke.org/sites/default/files/resources/tia-abcd2-tool.pdf?docID=5981
[633] Daffertshofer M, Mielke O, Pullwitt A, et al. Transient ischemic attacks are more than "ministrokes." Stroke. 2004;35:2453-2458.
[634] Thom T, Haase N, Rosamond W, et al. Heart disease and stroke statistics - 2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2006;113:e85-e151.
[635] Wolfe C. The burden of stroke. In: Wolfe C, Rudd T, Beech R, eds. Stroke services and research. London, UK: The Stroke Association; 1996.
[636] Heart and Stroke Foundation of Canada. Stroke statistics. 2012. http://www.heartandstroke.com (last accessed 21 October 2015).
[637] NHS National Services Scotland: Information Services Division. Statistical publication notice: stroke statistics update. October 2007. http://www.isdscotland.org (last accessed 21 October 2015).
[638] Thom T, Haase N, Rosamond W, et al. Heart disease and stroke statistics - 2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2006;113:e85-e151.
[639] Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke. 1993;24:35-41.

- Extracranial atherosclerosis 10% - usually the external carotid or vertebral arteries. It is a site for othrombus formation that then embolises.
- Intracranial atherosclerosis 10% - as above
- Cardioembolic 25% - this is usually due to cardiac thrombus and associated with atrial fibrillation although accumulating evidence suggests that aortic atherosclerotic plaque may be another source.
- Lacunar infarction (small vessel disease) 15% - due to thrombotic occlustion of a small penetrating artery affected by lipid accumulation due to ageing and hypertension. This results in a infarct <1.5 cm in the territory of the affected artery.
- Indeterminate aetiology 30%
- Other defined causes 10% - - include disease of the intra or extra cranial vessels eg dissection, vasculitis or haematological system eg sickle cell anaemia, antiphospholipid syndrome

It is more common in older people, males, African American and Hispanic people[640].

A wide variety of factors influence stroke prognosis, including

- Age – advancing age has a major negative impact on stroke morbidity, mortality and long term outcome[641]
- Stroke severity – is probably the most important factor affecting short and long term outcome. As a general rule large strokes with severe initial clinical deficits have a poorer outcome than smaller strokes[642]. There are scales available to quantify neurological impairment and hence attempt to predict outcome[643].
- Stroke mechanism – aetiology influences the prognosis for recovery with patients who have suffered lacunar infarcts having a better prognosis up to one year after recovery although ghere is little difference in the long term. Equally patients with a stroke secondary to cardioembolic or large artery aetiology tend to have a worse prognosis[644].
- Infarct location – prognosis may vary according to the affected vascular territory and site of ischemic brain injury.
- Comorbid conditions – many pre stroke premorbid events have a increased risk of poor outcome including[645] atrial fibrillation, cancer, coronary artery disease, dementia, dependency, diabetes mellitus, hyperglycaemia on admission[646], heart failure, myocardial infarction[647], renal dysfunction, poor nutritional status and low haemoglobin.

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[640] Goldstein LB, Adams R, Becker K, et al. Primary prevention of ischemic stroke: a statement for healthcare professionals from the Stroke Council of the American Heart Association. Stroke. 2001;32:280-299.

[641] Knoflach M, Matosevic B, Rücker M, Furtner M, Mair A, Wille G, Zangerle A, Werner P, Ferrari J, Schmidauer C, Seyfang L, Kiechl S, Willeit J, Austrian Stroke Unit Registry Collaborators. Functional recovery after ischemic stroke--a matter of age: data from the Austrian Stroke Unit Registry. Neurology. 2012 Jan;78(4):279-85. Epub 2012 Jan 11.

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[645] Saposnik G, Kapral MK, Liu Y, Hall R, O'Donnell M, Raptis S, Tu JV, Mamdani M, Austin PC, Investigators of the Registry of the Canadian Stroke Network, Stroke Outcomes Research Canada (SORCan) Working Group. IScore: a risk score to predict death early after hospitalization for an acute ischemic stroke. Circulation. 2011 Feb;123(7):739-49. Epub 2011 Feb 7.

[646] Desilles JP, Meseguer E, Labreuche J, Lapergue B, Sirimarco G, Gonzalez-Valcarcel J, Lavallée P, Cabrejo L, Guidoux C, Klein I, Amarenco P, Mazighi M. Diabetes mellitus, admission glucose, and outcomes after stroke thrombolysis: a registry and systematic review. Stroke. 2013 Jul;44(7):1915-23. Epub 2013 May 23.

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- Epidemiological factors – there is conflicting evidence with regards to the effect of sex differences on prognosis but it has been demonstrated that there are racial differences with black or non white people in the US having a higher risk for poor outcome[648]

Interventions such as thrombolysis, stroke unit care and rehabilitation can also play a major role in determining outcome.

The greatest proportion of recovery from stroke occurs in the first 3 to 6 months after the acute event although some patients experience further recovery up to 18 months[649]. In a Danish study those who had mild disability tended to recover within 2 months and those with a moderate disability recovered within 3 months[650]. Patients with severe disability who recovered did so within four months and those with the most severe disability, in 5 months. This may vary with specific neurological deficits but further discussion is outside the scope of this text.

Patients who have suffered a stroke or TIA remain at high risk of negative outcomes. In one study[651] death was the most common negative outcome (5.4% at 1 year and 26.8 % at 5 years) closely followed by further stroke (2.6% at 1 year and 7.9% at 2 years). Among those who survived the first year the event rate remained high at 5% per year both at 3 and 5 years. In figures published by the National Stroke Association[652] it is stated that at least 25 – 35% of Americans who have a stroke every year will have another stroke within their lifetime and that within 5 years of a first stroke the risk for another stroke can increase more than 40%. Within 5 years of a stroke 24% of women and 42% of men with experience a recurrent stroke. Equally recurrent strokes are associated with higher morbidity and disability.

All persons who have suffered a stroke or TIA must be fully assessed within a specialist unit and a specialist report outlining but not limited to the cause of the event, underlying comorbidities and their treatment, necessary secondary prevention measures, the risk of future thromboembolic or cardiac events and any follow up required must be obtained. This should form part of an individualised risk assessment which considers cerebrovascular events and other comorbidities, treatment and physical capability in the setting of the persons role and location and bearing in mind their ability to perform their routine and emergency duties.

[648] Centers for Disease Control and Prevention (CDC). Differences in disability among black and white stroke survivors--United States, 2000-2001.. MMWR Morb Mortal Wkly Rep. 2005 Jan;54(1):3-6.
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[651] Richard H Swartz, Jiming Fang, Moira K Kapral. High 1- to 5-Year Mortality and Morbidity in Stable Patients Without Early Complications After Stroke or TIA. Stroke. 2014;45:ATMP93
[652] http://www.stroke.org/we-can-help/survivors/stroke-recovery/first-steps-recovery/preventing-another-stroke

17.9.5.3 CAROTID STENOSIS

The presence of atherosclerotic disease at the carotid artery bifurcation may be asymptomatic or symptomatic with focal neurological symptoms with the carotid artery distribution. The prevalence of asymptomatic disease is low in the general population and varies with age[653]. The most feared outcome is ischaemic stroke and the risk with stenosis > 50% in asymptomatic individuals is approximately 0.5-1% per year[654] and the huge majority are preceded by TIA[655]. Because of the low prevalence of the disease and the low incidence of complication screening by either auscultation or non invasive ultrasound or MRI is not recommended[656]. Asymptomatic carotid atherosclerosis is also a marker of increased risk for myocardial infarction and vascular death.

Symptomatic carotid disease is defined as focal neurological symptoms eg amaurosis fugax, contralateral weakness or numbness of an extremity or the face, dysarthria or aphasia in the distribution of a carotid artery with a significant stenosis. Symptomatic disease can be treated with medical management or with carotid endarterectomy and the decision for treatment must be made in a specialist centre based on individual risk assessment including but not limited to the percentage of stenosis, the accessibility of the lesion, presence or not of comorbidities that would greatly increase the risk of surgery age and sex[657] [658].

Any person with documented carotid stenois, with or without symptoms should be assessed in a specialist centre and all treatment options considered. Asymptomatic carotid atherosclerosis is also a marker of increased risk for myocardial infarction and vascular death. Thus, asymptomatic carotid atherosclerosis is considered a risk equivalent for coronary heart disease – patients with any form of non coronary atherosclerotic disease have a 10 year risk of developing coronary heart disease that exceeds 20% ie 2% per year. A full report including but not limited to the degree of stenosis, risk of complication over the validity period of the certificate, other comorbidities, treatment and need for follow up should be obtained and form part of an individual risk assessment.

Reviewed 2016

[653] de Weerd M, Greving JP, Hedblad B, Lorenz MW, Mathiesen EB, O'Leary DH, Rosvall M, Sitzer M, Buskens E, Bots ML. Prevalence of asymptomatic carotid artery stenosis in the general population: an individual participant data meta-analysis. Stroke. 2010;41(6):1294.
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[655] Dodick DW, Meissner I, Meyer FB, Cloft HJ. Evaluation and management of asymptomatic carotid artery stenosis. Mayo Clin Proc. 2004;79(7):937.
[656] https://www.uptodate.com/contents/screening-for-asymptomatic-carotid-artery-stenosis?source=related_link
[657] North American Symptomatic Carotid Endarterectomy Trial. Methods, patient characteristics, and progress. Stroke. 1991;22(6):711.
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17.9.6 ARTERIAL CLAUDICATION

I 73	Arterial claudication. Likelihood of other circulatory disesase casuing sudden loss of capability. Limits to exercise capacity.	T – Until assessed. P – If incapable of performing duties	R, L – Consider restriction to non-watchkeeping duties in coastal waters provided symptoms are minor and do not impair essential duties or if they are resolved by surgery or other treatment and general standard of fitness can be met (Appendix C). Assess likelihood of future cardiac events (follow criteria in I20-25). Review at least annually.	Not applicable

Atherosclerosis of the noncardiac vessels is defined as peripheral arterial disease (PAD). Although other disease processes can lead to narrowing of the arteries (eg, inflammation, thrombosis) and symptoms of arterial insufficiency, PAD is by far the most prevalent etiology[659]. The lower extremity vessels are affected more commonly than the upper extremity vessels.

Whilst PAD is the commonest cause of claudication[660] there are other, rarer causes and these include aortic coarctation, arterial fibrodysplasia, arterial tumour, arterial dissection, arterial embolism, thrombosis, vasospasm, and trauma. Here we will be focusing on atherosclerosis and peripheral arterial disease.

The worldwide prevalence of PAD is estimated at 3 – 12% [661]and it was estimated that 202 million people around the world were living with PAD in 2010. The majority of individuals with PAD (70 %) live in low/middle income regions of the world, including 55 million individuals in southeast Asia and 46 million in the western pacific region[662]. PAD is more prevalent in older individuals (from 40 years), certain ethnic populations, families with atherosclerosis and in those with risk factors for cardiovascular disease. Risk factors that favor the development of peripheral artery disease (PAD) are similar to those that promote the development of coronary atherosclerosis and include smoking, hypertension, diabetes, hyperlipidemia, homocysteinemia

[659] AndersonJL, Halperin JL; Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: Circulation 2013; 127: 1425-1433.
[660] AndersonJL, Halperin JL; Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: Circulation 2013; 127: 1425-1433.
[661] Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Creager MAet al; ACA/AHA 2005 guidelines for the management of patients with peripheral aterial disease.
[662] Fowkes FG, Rudan D, Rudan I, Aboyans V, Denenberg JO, McDermott MM, Norman PE, Sampson UK, Williams LJ, Mensah GA, Criqui MH; Comparison of global estimates of prevalence and risk factors for peripheral artery disease in 2000 and 2010: a systematic review and analysis. Lancet. 2013;382(9901):1329

and metabolic syndrome[663]. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines on PAD identified the following groups at risk for lower extremity PAD[664]:

- Age ≥70 years
- Age 50 to 69 years with a history of smoking or diabetes
- Age 40 to 49 with diabetes and at least one other risk factor for atherosclerosis
- Leg symptoms suggestive of claudication with exertion or ischemic pain at rest
- Abnormal lower extremity pulse examination
- Known atherosclerosis at other sites (eg, coronary, carotid, renal artery disease)

The clinical manifestations of PAD depend upon the location and severity of arterial stenosis or occlusion and range from no symptoms at all to mild extremity pain with activity (eg claudication) to limb-threatening ischemia. Most patients with asymptomatic PAD have a benign course; however, clinical manifestations can develop or progress rapidly and unpredictably in those with PAD who continue to smoke, or those with concomitant diabetes or renal insufficiency. Management of these other diseases plays a huge part in the primary prevention of PAD.

Patients with PAD have increased risk for cardiovascular ischaemic events, whether or not they have symptoms of PAD. One study has demonstrated an annual cardiovascular event rate of 5 - 7 % for patients with PAD[665]. There is a 20% to 60% increased risk for myocardial infarction and a 2- to 6-fold increased risk of death due to coronary heart disease events. [1] The risk of stroke is increased by 40%.

The risk of progression from asymptomatic PAD to ischaemic symptoms that require intervention is generally low. PAD progression, as measured by the Ankle Brachial Index is similar for asymptomatic and symptomatic patients. The decline in ABI closely relates to the initial value of ABI upon initial diagnosis - a more rapid decline is seen in patients with lower initial ABI values[666]. However the risk of developing intermittent claudication (the most common symptom of PAD) in asymptomatic patients was increased in patients with elevated serum cholesterol (odds ratio increase of 1.2 for each 40 mg/dL [1 mmol/L] elevation), cigarette smoking (odds ratio increase 1.4 for each 10 cigarettes smoked per day), moderate hypertension (odds ratio increase 1.5 for mild and 2.2 for moderate hypertension), and diabetes mellitus (odds ratio 2.6)[667]. In patients with diabetes, 28 % had progression of disease, regardless of symptoms[668].

[663] Selvin E, Erlinger TP; Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. Circulation. 2004;110(6):738.
[664] 2011 WRITING GROUP MEMBERS, 2005 WRITING COMMITTEE MEMBERS, ACCF/AHA TASK FORCE MEMBERS; 2011 ACCF/AHA Focused Update of the Guideline for the Management of patients with peripheral artery disease (Updating the 2005 Guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. Circulation. 2011;124(18):2020.
[665] Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, TASC II Working Group; Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). J Vasc Surg. 2007;45 Suppl S:S5.
[666] Nicoloff AD, Taylor LM Jr, Sexton GJ et al. Relationship between site of initial symptoms and subsequent progression of disease in a prospective study of atherosclerosis progression in patients receiving long-term treatment for symptomatic peripheral arterial disease: J Vasc Surg. 2002;35(1):38.
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[668] Leibson CL, Ransom JE, Olson W, Zimmerman BR, O'fallon WM, Palumbo PJ. Diabetes Care. 2004 Dec;27(12):2843-9: Circulation. 1997;96(1):44.

For patients with intermittent claudication again the progress of disease is often slow. The ACC/AHA guidelines on PAD (2005 updated 2011, as quoted earler) estimated the following rates of limb and cardiovascular outcomes at five years in patients with intermittent claudication:

- Stable claudication in 70- 80%
- Worsening claudication in 10- 20%
- Critical limb ischemia in 1- 2%
- Nonfatal myocardial infarction or stroke in 20%
- Death in 15- 30 % (75% due to cardiovascular causes)
- Intermittent claudication as a manifestation of PAD is itself a strong marker for generalized atherosclerosis and other cardiovascular and cerebrovascular morbidity and mortality. In studies, the 5- and 10-year mortality rates among patients with intermittent claudication were 30 – 42% and
- 50- 65%, respectively[669][670]. In addition patients with intermittent claudication have a poor quality of life and high rates of depression[671] and a relative increase in the incidence of tumours and tumour related deaths, probably due to the high prevalence of smoking[672].
- Critical limb ischaemia occurs in 1- 2% of all patients with PAD and is manifest by ischaemic rest pain or tissue loss such as skin ulceration or gangrene. Patients with critical limb ischemia are at immediate risk for limb loss. Amputation rates remain high at 25% and long-term survival is poor. Nearly 25% of patients presenting with critical limb ischemia will suffer a cardiovascular death within one year of their initial diagnosis and in one review, only 50% of patients presenting with critical limb ischemia were alive with both limbs intact at the end of one year[673]. In studies of patients with nonreconstructible disease, 40% of patients with critical limb ischemia underwent amputation within six months, and 20% died within the same time period[674].
- The treatment and follow up required for a person with PAD will depend on the level and degree of stenosis and the presence or not of symptoms. However it is probably that annual follow up will be required for all of these persons so time limitation and/or restriction of a certificate is likely to be indicated. If specific treatment is required eg medication or revascularization (endovascular or surgical) the risks and complications of this should also be taken into consideration when making a risk assessment, as should the presence of other comorbidities.

Reviewed 2015

[669] Muluk SC, Muluk VS, Kelley ME, Whittle JC, Tierney JA, Webster MW, Makaroun MS. Outcome events in patients with claudication: a 15-year study in 2777 patients: J Vasc Surg. 2001;33(2):251.
[670] Dormandy J, Heeck L, Vig S. Intermittent claudication: a condition with underrated risks: Semin Vasc Surg. 1999;12(2):96.
[671] McDermott MM, Greenland P, Guralnik JM, Liu K, Criqui MH et al: Depressive symptoms and lower extremity functioning in men and women with peripheral arterial disease. J Gen Intern Med. 2003;18(6):461.
[672] Taute BM, Thommes S, Taute R, Rapmund I, Lindner K, Podhaisky H. Long-term outcome of patients with mild intermittent claudication under secondary prevention: Vasa. 2009;38(4):346.
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[674] Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, TASC II Working Group. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II): J Vasc Surg. 2007;45 Suppl S:S5.

17.9.7 VARICOSE VEINS

I 83	Varicose veins. Possibilty of bleeding if injured, skin changes and ulceration.	T – Until treated if impairing symptoms. Post-surgery for up to one month	Not applicable	No impairing symptoms or complications

Varicose veins are one of the signs and symptoms of venous disease and are defined as subcutaneous, permanently dilated veins 3 mm or more in diameter when measured in a standing position. Other signs and symptoms of venous disease include telangiectasias and chronic venous insufficiency as manifest by oedema, skin changes and/or ulceration. Varicose veins are more prevalent in industrialised countries and in more developed regions with the prevalence of varicose veins in a Western population older than 15 years of age estimated at 10- 15% for men and 20- 25% in women[675]. Although many factors such as gender, pregnancy, occupation, weight, and race have been implicated as predisposing factors for varicose veins, only previous deep vein thrombosis and genetic factors may be causative factors. Common symptoms include aching of the lower legs and leg cramps and more uncommon symptoms/complications can include itching, ulceration, oedema, thrombophlebitis or bleeding. These tend to develop over a long time frame. Treatment is often with life style modification eg weight loss, avoiding long periods of standing or compression stockings. If these are unsuccessful, symptoms are impairing or complications occur phlebectomy, sclerotherapy or ablative procedures are recommended. Following therapy a short period of reduced activity is required and follow up will depend on the type of procedure performed. Hence a time limited certificate may be appropriate. Each case should be assessed on an individual basis taking into consideration the risks, specific tasks and other comorbidities.

Reviewed 2015

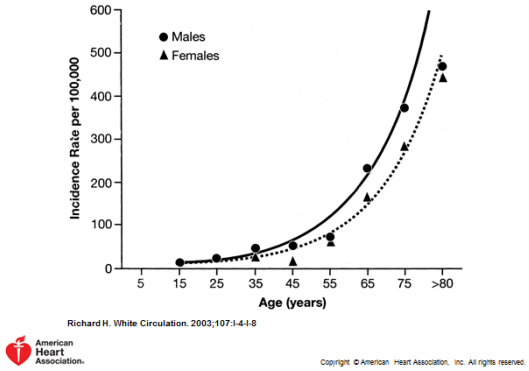
17.9.8 DEEP VEIN THROMBOSIS AND PULMONARY EMBOLI

I 80.2-3	Deep vein thrombosis/pulmonary embolus Likelihood of recurrence and of serious pulmonary embolus. Likelihood of bleeding from anticoagulant treatment.	T – Until investigated and treated and normally while on short-term anticoagulants P – Consider if recurrent events or on permanent anticagulants	R, L – May be considered fit for work with a low liability for injury; in near-coastal waters; once stabilised on anticoagulants with regular monitoring of level of anticoagulation	Full recovery with no anticoagulant use

[675] Callum MJ. Epidemiology of Varicose Veins: Br J Surg. 1994: 81: 167 - 173

Deep vein thrombosis (DVT) and Pulmonary Embolism (PE) are two manifestations of venous thromboembolism (VTE). VTE is relatively common with a yearly incidence estimated at 1 in every 1000 adults with two thirds of cases manifest as DVT and one third as PE. There is a higher incidence in winter than summer and there is a significantly higher incidence among Caucasians and African Americans than among Hispanics and Pacific Islanders. The incidence also rises sharply with age.About 25 – 50% of patient with first time VTE have an idiopathic condition with no easily identifiable risk factor[676].

Figure 1. Annual incidence of VTE among residents of Worcester MA 1986, by age and sex.



Richard H. White Circulation. 2003;107:I-4-I-8

American Heart Association.

17.9.8.1 DEEP VEIN THROMBOSIS

DVT is the development of a blood clot in a major deep vein in the leg, thigh, pelvis or abdomen which may result in impaired venous blood flow and consequent leg swelling and pain. DVT may also occur in the upper limbs or brain. There is a clear association between DVT and the following:

- Active malignancy
- Recent major surgery
- Recent hospitilisation
- Recent trauma
- Medical illness

In the absence of any of these factors the DVT is considered idiopathic. Whether a DVT is provoked by any of these factors or is idiopathic is a significant determinant of recurrence. Consensus is that all DVT should be treated with three months of oral anticoagulation but with consideration to ongoing treatment if the DVT is idiopathic or unprovoked[677] as about 50% of these patients will have a recurrence within 10 years if treatment is stopped at this point. In patients with acute DVT or PE enrolled in prospective cohort studies only 5% developed a recurrence in the first 6 months of anticoagulation however 30% developed a recurrence between 6 months and 5 years after the initial event if off anticoagulation[678]. A lower risk of recurrence is associated with female sex, absence of a major thrombophilic disorder and absence of residual thrombus on ultra sound[679].

[676] White RH. The epidemiology of venous thromboembolism. Circulation. 2003;107(23 suppl 1):I4-I8.
[677] Kearon C. Extended anticoagulation for unprovoked venous thromboembolism: a majority of patients should be treated. J Thromb Thrombolysis. 2011;31:295-300.
[678] Schulman S, Rhedin AS, Lindmarker P, et al. A comparison of six weeks with six months of oral anticoagulant therapy after a first episode of venous thromboembolism. Duration of Anticoagulation Trial Study Group. N Engl J Med. 1995;332:1661-1665.
[679] Prandoni P, Prins MH, Lensing AW, et al. Residual thrombosis on ultrasonography to guide the duration of anticoagulation in patients with deep venous thrombosis: a randomized trial. Ann Intern Med. 2009;150:577-585.

The risks and benefits of treatment with appropriate anticoagulation along with the risk of recurrence of VTE, the monitoring requirments of treatment, any underlying medical condition and access to medical care are all factors that must be included in an individual risk assessment for any person with a history of DVT. Specialist input may be invaluable.

17.9.8.2 PULMONARY EMBOLUS (PE)

Acute PE is a form of venous thromboembolism (VTE) that is common and sometimes fatal. Reports of the incidence of PE within the general population have increased with the introduction of CT angiography and overall it is estimated at 56 per 100 000 and 48 per 100 000 for males and females respectively[680]. Incidence increase with age, particularly in women so that after 75 years the incidence is >500 per 100 000[681]. It's prevalence is estimated at 1%[682] and mortality rates have been estimated at 4% at 30 days and 13% at 1 year, increasing with age[683]. Age adjusted mortality rates for African-Americans are 50% higher than those for whites and in turn the mortality rate for whites is 50% higher than those for other races[684]. Increased mortality has been reported for as long as 30 years although late mortality is mostly due to predisposing comorbidities and less commonly due to recurrent thromboembolism or chronic thromboembolic pulmonary hypertension. One database analysis of over 128 000 patients with venous thromboembolism reported a three fold increase in mortality at 30 years in patients with PE compared to age and sex matched controls who did not suffer a PE during the same period[685].

The rate of recurrence of PE is greatest in the first two weeks and declines there after. The cumulative rate of recurrence while on anticoagulant therapy amounts to 2% at two weeks and 6% at three months, 8% at six months, 13% at one year, 23% at five years and 30% at ten years[686] [687] [688]. However the rate generally decreases with therapeutic anticoagulation but is increased by the presence of specific risk factors eg unprovoked PE, malignancy.

Anticoagulation is indicated for patients with pulmonary embolus in whom the risk of bleeding is low. If the risk of bleeding outweighs the potential benefit of anticoagulation other treatment

[680] Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003;163(14):1711.
[681] Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ 3rd. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Arch Intern Med. 1998 Mar;158(6):585-93.
[682] Stein PD, Henry JW. Prevalence of acute pulmonary embolism among patients in a general hospital and at autopsy. Chest. 1995;108:978-981.
[683] Alotaibi GS, Wu C, Senthilselvan A, McMurtry MS. Secular Trends in Incidence and Mortality of Acute Venous Thromboembolism: The AB-VTE Population-Based Study. Am J Med. 2016 Aug;129(8):879.e19-25. Epub 2016 Feb 27.
[684] Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003;163(14):1711.

[685] Søgaard KK, Schmidt M, Pedersen L, Horváth-PuhóE, Sørensen HT. 30-year mortality after venous thromboembolism: a population-based cohort study. Circulation. 2014;130(10):829.
[686] Kyrle PA, Rosendaal FR, Eichinger S. Risk assessment for recurrent venous thrombosis. Lancet. 2010;376(9757):2032.
[687] Heit JA. Predicting the risk of venous thromboembolism recurrence. Am J Hematol. 2012 May;87 Suppl 1:S63-7. Epub 2012 Feb 24.
[688] Zhu T, Martinez I, Emmerich J. Venous thromboembolism: risk factors for recurrence. Arterioscler Thromb Vasc Biol. 2009 Mar;29(3):298-310.

strategies will need to be considered eg inferior vena cava filter. A detailed discussion with regards to the type of anticoagulant and the length of time treatment is continued is beyond the scope of this guidance but these questions, the risks associated with treatment, the likelihood of recurrence, access to medical care and the need for monitoring are all factors that must be taken into consideration when assessing the fitness of a person to return to work at sea.

Reviewed 2016

17.9.9 OTHER HEART DISEASE

I00-99	Other heart disease eg cardiomyopathy, pericarditis, heart failure.	T – Until investigated, treated and adequacy of treatment confirmed P – If impairing symptoms or likelihood of impairment from recurrence	Case by case assessment based on specialist reports	Case by case assessment, very low likelihood of recurrence

17.9.9.1 CARDIOMYOPATHY

In 1995 the WHO/International Society and Federation of Cardiology Task Force defined cardiomyopathies as 'diseases of the myocardium associated with cardiac dysfunction' and classified them according to anatomy and physiology.

DILATED CARDIOMYOPATHY (DCM)

DCM is characterised by dilatation and impaired contraction of one or both ventricles[689]. Patients have decreased systolic function and may or may not develop the clinical signs of heart failure. DCM is responsible for 10 000 deaths and 46 000 hospitilisations in the US and idiopathic DCM is the primary indication for cardiac transplantation[690]. Most patients present between the age of 20 and 60 years but DCM can occur at any age. Presentation can be in a number of different ways including[691] [692]

- Symptoms of heart failure (most common)
- Atrial and/or ventricular arrhythmias
- Thromboembolic complications
- Sudden death
- Incidental finding in asymptomatic person

[689] Richardson P, McKenna W, Bristow M, Maisch B, Mautner B, O'Connell J, Olsen E, Thiene G, Goodwin J, Gyarfas I, Martin I, Nordet P. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies. Circulation. 1996;93(5):841.
[690] Manolio TA, Baughman KL, Rodeheffer R, Pearson TA, Bristow JD, Michels VV, Abelmann WH, Harlan WR. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute workshop. Am J Cardiol. 1992;69(17):1458.
[691] Dec GW, Fuster V. Idiopathic dilated cardiomyopathy. N Engl J Med. 1994;331(23):1564.
[692] Abelmann WH, Lorell BH. The challenge of cardiomyopathy. J Am Coll Cardiol. 1989;13(6):1219.

DCM can be caused by a number of different disorders including[693] idiopathic (50%), myocarditis (9%), ischaemic heart disease (7%), infiltrative disease (5%), peripartum cardiomyopathy (4%), hypertension (4%) and others.

The prognosis of patients with DCM is related to the cause of the cardiomyopathy and hence any decision regarding a person's fitness to work at sea must only be taken after a detailed individual risk assessment including a comprehensive specialist report detailing the cause of the DCM, current symptoms and treatment, likely prognosis and the need for follow up. Full consideration must also be given to any other comorbidities, the physical capability of the person and their ability to perform their routine and emergency duties.

HYPERTROPHIC CARDIOMYOPATHY (HCM)

HCM is a genetically determind heart muscle disease caused by mutations in one of several sarcomere genes. The prevalence is estimated at 1 in 200 adults (0,5%)[694]and there is huge variation in the location, pattern and extent of left ventricular hypertrophy. HCM patients can develop one or more morphological abnormalities:

- LV outflow obstruction
- Diastolic dysfunction
- Myocardial ischaemia
- Mitral regurgitation
- Systolic dysfunction ie end stage with an ejection fraction less than 50%.

These structural changes and functional abnormalities lead to a variety of symptoms broadly classified as those related to heart failure, chest pain or arrhythmias. For the majority of patients HCM is not progressive and the clinical course is relatively benign[695]. The major disease related complications are ventricular arrhythmias leading to sudden death, chest pain, progressive heart failure, atrial arrhythmias and embolic stroke[696].

[693] Felker GM, Thompson RE, Hare JM, Hruban RH, Clemetson DE, Howard DL, Baughman KL, Kasper EK. Underlying causes and long-term survival in patients with initially unexplained cardiomyopathy. N Engl J Med. 2000;342(15):1077.
[694] Semsarian C, Ingles J, Maron MS, Maron BJ. New perspectives on the prevalence of hypertrophic cardiomyopathy. J Am Coll Cardiol. 2015 Mar;65(12):1249-54.
[695] Maron BJ, Casey SA, Poliac LC, Gohman TE, Almquist AK, Aeppli DM. Clinical course of hypertrophic cardiomyopathy in a regional United States cohort. JAMA. 1999;281(7):650.
[696] Gersh BJ, Maron BJ, Bonow RO, Dearani JA, Fifer MA, Link MS, Naidu SS et al. 2011 ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2011;124(24):2761.

Mortality

Studies of large, unselected HCM patient populations have shown that the annual mortality rate is approsimately 1% or less per year[697] [698]and in a report from a referral population of 312 patients it was observed that 23% lived at least 75 years[699]. The major causes of death are sudden cardiac death (SCD - 51%), heart failure (36%) and stroke (13%)[700] [701]. SCD is most common in younger people while death from heart failure or stroke occurred more commonly from midlife and beyond. Many factors can influence the mortality rate and these include:

- Age of diagnosis – in a review of 277 patients who were followed for 8 years the mean age of death was 56 years. The annual mortality compared to the general population was substantially increased in patients diagnosed during childhood (1,3 v 0,8%) but not in those identified as adults (2,2 v 1,9%)[702].
- Presence of symptoms – the same study showed that advanced symptoms at diagnoses increased the likelihood of HCM related death.
- Obstruction – regardless of symptom status the presence of left ventricular outflow tract (LVOT) obstruction at rest >30mmHg is an independent predictor of progressive heart failure and stroke death in patients with HCM[703].
- Coronary artery disease – the adverse effect of CAD on prognosis in HCM was demonstrated in a study of 433 adult patients in which 10 year survival was 46%, 71% and 77% for patients with severe, mild/moderate and no CAD respectively[704].
- Heart failure – progression to severe heart failure symptoms (NYHA Class III or IV) is associated with a marked increase in cardiovascular mortality, particularly in patients without LVOT obstruction as referenced above (617).
- Arrythmias – see below

Atrial fibrillation in HCM patients

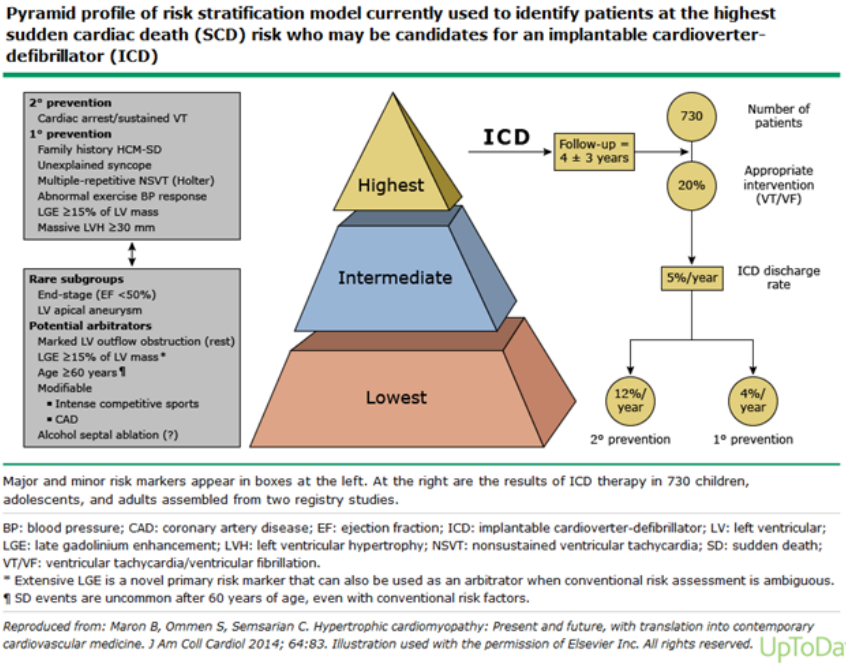
HCM patients have been noted to have a prevalence of AF that is 4 – 6 fold higher than the average population and with an incidence in the range of 2% per year[705]. It is paroxysmal in approximately two thirds of patients but persists in the remainder. AF is often poorly tolerated in patients with HCM and acutely was associated with a worsening of symptoms in 89% of the

[697] Maron BJ, Casey SA, Poliac LC, Gohman TE, Almquist AK, Aeppli DM. Clinical course of hypertrophic cardiomyopathy in a regional United States cohort. JAMA. 1999;281(7):650.
[698] Maron BJ, Rowin EJ, Casey SA, Link MS, Lesser JR, Chan RH, Garberich RF, Udelson JE, Maron MS. Hypertrophic Cardiomyopathy in Adulthood Associated With Low Cardiovascular Mortality With Contemporary Management Strategies. J Am Coll Cardiol. 2015 May;65(18):1915-28.
[699] Maron BJ, Casey SA, Hauser RG, Aeppli DM. Clinical course of hypertrophic cardiomyopathy with survival to advanced age. J Am Coll Cardiol. 2003;42(5):882.
[700] Maron BJ, Olivotto I, Spirito P, Casey SA, Bellone P, Gohman TE, Graham KJ, Burton DA, Cecchi F. Epidemiology of hypertrophic cardiomyopathy-related death: revisited in a large non-referral-based patient population. Circulation. 2000;102(8):858.
[701] Elliott PM, Gimeno JR, Thaman R, Shah J, Ward D, Dickie S, Tome Esteban MT, McKenna WJ. Historical trends in reported survival rates in patients with hypertrophic cardiomyopathy. Heart. 2006;92(6):785.
[702] Maron BJ, Casey SA, Poliac LC, Gohman TE, Almquist AK, Aeppli DM. Clinical course of hypertrophic cardiomyopathy in a regional United States cohort. JAMA. 1999;281(7):650
[703] Maron MS, Olivotto I, Betocchi S, Casey SA, Lesser JR, Losi MA, Cecchi F, Maron BJ. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic cardiomyopathy. N Engl J Med. 2003;348(4):295.
[704] Sorajja P, Ommen SR, Nishimura RA, Gersh BJ, Berger PB, Tajik AJ. Adverse prognosis of patients with hypertrophic cardiomyopathy who have epicardial coronary artery disease. Circulation. 2003;108(19):2342.
[705] Olivotto I, Cecchi F, Casey SA, Dolara A, Traverse JH, Maron BJ. Impact of atrial fibrillation on the clinical course of hypertrophic cardiomyopathy. Circulation. 2001;104(21):2517.

52 patients studed in one series[706]. However symptoms returned to their baseline with control of rhythm and rate.

Ventricular arrhythmias in HCM patients

These are common and can range from isolated premature ventricular beats to non sustained ventricular tachycardia (VT) to sustained VT and ventricular fibrillation (VF). The frequency of ventricular arrhythmias is variable and has been the subject of many studies but as stated above the rate of sudden death is estimated at around 1%. As an example a study of 178 patients who underwent ambulatory monitoring showed premature ventricular beats occurred in 88% of patients and non sustained VT was present in 31%[707]. Clincally documented sustained VT was rare. Equally the clinical presentation of such arrhythmias was highly variable and ranges from an absence of symptoms to palpitations and presyncope/syncope to sudden death.



Pyramid profile of risk stratification model currently used to identify patients at the highest sudden cardiac death (SCD) risk who may be candidates for an implantable cardioverter-defibrillator (ICD)

Major and minor risk markers appear in boxes at the left. At the right are the results of ICD therapy in 730 children, adolescents, and adults assembled from two registry studies.

BP: blood pressure; CAD: coronary artery disease; EF: ejection fraction; ICD: implantable cardioverter-defibrillator; LV: left ventricular; LGE: late gadolinium enhancement; LVH: left ventricular hypertrophy; NSVT: nonsustained ventricular tachycardia; SD: sudden death; VT/VF: ventricular tachycardia/ventricular fibrillation.
* Extensive LGE is a novel primary risk marker that can also be used as an arbitrator when conventional risk assessment is ambiguous.
¶ SD events are uncommon after 60 years of age, even with conventional risk factors.

Reproduced from: Maron B, Ommen S, Semsarian C. Hypertrophic cardiomyopathy: Present and future, with translation into contemporary cardiovascular medicine. J Am Coll Cardiol 2014; 64:83. Illustration used with the permission of Elsevier Inc. All rights reserved. UpToDate®

A detailed specialist assessment must form the basis of an individualised risk assessment for any person with a history of HCM. This should include details of current symptoms, exercise tolerance, treatment, follow up requirements and prognosis. As part of this assessment there should alsok be a risk stratification for sudden cardiac death such as the one shown here. Again comorbidities, physical capability and the person's ability to perform his/her regular and emergency duties must also be taken into consideration.

RESTRICTIVE CARDIOMYOPATHY (RCM)

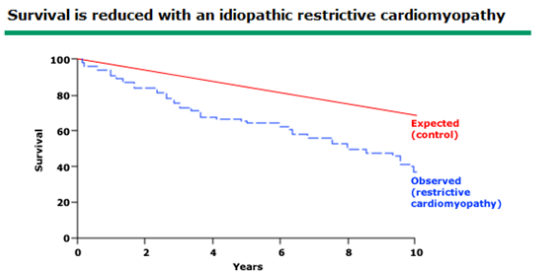
RCM is characterised by non dilated ventricles with impaired ventricular filling. Systolic function remains normal, at least in the early stages and hypertrpophy is classically absent. RCM is much less common than either DCM or HCM outside of the tropics but is a frequent cause of death in

[706] Robinson K, Frenneaux MP, Stockins B, Karatasakis G, Poloniecki JD, McKenna WJ. Atrial fibrillation in hypertrophic cardiomyopathy: a longitudinal study. J Am Coll Cardiol. 1990;15(6):1279.
[707] Adabag AS, Casey SA, Kuskowski MA, Zenovich AG, Maron BJ. Spectrum and prognostic significance of arrhythmias on ambulatory Holter electrocardiogram in hypertrophic cardiomyopathy. J Am Coll Cardiol. 2005;45(5):697.

Africa, India, South and Central America and Asia primarily due to the high incidence of endomyocardial fibrosis in these areas[708].

Causes of RCM can be classified as familial non infiltrative, infiltrative, storage diseases and others eg diabetes, scleroderma and endomyocardial fibrosis. The disease may present at any age and individuals usually present with signs of pulmonary and systemic congestion[709] [710].

Patients with RCM also appear to have reduced survival as demonstrated in a series of 94 patients followed for 68 months at the Mayo Clinic. Their survival was significantly lower than expected compared to an age and gender matched group – 64 v 85% at five years and 37 v 70% at ten years. Approximately two thirds of death were cardiovascular and due to heart failure, sudden death, arrhythmia or cerebrovascular accident. Among the survivors 28%, 46% and 17% were in NYHA Class I, II and III respectively[711]. Adverse risk factors included male gender, age over 70 years, increasing NYHA class and left atrial diameter > 60mm. Survial was not related to the presence of atrial fibrillation or left ventricular systolic dysfunction.



Kaplan-Meier survival curves in 94 patients with an idiopathic restrictive cardiomyopathy and in an aged and sex-matched control group (expected) show that survival with a restrictive cardiomyopathy at 5 (64 versus 85 percent expected) and 10 years (37 versus 70 percent, p <0.0001).
Data from Ammash, NM, Seward, JB, Bailey, KR, et al. Circulation 2000; 101:2490.
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Again a detailed specialist report will be necessary in order to conduct a full, individualised risk assessment which must take into account any comorbidities, likely course of the disease, current functional status and physical capability and the ability for the person to fulfil his routine and emergency tasks.

ARRYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY/DYSPLASIA (ARVC/D)

ARVC/D is a clinical entity characterised by ventricular arrhythmias and a characteristic ventricular pathology[712]. It's prevalence is reported as approximately 1 in 2000 to 1 in 5000[713] and it is an important cause of SCD in young adults accounting for 11% of cases overall and 22% of cases in young athletes in a study from Italy[714]. On the other hand it is rarely diagnosed in the US which may reflect a different genetic prevalence or be due to under recognition of the diease. Studies have suggested that 30% of cases are familial and presentation is most common

[708] Kushwaha SS, Fallon JT, Fuster V. Restrictive cardiomyopathy. N Engl J Med. 1997;336(4):267.
[709] Benotti JR, Grossman W, Cohn PF. Clinical profile of restrictive cardiomyopathy. Circulation. 1980;61(6):1206.
[710] Ammash NM, Seward JB, Bailey KR, Edwards WD, Tajik AJ. Clinical profile and outcome of idiopathic restrictive cardiomyopathy. Circulation. 2000;101(21):2490.
[711] Kubo T, Gimeno JR, Bahl A, Steffensen U, Steffensen M, Osman E, Thaman R, Mogensen J, Elliott PM, Doi Y, McKenna WJ. Prevalence, clinical significance, and genetic basis of hypertrophic cardiomyopathy with restrictive phenotype. J Am Coll Cardiol. 2007;49(25):2419.
[712] Gemayel C, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular cardiomyopathy. J Am Coll Cardiol. 2001;38(7):1773.
[713] Corrado D, Thiene G. Arrhythmogenic right ventricular cardiomyopathy/dysplasia: clinical impact of molecular genetic studies.
[714] Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med. 1998;339(6):364.

between the ages of 10 and 50 years with a mean of 30 years[715]. Up to 40% of patients are asymptomatic whilst the remainder show symptoms of[716]:

- Palpitations (67%)
- Syncope (32%)
- Atypical chest pain (27%)
- Dyspnea (11%)
- RV failure (6%)

Palpitations and syncope are classically due to ventricular arrhythmias which vary from frequent ventricular premature beats to sustained VT. The frequency increases with the severity of the disease with one study showing ventricular arrhythmias in 100%, 82% and 23% of patients with severe, moderate and mild disease respectively.

For patients who are asymptomatic and diagnosed on screening one study has shown that 9.6% develop structural disease that can be seen on echocardiography and 50% had symptomatic ventricular arrythmias[717]. For patients with VT the prognosis is less clear. Patients with mild disease and non sustained VT appear to have a relatively low risk of arrhythmic death and in the report from Italy referenced above only 1 of 49 patients treated with anti arrhythmic drugs died diring the follow up period that averaged 8.5 years. He had stopped his therapy 20 days earlier. Other studies have shown that the following groups of patients are at higher risk of SCD or ICD activation if one is fitted[718] [719]:

- Younger patients
- Patients with syncope
- Patients with a previous history of cardiac arrest or of VT with compromise
- Patients with left ventricular involvement

As stated previously a full and detailed individual risk assessment must be completed for a person with a history or family history of ARVC/D. A specialist report also containing relevant family history is an essential part of the decision making process.

[715] Nava A, Bauce B, Basso C, Muriago M, Rampazzo A, Villanova C, Daliento L, Buja G, Corrado D, Danieli GA, Thiene G. Clinical profile and long-term follow-up of 37 families with arrhythmogenic right ventricular cardiomyopathy. J Am Coll Cardiol. 2000;36(7):2226.
[716] Hulot JS, Jouven X, Empana JP, Frank R, Fontaine G. Natural history and risk stratification of arrhythmogenic right ventricular dysplasia/cardiomyopathy. Circulation. 2004;110(14):1879.
[717] Nava A, Bauce B, Basso C, Muriago M, Rampazzo A, Villanova C, Daliento L, Buja G, Corrado D, Danieli GA, Thiene G. Clinical profile and long-term follow-up of 37 families with arrhythmogenic right ventricular cardiomyopathy. J Am Coll Cardiol. 2000;36(7):2226.

[718] Corrado D, Leoni L, Link MS, Della Bella P, Gaita F, Curnis A, Salerno JU, Igidbashian D, Raviele A, Disertori M, Zanotto G, Verlato R, Vergara G, Delise P, Turrini P, Basso C, Naccarella F, Maddalena F, Estes NA 3rd, Buja G, Thiene G. Implantable cardioverter-defibrillator therapy for prevention of sudden death in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia. Circulation. 2003;108(25):3084.
[719] Corrado D, Calkins H, Link MS, Leoni L, Favale S, Bevilacqua M, Basso C, Ward D, Boriani G, Ricci R, Piccini JP, Dalal D, Santini M, Buja G, Iliceto S, Estes NA 3rd, Wichter T, McKenna WJ, Thiene G, Marcus FI. Prophylactic implantable defibrillator in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia and no prior ventricular fibrillation or sustained ventricular tachycardia. Circulation. 2010;122(12):1144.

UNCLASSIFIED CARDIOMYOPATHIES

This group includes all other cardiomyopathies that do not fit into one of the groups above. Examples include:

- Left ventricular non compaction/isolated ventricular non compaction
- Stress induced cardiomyopathy
- Cirrhotic cardiomyopathy

Whatever the cause of the cardiomyopathy a thorough risk assessment must be carried our for each person who presents for examination. Relevant information must be collected and all aspects of fitness considered.

17.9.9.2 PERICARDITIS

Acute pericarditis is the most common disorder involving ther pericardial sac. It is more common in adults (20 to 50 years old) and in men[720]. The true incidence and prevalence of the disease are unknown due to under diagnosis but it may account for up to 5% of presentations to emergency departments with chest pain and up to 0.1% of hospital admissions. As many as 90% of cases are either idiopathic or due to viral infections including Coxsackie, mumps, EBV, CMV, varicella, rubella and HIV[721] [722]. There are multiple other causes eg systemic autoimmune disorders, metabolic disorders, post myocardial infarction and neoplasms. Mycobacterium tuberculosis is a common cause in developing countries[723].

In most cases pericarditis follows a relatively benign course and it is not necessary to search for the aetiology but rather focus on excluding a significant effusion or cardiac tamponade (rare). Constrictive pericarditis may occur in 1% of patients with acute idiopathic pericarditis but, like tamponade, it is more common in those with a specific aetiology. Approximately 15 – 30% of patients not treated in the acute stage will develop recurrent pericarditis.

The assessment of a person who has suffered with pericarditis should include a specialist report outlining the possible cause, any complications and the risk of recurrence within the validity period. A detailed individual risk assessment must be performed in every case, also considering any underlying disorder and physical capability.

[720] Ariyarajah V, Spodick DH. Acute pericarditis: diagnostic cues and common electrocardiographic manifestations. Cardiol Rev. 2007;15:24-30.
[721] Imazio M, Brucato A, Mayosi BM, et al. Medical therapy of pericardial diseases: part I: idiopathic and infectious pericarditis. J Cardiovasc Med (Hagerstown). 2010;11:712-722.
[722] Little WC, Freeman GL. Pericardial disease. Circulation. 2006;113:1622-1632.
[723] Ariyarajah V, Spodick DH. Acute pericarditis: diagnostic cues and common electrocardiographic manifestations. Cardiol Rev. 2007;15:24-30.

17.9.9.3 HEART FAILURE

An ageing population and the prolongation of life for those with cardiac disease eg hypertension, coronary artery disease has led to an increasing prevalence of heart failure and despite improvements in therapy the morbidity and mortality rates remain very high[724]. It is very difficult to be precise with regards to the incidence and prevalence of heart failure as there is huge variation in the definition of the condition and the methods used to establish its presence. In 2013 the American Heart Association estimated that there were 5.1 million people with heart failure in the US in 2006[725] and there are an estimated 23 million people with heart failure worldwide[726]. However all studies agree that there is an increase in both incidence and prevalence with age e.g. the Framingham study referenced above shows a prevalence of 8 per 1000 at 50 – 59 years, increasing to 66 per 1000 at 80 – 89 years. The prevalence is also reported as being 25% higher in African-Americans than whites.

Many conditions predispose to the development of heart failure and the impact of these is variable. Looking at the population attributable risk (the reduction in incidence that would be observed if the population were entirely unexposed) in a study that followed over 13000 patients over 19 years the risk factors and PAR for heart failure were[727]:

- Coronary heart disease – relative risk 8.1; PAR 62%
- Cigarette smoking – relative risk 1.6, PAR 17%
- Hypertension – relative risk 1.4, PAR 10%
- Obesity – relative risk 1.3, PAR 8% percent
- Diabetes – relative risk 1.9, PAR 3%
- Valvular heart disease – relative risk 1.5, PAR 2%

Any and all of these conditions in a person must also be taken into consideration when assessing their fitness to work at sea.

Long term mortality still remains high although it has improved over time. The Franingham study showed age adjusted mortality decreased from 1950 – 1969 compared to 1990 – 1999[728]:

[724] Ho KK, Pinsky JL, Kannel WB, Levy D. The epidemiology of heart failure: the Framingham Study. J Am Coll Cardiol. 1993;22(4 Suppl A):6A. Circulation. 2006;113(13):1634.
[725] Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S. Heart disease and stroke statistics--2013 update: a report from the American Heart Association. Circulation. 2013;127(1):e6.
[726] McMurray JJ, Petrie MC, Murdoch DR, Davie AP. Clinical epidemiology of heart failure: public and private health burden. Eur Heart J. 1998;19 Suppl P:P9.
[727] He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, Whelton PK. Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study. Arch Intern Med. 2001;161(7):996.
[728] Levy D, Kenchaiah S, Larson MG, Benjamin EJ, Kupka MJ, Ho KK, Murabito JM, Vasan RS. Long-term trends in the incidence of and survival with heart failure. N Engl J Med. 2002;347(18):1397.

Adverse predictors of survival in patients with heart failure

High New York Heart Association (NYHA) functional class
Reduced left ventricular ejection fraction
Diastolic dysfunction
Right ventricular dysfunction
Reduced right ventricular ejection fraction
Right ventricular enlargement
Tricuspid regurgitation
Low peak VO2 with maximal exercise
Exercise hemodynamics
Resting sinus tachycardia
Signs of reduced tissue perfusion
Low mean arterial pressure
Renal insufficiency
Neurohumoral activation
Hyponatremia due to increased antidiuretic hormone secretion
Increases in plasma renin, norepinephrine, brain natriuretic peptide, and big endothelin-1
Comorbid factors
Diabetes mellitus
Ischemic heart disease, including extent of coronary artery disease
Additional predictors
Echocardiographic findings
Hemodynamic parameters
Increase in pulse pressure
Low heart rate response to exercise
Reduced heart rate variability
Hematologic abnormalities
Low lymphocyte percentage
White blood cell count above 7000/μL in ischemic cardiomyopathy
Erythrocyte sedimentation rate above 15 mm/h
Other
Left bundle branch block
Depression
Mutant allele of the adenosine monophosphate deaminase 1 gene
Nocturnal Cheyne-Stokes respiration
More than 30 apneic or hypopneic episodes per hour

- One year mortality declined from 30 % to 28% in men and from 28% to 24% in women

- Five year mortality declined from 70% to 59% in men and from 57% to 45% in women

There was a significant overall trend of a 12% reduction in mortality per decade during this time period with almost all of the improvement occurring after 1980 and particularly after 1990.

Many individual factors have been used to try and predict survival in heart falure and identification of these factors should be included in the assessment. Examples include the EFFECT model, the Heart Failure Survival Score and the Seattle Heart Failure Model.

In addition to these, comorbidities and the cause of the heart failure should also be taken into account when making an attempt to predict the prognosis. For example, the prognosis is worse in patient with diabetes mellitus[729] [730] and ischaemic cardiomyopathy where the extent of the coronary artery disease is important[731] [732]. Ventricular tachycardia is also an adverse prognostic factor[733]. Other demographic factors which influence the survival of patients with heart failure include age, gender and the cause of the cardiomyopathy:

- Age – the mortality rate in treated patients with heart failure increases with age[734] [735]
- Gender – the prognosis is generally better in women than men[736] [737]
- Race – different studies have revealed contrasting findings
- Cause of cardiomyopathy – this was studied in 1230 patients with an initially unexplained cardiomyopathy and compared to a reference group of those with idiopathic cardiomyopathy[738]:

[729] Shindler DM, Kostis JB, Yusuf S, Quinones MA, Pitt B, Stewart D, Pinkett T, Ghali JK, Wilson AC. Diabetes mellitus, a predictor of morbidity and mortality in the Studies of Left Ventricular Dysfunction (SOLVD) Trials and Registry. Am J Cardiol. 1996;77(11):1017.
[730] Pocock SJ, Wang D, Pfeffer MA, Yusuf S, McMurray JJ, Swedberg KB, Ostergren J, Michelson EL, Pieper KS, Granger CB. Predictors of mortality and morbidity in patients with chronic heart failure. Am J Cardiol. 1996;77(11):1017.
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[733] Wilson JR, Schwartz JS, Sutton MS, Ferraro N, Horowitz LN, Reichek N, Josephson ME. Prognosis in severe heart failure: relation to hemodynamic measurements and ventricular ectopic activity. J Am Coll Cardiol. 1983;2(3):403.
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[735] Jong P, Vowinckel E, Liu PP, Gong Y, Tu JV. Prognosis and determinants of survival in patients newly hospitalized for heart failure: a population-based study. Arch Intern Med. 2002;162(15):1689.
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[737] Frazier CG, Alexander KP, Newby LK, Anderson S, Iverson E, Packer M, Cohn J, Goldstein S, Douglas PS. Associations of gender and etiology with outcomes in heart failure with systolic dysfunction: a pooled analysis of 5 randomized control trials.
J Am Coll Cardiol. 2007;49(13):1450.
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- Survival was better in patients with peripartum cardiomyopathy (hazard ratio 0.31).
- Survival was worse in patients with infiltrative myocardial disease, particularly amyloidosis or hemochromatosis (hazard ratio 7.41 and 8.88, respectively), HIV infection (hazard ratio 5.86), doxorubicin therapy (hazard ratio 3.46), ischemic heart disease (hazard ratio 1.52), or connective tissue disease (hazard ratio 1.75).
- Survival was the same in patients with hypertension, myocarditis, sarcoidosis, substance abuse, or other causes.

The two main causes of death in heart failure are sudden or arrhythmic death and progressive pump failure[739] [740] and the three major classes of drugs that are used to improve survival in heart failure have different effects on the causes of death. The benefit of ACE inhibitors is derived from prevention of progressive myocardial dysfunction, rather than the prevention of sudden cardiac death as referenced above. However beta blockers and aldosterone antagonists reduce both sudden cardiac death and progressive pump failure[741] [742].

Given the multiple and varied aetilogies of heart failure and the complexities of predicting adverse events and prognosis it is especially important that an individualized risk assessment is carried out on all persons with heart failure. A detailed specialist report outlining all of the above factors is essential and the physical capability of the seaefarer to perform his/her routine or emergency duties must also be assessed. A restricted or time limited certificate may well be appropriate if the person is considered fit to work at sea at all.

17.9.9.4 THORACIC AORTIC ANEURYSM (TAA)

Complications of aortic aneurysmal disease (thoracic and abdominal) are a leading cause of death in the US, particularly in those over 55 years[743]. TAA represents about one third of aortic aneurysm admissions with the remainder related to abdominal aortic disease. However it is difficult to estimate it's prevalence and incidence as it is a clinically silent disease and fatalities due to a TAA are also often attributed to other causes in the absence of a post mortem[744]. In

[739] Effects of enalapril on mortality in severe congestive heart failure. Results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). The CONSENSUS Trial Study Group. N Engl J Med. 1991;325(5):293.
[740] Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure. The SOLVD Investigators. N Engl J Med. 1991;325(5):293.
[741] Bonet S, AgustíA, Arnau JM, Vidal X, Diogène E, Galve E, Laporte JR. Beta-adrenergic blocking agents in heart failure: benefits of vasodilating and non-vasodilating agents according to patients' characteristics: a meta-analysis of clinical trials. Arch Intern Med. 2000;160(5):621.
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[743] http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html
[744] Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, Eagle KA, Hermann LK, Isselbacher EM et al. 2010. ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266.

two separate studies the incidence of TAA was estimated at 5.6 and 10.4 cases per 100 000 pateint years and there appears to be a real increase in the incidence of TAA over the past decades[745] [746] along with an increasing incidence of TAA rupture[747]. Thoracic aneurysms are most likely to occur in the sixth and seventh decade of likfe and occur in men two to four times more commonly than in females. Most TAAs are degenerative and occur in association with the risk factors for atherosclerosis eg smoking, high cholesterol and hypertension[748] . Hypertension is an important risk factor that is present in over 60% of patients with a TAA[749], however despite diabetes mellitus being associated with atherosclerosis, similar to abdominal aortic aneurysm, it is negatively correlated with TAA[750]. Apart from this it is estimated that as many as 20% of patients with a TAA have a family history of aneurysmal disease independent of known genetic syndromes[751]. Other risk factors for a TAA include:

- Aortitis
- Infection
- Inflammatory disorders eg giant cell artertitis, Takayasu arteritis, rheumatoid arthritis, ankylosing spondylitis
- Genetic predisposition
    - Familial
    - Marfan syndrome
    - Loeys-Dietz syndrome
    - Ehlers-Danlos syndrome
- Congenital conditions
    - Bicuspid aortic valve

The natural history of a TAA is one of slow expansion with an increasing risk of sudden dissection as the aorta enlarges. The rate of expansion varies from 0.1 to 1.0 cm per year depending upon aetiology, diameter and location within the aorta[752] [753] [754]. The risk of complications of TAA (rupture and dissection) increase with larger aortic diameter. The annual risk of rupture is <2% for TAAs between 4.0 and 4.9cm but nearly 7% for TAAs >6 cm[755]. In

[745] Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, Cherry KJ, Joyce JW, Lie JT. Thoracic aortic aneurysms: a population-based study. Surgery. 1982;92(6):1103.
[746] Clouse WD, Hallett JW Jr, Schaff HV, Gayari MM, Ilstrup DM, Melton LJ 3rd. Improved prognosis of thoracic aortic aneurysms: a population-based study. JAMA. 1998;280(22):1926.
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[749] Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, Cherry KJ, Joyce JW, Lie JT. Thoracic aortic aneurysms: a population-based study. Surgery. 1982;92(6):1103.

[750] Prakash SK, Pedroza C, Khalil YA, Milewicz DM. Diabetes and reduced risk for thoracic aortic aneurysms and dissections: a nationwide case-control study. J Am Heart Assoc. 2012 Apr;1(2) Epub 2012 Apr 24.
[751] Coady MA, Davies RR, Roberts M, Goldstein LJ, Rogalski MJ, Rizzo JA, Hammond GL, Kopf GS, Elefteriades JA. Familial patterns of thoracic aortic aneurysms. Arch Surg. 1999;134(4):361.
[752] Kuzmik GA, Sang AX, Elefteriades JA. Natural history of thoracic aortic aneurysms. J Vasc Surg. 2012 Aug;56(2):565-71.
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another study of 370 patients the median diameter of the aorta at the time of rupture was 5.9cm for ascending aneurysms and 7.2 cm for descending aneurysms[756]. As the aorta approaches 6cm its distensability decreases rapidly and the vessel loses much of its inate elasticity. At a blood pressure of 200mmHg the stress generated in the wall of an aorta at this diameter can reach or exceed the maximum tensile strength of aortic tissue[757]. In several series the aneurysm ruptured in 32 – 68% of medically treated patients and accounted for 32 – 47% of deaths[758] [759] [760]. Survival rates were reported as 65% at 1 year, 36% at three years and 20% at five years. Concomitant cardiovascular disease is the second most common cause of death in these patients.

When assessing a person with a TAA the seafarer's doctor must carry out a detailed individual risk assessment that should include but is not limited to the size of the aneurysm, likely progression and likelihood of surgical intervention, risk assessment for rupture/dissection, follow up requirements and concomitant medical diagnoses and physical capability. A specialist report will need to be acquired and a restricted or time limited certificate may be appropriate if the person is deemed fit to work at sea at all.

Reviewed 2016

[756] Coady MA, Rizzo JA, Hammond GL, Kopf GS, Elefteriades JA. Surgical intervention criteria for thoracic aortic aneurysms: a study of growth rates and complications. Ann Thorac Surg. 1999;67(6):1922.
[757] Koullias G, Modak R, Tranquilli M, Korkolis DP, Barash P, Elefteriades JA. Mechanical deterioration underlies malignant behavior of aneurysmal human ascending aorta. J Thorac Cardiovasc Surg. 2005 Sep;130(3):677-83.
[758] Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, Cherry KJ, Joyce JW, Lie JT. Thoracic aortic aneurysms: a population-based study. Surgery. 1982;92(6):1103.
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[760] Crawford ES, DeNatale RW. Thoracoabdominal aortic aneurysm: observations regarding the natural course of the disease. J Vasc Surg. 1986;3(4):578.

17.10 J 00-99 RESPIRATORY SYSTEM

17.10.1 NOSE, THROAT AND SINUS CONDITIONS

J 02-04 J 30-39	Nose, throat and sinus conditions impairing for individual. May recur. Transmission of infection to food/other crew in some	T – Until resolved P – If impairing and recurrent.	Case-by-case assessment	When treatment complete if no factors predisposing to recurrence

17.10.1.1 ACUTE RHINITIS

Acute rhinitis is an acute self limiting inflammation that may involve the nose, sinuses, throat and larynx. It is responsible for 11% of primary care consultations in western countries and it is estimated that adults suffer two to three infections per year[761]. Symptoms vary with person and pathogen but have usually resolved within two weeks. Complications can include otitis media, acute rhinosinusitis and more rarely acute rhinitis can lead to an exacerbation of asthma or chronic obstructive airway disease or a community acquired pneumonia.

Any person suffering from acute rhinitis is temporarily unfit and should not join a ship until symptoms are resolved. Closer monitoring of persons with a previous history of otitis media, sinusitis, pneumonia, asthma or chronic obstructive airway disease may be warranted and if a flight is necessary to join a ship the person should also be fit to fly.

17.10.1.2 ALLERGIC RHINITIS

Allergic rhinitis is a common inflammatory condition of the nasal mucosa, characterised by nasal pruritus, sneezing, rhinorrhoea, and nasal congestion. Frequently there is associated palate, throat, ear and eye itching as well as eye redness, puffiness, and watery discharge. Allergic rhinitis is common with one UK survey showing that 27% of adults suufered with allergic rhinitis based on the reporting of symptoms [762]. However prevalence varies between countries with lower rates reported in several Eastern European countries, Indonesia, Greece, China, Taiwan, Uzbekistan, India, and Ethiopia[763]. Symptoms can be begin at any age although 80% report the onset of symptoms before the age of 20 years[764]. Follow up is required 2 – 4 weeks after treatment has started to review ongoing symptoms and the effect of medication. The majority of patients who experience symptoms as a young adult will show improvement if not resolution over the following years[765]. However complications such as asthma and acute or chronic sinusitis as well as the side

[761] Fry J, Sandler G. Common conditions. Their nature, prevalence and care. Dordrecht, The Netherlands: Kluwer Academic. 1993
[762] Gupta R, Sheifk A et al. Burden of allergic disease in the UK: secondary analyses of national databases. Clin Exp Allergy; 2004 Apr;34(4):520-6.
[763] Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Lancet; 1998 Apr 25;351(9111):1225-32.
[764] Skoner DP. Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin Immunol; 2001 Jul;108(1 Suppl):S2-8.
[765] Greisner WA 3rd, Settipane RJ, Settipane GA. Natural history of hay fever: a 23-year follow-up of college students. Allergy Asthma Proc; 1998 Sep-Oct;19(5):271-5.

effects of medication can develop. Any risk assessment of a person must include the impact of symptoms, complications and the effects of any medication prescribed and taken.

17.10.1.3 RHINOSINUSITIS

ACUTE RHINOSINUSITIS

Acute rhinosinusitis is defined as symptomatic inflammation of the nasal cavity and paranasal sinuses lasting less than four weeks. It is most commonly associated with acute rhinitis caused by a viral infection and resolves within 7 – 10 days. Acute viral rhinitis is complicated by a bacterial infection in 0.5 – 13% of cases[766] and symptoms here may last up to four weeks. Treatment is often symptomatic although anti microbial agents are indicated for bacterial rhinosinusitis. There is a low risk of complications in adults and the illness is usually self limiting. Recurrence may occur if the infection is only partially treated, if there is an ongoing viral illness or in persons with structural anatomical variants. Any person suffering symptoms of acute rhinosinusitis are temporarily unfit. Individual risk assessment must include any ongoing symptoms, the risk of recurrence and the effects of medication.

CHRONIC RHINOSINUSITIS

Chronic sinusitis is inflammation of the paranasal sinuses lasting for more than 12 weeks. Symptoms include facial pressure, rhinorrhoea, postnasal drainage, congestion, and general malaise. Prevalence varies worldwide largely due to the differences in diagnostic criteria but in the UK it is estimated that there are 25 cases of chronic sinusitis per 10,000 person-years in an average primary care surgery[767]. It can occur at any age but a number of studies have shown the mean age of diagnosis to be 39 years of age[768].

Chronic sinusitis is the end point (sinonasal inflammation) from many different causes, not a disease entity in and of itself. The main cause is thought to be anatomical obstruction of the osteomeatal complex (a common drainage pathway for several sinuses) leading to inadequate sinus drainage of mucus. Conditions that impair normal mucociliary clearance (the manner in which mucus is produced and characteristically moved out of the sinuses into the nasal cavity) are also implicated. They can be categorised into three overlapping groups:

- Genetic/physiological factors (e.g., cystic fibrosis/primary ciliary dyskinesia).
- Environmental factors (e.g., smoking).
- Structural factors (e.g., severe mid-septal deviations).

[766] Gwaltney JM Jr. Acute community-acquired sinusitis. Clin Infec Dis; 1996 Dec;23(6):1209-23; quiz 1224-5.
[767] McCormick A, Fleming D, Charlton J. Morbidity statistics from general practice: fourth national study 1991-1992. Office of Population Censuses and Surveys, series MB5 no 3. London, UK: HMSO; 1995.
[768] Shashy RG, Moore EJ, Weaver A. Prevalence of the chronic sinusitis diagnosis in Olmsted County, Minnesota. Arch Otolaryngol Head Neck Surg. 2004 Mar;130(3):320-3.

Treatment may be with local or systemic medication or surgery along with lifestyle changes and follow up depends on the level of symptoms and the need for surgery. Most patients also suffer a decreased quality of life and the prognosis is variable depending on any underlying factors. An individualized risk assessment must be carried out in each case and take into account the impact of symptoms, use of medication, need for surgery, follow up requirements and general health.

17.10.1.4 TONSILLITIS

Acute tonsillitis is an acute infection of the parenchyma of the palatine tonsils. The clinical distinction between tonsillitis and pharyngitis is unclear in the literature, and the condition is often referred to simply as "acute sore throat". Tonsillitis is common with an annual incidence of 100 per 1000 population in the UK[769]. It is usually viral but may be caused by bacterium, the most common of which is group A beta-haemolytic streptococci which accounts for 5- 10% of all cases of tonsillitis in adults[770]. Tonsillitis is usually a self limiting condition which resolves within 7 days although there is a medium risk of complications such as acute sinusitis, acute otitis media or scarlet fever and some patients may develop recurrent tonsillitis. Any person with an acute tonsillitis is temporarily unfit and an individualised risk assessment must include the impact of symptoms, effects of any medication and the need for further investigation or surgery if the disease is recurrent.

17.10.1.5 EPISTAXIS

Epistaxis is a common problem occurring in 60% of the population. It most often originates in the anterior segment of the nose and is a self limiting condition[771]. Anterior nose bleeds most often result from recurrent irritation or trauma and both anterior and posterior nose bleeds may be caused by a number of associated conditions.:

Anticoagulation therapy. One study suggests an annual incidence of epistaxis of 25% among anticoagulated patients[772] although reversal of therapy was necessary in only 1.5 per 1000 patient years[773].

Epistaxis is the most common presenting symptom among patients with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease)[774]. The bleeding can be quite difficult to

[769] BMJ Clinical Evidence. Recurrent throat infections (tonsillitis).
[770] Snow V, Mottur-Pilson C, Cooper RJ, Hoffman JR. Principles of appropriate antibiotic use for acute pharyngitis in adults.Ann Intern Med. 2001;134(6):506.
[771] Kucik CJ, Clenney T. Management of epistaxis. Am Fam Physician. 2005;71(2):305.
[772] Lavy J. Epistaxis in anticoagulated patients: educating an at-risk population. Br J Haematol. 1996;95(1):195.
[773] Nitu IC, Perry DJ, Lee CA. Clinical experience with the use of clotting factor concentrates in oral anticoagulation reversal. Clin Lab Haematol. 1998;20(6):363.
[774] Shah RK, Dhingra JK, Shapshay SM. Hereditary hemorrhagic telangiectasia: a review of 76 cases. Laryngoscope. 2002;112(5):767.

control in these individuals. The friable lesions may appear to bleed more with treatment than without.

Patients with familial blood dyscrasias, particularly platelet disorders, von Willebrand disease, and hemophilia, are prone to epistaxis.

Recurrent posterior bleeds or massive hemorrhage may be due to aneurysm of the carotid artery[775]. This is of particular concern in a patient with a prior history of head and neck surgery, or following trauma (pseudoaneurysm), but most often posterior bleeds arise spontaneously.

Epistaxis may be a symptom of a nasal neoplasm. The most common tumors associated with epistaxis are squamous cell carcinoma, adenoid cystic carcinoma, melanoma, and inverted papilloma[776]. Nasopharyngeal cancers are more common in patients of Chinese or southeast Asian heritage. Patients who have had significant epistaxis (posterior bleed) should receive a thorough ENT evaluation after the bleeding has been controlled.

The data on the importance of aspirin as a risk factor for epistaxis are not definitive[777]. There is no reported increase in risk associated with other nonsteroidal anti-inflammatory drugs (eg, ibuprofen).

The association between hypertension and epistaxis is uncertain. Multiple studies have related hypertension to nosebleeds[778], although studies specifically exploring this relationship have been unable to confirm the association[779].

Alcohol may increase the risk for epistaxis[780], as may intranasal preparations for seasonal allergies[781].

Whilst the huge majority of nose bleeds are self limiting a minority will require surgical intervention[782][783] and thus place the person and vessel at risk. If a person reports recurrent epistaxis careful assessment to exclude an underlying condition is required and an individual risk assessment performed once the appropriate specialist report has been received.

Reviewed 2015

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[782] Villwock JA, Jones K. Recent trends in epistaxis management in the United States: 2008-2010. JAMA Otolaryngol Head Neck Surg. 2013 Dec;139(12):1279-84.
[783] Kotecha B, Fowler S, Harkness P, Walmsley J, Brown P, Topham J. Management of epistaxis: a national survey. Ann R Coll Surg Engl. 1996;78(5):444.

17.10.2 OBSTRUCTIVE LUNG DISEASE

J 40-44	Chronic bronchitis and/or emphysema Reduced exercise tolerance and impairing symptoms	T – If acute episode P – If repeated severe recurrences or if general fitness requirements cannot be met or if impairing shortness of breath	R, L – Case-by-case assessment More stringency for distant water duties. Consider fitness for emergencies and ability to meet general requirements for physical fitness (C – Physical capability requirements). Annual review.	Not applicable

17.10.2.1 CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a common respiratory condition characterised by airflow limitation that is not fully reversible. It encompasses both chronic bronchitis and emphysema[784] and is primarily caused by cigarette smoking. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gas. Whilst it primarily affects the lungs, COPD also has significant systemic effects.

CLASSIFICATION OF COPD

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) uses the FEV1 and FEV1/FVC to predict the severity of disease:

•Stage I - Mild: FEV1/FVC <70% and FEV1 ≥80% of predicted value, with or without symptoms

•Stage II - Moderate: FEV1/FVC <70% and FEV1 50% to 80% of predicted value, with or without symptoms

•Stage III - Severe: FEV1/FVC <70% and FEV1 30% to 50% of predicted value, with or without symptoms

•Stage IV - Very severe: FEV1/FVC <70% and FEV1 <30% of predicted value or FEV1 <50%, with chronic respiratory failure.

When this is used alongside a tool for evaluating symptom severity eg COPD Assessment Test (CAT) and/or the Modified British Medical Research Council (mMRC) and the degree of airflow obstruction and number of previous exacerbations the patient can be classified into a group for the future risk of exacerbations:

[784] Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of COPD 2016. December 2015. http://www.goldcopd.org

•Group A: Low risk (0-1 exacerbation per year, not requiring hospitalisation, and/or spirometric classification of GOLD 1-2) and less symptom (mMRC 0-1 or CAT <10)

•Group B: Low risk (0-1 exacerbation per year, not requiring hospitalisation, and/or spirometric classification of GOLD 1-2) and more symptom (mMRC ≥2 or CAT ≥10)

•Group C: High risk (≥2 exacerbation per year, or one requiring hospitalisation, and/or spirometric classification of GOLD 3-4) and less symptom (mMRC 0-1 or CAT <10)

•Group D: High risk (≥2 exacerbation per year, or one requiring hospitalisation, and/or spirometric classification of GOLD 3-4) and more symptom (mMRC ≥2 or CAT ≥10).

COPD is more common in older people, especially over the age of 65 years. The Burden of Obstructive Lung Disease (BOLD) Initiative estimates a worldwide population prevalence of COPD for Stages II or higher as equivalent to 10.1+/- 4.8 overall[785]. It's associated mortality in women has more than doubled over the last 20 years and now matches that in men. In the US it is reported to affect more than 5% of the population and is associated with a very high morbidity and mortality[786] and a retrospective study in the UK from 1990 – 1997 estimated COPD prevalence to be 2% in men and 1% in women[787]. As outlined above there are systems to estimate the risk of future acute exacerbations and it appears that the single best predictor is a history of prior exacerbations, regardless of the severity of the COPD[788]. Respiratory infections are estimated to trigger approximately 70% of exacerbations with the remaining 30% being due to environmentalk pollution, pulmonary embolism or unknown aetiology[789].

People with COPD should be monitored on a regular basis and even those with mild and stable disease are likely to require follow up within the 2 year validity period. Hence a restricted or time limited certificate may be appropriate for this reason alone. Patients with a recent acute exacerbation or more severe disease may require more frequent review and hence may be temporarily or permanently unfit to work at sea. The risk of an acute exacerbation during the validity period and the need for acute medical care should also be taken into consideration when issuing a full or limited certificate. When assessing the person with COPD it is also important that the seafarer's doctor considers the person's ability to meet the general physical capability requirements and to perform any physically challenging aspect of their routine or emergency duties. A specialist report outlining the current medical regimen, response to

[785] Buist AS, Vollmer WM, McBurnie MA. Worldwide burden of COPD in high- and low-income countries. Part I. The burden of obstructive lung disease (BOLD) initiative. Int J Tubecr Lung Dis. 2008;12:703-708.
[786] Centers for Disease Control and Prevention (CDC). Chronic obstructive pulmonary disease among adults--United States, 2011. MMWR Morb Mortal Wkly Rep. 2012;61(46):938.
[787] Soriano JB, Maier WC, Egger P, et al. Recent trends in physician diagnosed COPD in women and men in the UK. Thorax. 2000;55:789-794.
[788] Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of COPD 2016. December 2015. http://www.goldcopd.org
[789] Sapey E, Stockley RA. COPD exacerbations . 2: aetiology. Thorax. 2006;61(3):250.

therapy, present functional status, lung function, disease progression and follow up requirements is required.

Reviewed 2016

17.10.3 ASTHMA

J 45-46	Asthma (detailed assessment with information from specialist in all new entrants) Unpredictable episodes of severe	T – Until episode resolved, cause investigated (including any occupational link) and effective treatment regime in place In person under age 20 with hospital admission or oral steroid use in last three years P – If foreseeable likelihood of rapid life-threatening asthma attack while at sea or history of uncontrolled asthma, e.g. history of multiple hospital admissions.	R, L – Near-coastal waters only or on ship with doctor if history of moderate adult asthma, with good control with inhalers and no episodes requiring hospital admission or oral steroid use in last two years, or history of mild or exercise-induced asthma that requires regular treatment	Under age 20: If history of mild or moderate childhood asthma, but with no hospital admissions or oral steroid treatment in last three years and no requirements for continuing regular treatment Over age 20: If history of mild or exercise-induced asthma and no requirements for continuing regular treatment.

Asthma is a chronic inflammatory airway disease characterised by intermittent airway obstruction and hyper activity with recurrent episodes of wheezing, breathlessness, chest tightness and coughing[790]. Asthma affects approximately 30 million people in Europe and more than 25 million people in the US whilst the global burden is estimated to be 300 million people, increasing to 400 million by 2025[791][792]. In 2010 the prevalence of asthma in the US was estimated at 8.4% in the previous year there were 1.2 million hospital outpatient department visits and 479 300 hospitilisations for asthma. The highest hospitilisation and death rates were amongst black people[793]. Asthma is a complex disease with multi-gene association interacting with environmental exposure. A patient's genetic make up may predispose them to hyper responsiveness to environmental triggers that include but are not limited to:

- Infections – viral and bacterial
- Allergen exposure
- Occupational exposures
- Food additives and chemicals
- Irritants
- Aspirin and other medications
- Strong emotions

[790] National Institutes of Health; National Heart, Lung, and Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: Guidelines for the diagnosis and management of asthma. August 2007. http://www.nhlbi.nih.gov
[791] Braman SS. The global burden of asthma. Chest. 2006 Jul;130(1 Suppl):4S-12S.
[792] Masoli M, Fabian D, Holt S, et al; Global Initiative for Asthma (GINA) Program. The global burden of asthma: executive summary of the GINA Dissemination Committee report. Allergy. 2004;59:469-478.
[793] Moorman JE, Akinbami LJ, Bailey CM, et al. National surveillance of asthma: United States, 2001-2010. Vital Health Stat 3. 2012;35:1-67.

Asthma is classified based upon an assessment of the current impairment of function and the risk of future exacerbations. This itself is based upon the number of serious exacerbations within the past year[794]:

- Reported daytime and nighttime symptoms and exercise limitation over the previous two to four weeks.
- Current values of peak expiratory flow (PEFR) or FEV1 and FEV1/FVC
- Number of exacerbations in the previous year requiring oral glucocorticoids

Classifying asthma severity and initiating treatment in youths greater than or equal to to 12 years of age and adults

Components of severity		Classification of asthma severity (≥12 years of age)			
		Intermittent	Persistent		
			Mild	Moderate	Severe
Impairment Normal FEV_1/FVC: 8 to 19 years 85 percent 20 to 39 years 80 percent 40 to 59 years 75 percent 60 to 80 years 70 percent	Symptoms	≤2 days/week	>2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	≤2x/month	3 to 4x/month	>1x/week but not nightly	Often 7x/week
	Short-acting $beta_2$-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week but not daily, and not more than 1x on any day	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
	Lung function	• Normal FEV_1 between exacerbations • FEV_1 >80 percent predicted • FEV_1/FVC normal	• FEV_1 ≥80 percent predicted • FEV_1/FVC normal	• FEV_1 >60 but <80 percent predicted • FEV_1/FVC reduced 5 percent	• FEV_1 <60 percent predicted • FEV_1/FVC reduced >5 percent
Risk	**Exacerbations requiring oral systemic glucocorticoids**	**0 to 1/year (see footnote)**	**≥2/year (see footnote)**		
		Consider severity and interval since last exacerbation			
		Frequency and severity may fluctuate over time for patients in any severity category			
		Relative annual risk of exacerbations may be related to FEV_1			
Recommended step for initiating treatment		Step 1	Step 2	Step 3	Step 4 or 5
				And consider short course of oral systemic glucocorticoids	
		In two to six weeks, evaluate level of asthma control that is achieved and adjust therapy accordingly.			

Assessing severity and initiating treatment for patients who are not currently taking long-term control medications. The stepwise approach is meant to assist, not replace, the clinical decision-making required to meet individual patient needs. Level of severity is determined by assessment of both impairment and risk. Assess impairment domain by patient's/caregiver's recall of previous two to four weeks and spirometry. Assign severity to the most severe category in which any feature occurs. At present, data are inadequate to correlate frequencies of exacerbations with different levels of asthma severity. In general, more frequent and intense exacerbations (eg, requiring urgent, unscheduled care, hospitalization, or ICU admission) indicate greater underlying disease severity. For treatment purposes, patients who had ≥2 exacerbations requiring oral systemic glucocorticoids in the past year may be considered the same as patients who have persistent asthma, even in the absence of impairment levels consistent with persistent asthma.
FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; ICU: intensive care unit.
Reproduced from: National Heart, Blood, and Lung Institute Expert Panel Report 3 (EPR 3): Guidelines for the Diagnosis and Management of Asthma. NIH Publication no. 08-4051, 2007.

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The aim of treatment is to achieve the best possible control of symptoms with the fewest medications. Asthma control refers to the extent top which the manifestations of asthma have been reduced or removed by treatment. This involves the avoidance of potential triggers where possible/appropriate and step wise long term therapy with a personal asthma action plan for each patient, often including personal monitoring of peak flow rate. Any acute exacerbation must be treated in a timely and aggressive manner.

[794] National Institutes of Health; National Heart, Lung, and Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: Guidelines for the diagnosis and management of asthma. August 2007. http://www.nhlbi.nih.gov

In assessing a person's fitness to work at sea the seafarer's doctor must consider the current status of the worker, the severity of the asthma and the likelihood of an exacerbation, particularly if the worker is likely to be exposed to triggering factors. The ease or not of access to medical care should also be taken into account and a specialist report may be valuable. An individualised risk assessment must be undertaken.

Reviewed 2016

17.10.4 PNEUMOTHORAX

J 93	Pneumothorax (spontaneous or traumatic) Acute impairment from recurrence	T – 12 months after initial episode or shorter duration as advised by specialist P – After recurrent episodes unless pleurectomy or pleurodesis performed	R – Duties in harbour areas only once recovered	Normally 12 months after initial episode or shorter as advised by specialist. Post surgery based on advice of treating specialist

17.10.4.1 PRIMARY SPONTANEOUS PNEUMOTHORAX

A primary spontaneous pneumothorax (PSP) is one that occurs without a precipitating event in a person who does not have known lung disease. The incidence is reported as 24/1000 000 per year for men and 10/100 000 per year for women in the UK[795] although it is lower in the US. Smoking increases the likelihood of spontaneous pneumothorax by 22 times for men and 8 times for women and the incidence is directly related to the amount smoked [796]. Other risk factors include Marfan's syndrome, homocystinuria and a positive family history of pneumothorax. Patients with a PSP tend to be tall, slender and young males [797]. Whilst it may be impossible to predict which person is most likely to suffer a PSP it is important that persons with any risk factors are assessed thoroughly on an individual basis and that advice regarding smoking cessation is given.

Patients who suffer from a PSP are at risk of recurrence and this is reported at between 30 – 50% over a one to five year period and the highest risk is in the first 30 days [798] and the majority within the first year[799]. Unless an intervention is performed in a patient with one recurrence and third or fourth pneumothorax can be expected in 62% and 83% of patients respectively. Equally these patients are at risk of a contralateral pneumothorax[800] and risk factors for recurrence are female gender,tall stature in men, low body weight and failure to stop smoking.

[795] Gupta D, Hansell ANicols T, et al: Epidemiology of Pneumothorax in England. Thorax 2000; 55:666-671
[796] Bense L, Eklund G, Wimn LG: Smoking and the increased risk of contracting spontaneous pneumothorax. Chest. 1987: 92:1009-1012.
[797] Abolnik IZ, Lossos IS, Gillis D et al: Primary spontaneous pneumothorax in men. Am J Med Sci. 1993: 305: 297-303.
[798] Baumann MH, Strange C, Heffner JE, Light R, Kirby TJ, Klein J, Luketich JD, Panacek EA, Sahn SA, AACP Pneumothorax Consensus Group: Management of spontaneous pneumothorax: an American College of Chest Physicians Delphi consensus statement: Chest. 2001;119(2):590.
[799] Light RW. Pleural Diseases, 6th ed, Lippincott, Williams and Wilkins, Philadelphia 2013
[800] Gobbel WG Jr, Nelson IA et al.: Spontaneous pneumothorax: J Thorac Cardiovasc Surg. 1963:46:331-345

In patients who have suffered at least one recurrence consideration should be given to an intervention to prevent further recurrences, once the acute situation has resolved. Options for prevention of recurrence include video assisted thorascopic surgery (VATS), chemical pleurodesis and thoracotomy. The preferred procedure is best dictated by local practice and expertise. There are advantages and disadvantages to all of the techniques and each patient should be assessed on a case by case basis. Unfortunately no procedure can give a guarantee of no further recurrence although the risk is significantly reduced.

VATS with bleb/bullae resection and pleurodesis: risk of recurrence = 5%[801]

Chemcal pleurodesis with tetracycline derivative: risk of recurrence = 20 – 25%[802]

Talc pleurodesis: risk of recurrence rate = 5 – 8%[803]

Each patient should be assessed on an individual basis and the risk assessment should also take into consideration exposure to sudden barometric pressure changes such as flying or underwater diving.

17.10.4.2 SECONDARY SPONTANEOUS PNEUMOTHORAX

A secondary spontaneous pneumothorax (SSP) is a pneumothorax that occurs as a complication of an underlying lung disease. This can occur in almost every lung disease although the most commonly associated disease are Chronic Obstructive Pulmonary Disease (COPD, Cystic Fibrosis, primary or metastatic lung malignancy and necrotizing pneumonia including TB. COPD is the most common cause of SSP with 50 – 70% in one case series attributed to COPD[804]. SSP is more likely to be severe and life threatening than a PSP and there is a 50% recurrence rate over 3 years in patients who had suffered an SSP associated with COPD[805], hence definitive treatment is recommended after the first incidence. Treatment options are similar to those for PSP.

When considering the risk assessment of a patient who has suffered SSP it is imperative that the underlying disease process is assessed thoroughly along with the risk of recurrence of a pneumothorax.

[801] Ayed AK, Al-Din HJ: The results of thoracoscopic surgery for primary spontaneous pneumothorax: Chest. 2000;118(1):235
[802] Light RW, O'Hara VS, Moritz TE, McElhinney AJ, Butz R, Haakenson CM, Read RC, Sassoon CS, Eastridge CE, Berger R: Intrapleural tetracycline for the prevention of recurrent spontaneous pneumothorax. Results of a Department of Veterans Affairs cooperative study. JAMA. 1990;264(17):2224
[803] Györik S, Erni S, Studler U, Hodek-Wuerz R, Tamm M, Chhajed PN: Long-term follow-up of thoracoscopic talc pleurodesis for primary spontaneous pneumothorax. Eur Respir J. 2007;29(4):757
[804] Noppen M, De Keukeleire T: Pneumothorax. Respiration. 2008;76(2):121.
[805] Light RW, O'Hara VS, Moritz TE, McElhinney AJ, Butz R, Haakenson CM, Read RC, Sassoon CS, Eastridge CE, Berger R: Intrapleural tetracycline for the prevention of recurrent spontaneous pneumothorax. Results of a Department of Veterans Affairs cooperative study. JAMA. 1990;264(17):2224

17.10.4.3 TRAUMATIC PNEUMOTHORAX

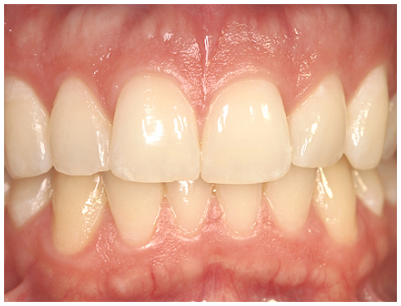
More than 50,000 trauma-related pneumothoraces occur annually in the US. Pneumothorax ranks second only to rib fractures as the most common manifestation of significant chest injury. Pneumothoraces are seen in as many as 40% to 50% of chest trauma victims[806]. Treatment will largely depend on the injuries sustained and any risk assessment post injury must take all factors into consideration, including the functional status of the person.

Reviewed 2015

17.11 K 00-99 DIGESTIVE SYSTEM

17.11.1 ORAL HEALTH

K 01-06	Oral health Acute pain from toothache. Recurrent mouth	T – If visual evidence of untreated dental defects or oral disease. P – If excess likelihood of dental emergency remains after treatment completed or person non compliant with dental recommendations	R – Limited to near coastal waters, if criteria for full fitness not met and type of operation will allow for access to dental care without safety-critical manning issues for the vessel	If teeth and gums (gums alone if edentulous and with well fitting dentures in good repair) appear to be good. No complex prosthesis or if dental check in last year with follow up completed and no problems since.



Periodontal, or gum disease is a common condition affecting the tissues that comprise the dental supporting structure. It is broadly classified as either gingivitis or periodontitis; these conditions are distinguished by the presence of alveolar bone involvement that occurs with periodontitis, and not with gingivitis[807]. It is important that the mouth is always inspected at the medical examination and that the seafarer's doctor is aware of what healthy gingival tissues should look like. Healthy gingival tissues are pink, stipled (like an orange peel) and firm. All persons with evidence of acute dental disease should be considered as temporarily unfit until they have been reviewed by a dentist and a treatment plan agreed. Only then can an individualized risk assessment be carried out taking into consideration the disease, necessary treatment plan including the need for follow up, current treatment and the likelihood of recurrence and/or complications.

Reviewed 2015

[806] Bridges KG, Welch G, Silver M et al: CT detection of occult pneumothorax in multiple trauma patients. J Emerg Med. 1993: 11: 179-186
[807] Williams RC. Periodontal disease. N Engl J Med. 1990;322(6):373.

17.11.2 PEPTIC ULCER

K 25-28	Peptic ulcer. Recurrence with pain, bleeding or perforation.	T – Until healing or cure by surgery or by control of helicobacter and on normal diet for 3 months. P – If ulcer persists despite surgery and medication	R – Consider case by case assessment for earlier return to near coastal duties	When cured and on normal diet for 3 months

Peptic ulcer disease (PUD) is a common problem although accurate data regarding the prevalence is difficult to obtain as symptoms are insensitive and non specific and diagnosis can only be confirmed on endoscopy. A recent large endoscopic study of a population sample from Sweden reported a prevalence of 4% (2% gastric, 2% duodenal ulcers)[808] although this has been found to be higher (17.2%) in China[809] and in Taiwan (9.4%)[810] The two common aetiological factors in PUD are Helicobacter Pylori (H Pylori) infection and the use of non steroidal anti inflammatory (NSAID) agents, including aspirin. The global trend has been for a decrease in the number of cases of PUD along with a decrease in the complication rate and this is likely to be linked to several factors[811]:

- The incidence of H. pylori in patients younger than 60 years is falling dramatically in developed countries due in part to improved hygiene and socioeconomic conditions starting after World War II. However, the prevalence remains high for older individuals and in certain predisposed subpopulations.
- NSAID use increases as a function of age and is an independent risk factor for ulcers. In addition, older subjects are more likely to develop complications from NSAID ulcers and to suffer increased morbidity and mortality from these complications because of comorbidities. Increased NSAID use, especially in the elderly, opposed the fall in H. pylori prevalence.
- Smoking clearly exacerbates at least H. pylori associated ulcer disease. The decline in smoking in younger individuals, particularly males, and increase in women, may be a factor in the declining male/female ratio of ulcer disease. Smoking does not appear to be a factor in the ulcer complications found in older women or in NSAID-related ulcers.

Evidence from the time before H pylori and proton pump inhibitors demonstrates the natural course of the disease. In particular one study followed patients for 12 months after documented healing of duodenal ulcers[812] and found that relapse occurred in 74% of cases:

- 33% had one recurrence
- 24% had two recurrences
- 17% experienced three or more recurrences

[808] Aro P, Storskrubb T, Ronkainen J, et al. Peptic ulcer disease in a general adult population: the Kalixanda study: a random population-based study. Am J Epidemiol. 2006;163:1025-1034.
[809] Li Z, Zou D, Ma X, Chen J, Shi X, Gong Y et al. Epidemiology of peptic ulcer disease: endoscopic results of the systematic investigation of gastrointestinal disease in China. Am J Gastroenterol. 2010;105(12):2570.
[810] Wang FW, Tu MS, Mar GY, Chuang HY, Yu HC, Cheng LC, Hsu PI. Prevalence and risk factors of asymptomatic peptic ulcer disease in Taiwan. World J Gastroenterol. 2011 Mar;17(9):1199-203.
[811] Sonnenberg A. Temporal trends and geographical variations of peptic ulcer disease. Aliment Pharmacol Ther. 1995;9 Suppl 2:3.
[812] Bardhan KD, Cole DS, Hawkins BW, Franks CR. Does treatment with cimetidine extended beyond initial healing of duodenal ulcer reduce the subsequent relapse rate? Br Med J (Clin Res Ed). 1982;284(6316):621.

However the treatment of H pylori in infected individuals has significantly reduced the incidence of recurrence. A meta anlaysis of 14 studies[813] demonstrated that duodenal ulcers recurred in fewer than 10% of patients successfully treated for H. pylori compared with 65- 95% of those who remained infected. As would be expected when the cause of the ulcer cannot be identified or removed (eg, continued NSAID use, or non-H. pylori, non-NSAID ulcers), recurrences are frequent[814]. Other ulcers cause complications or remain refractory despite therapy. The patient's prior ulcer history tends to predict future behavior; those with a history of complications have an increased risk of future complications. Ulcers that take longer to heal initially are more likely to recur rapidly and ulcers that have recurred frequently are likely to continue to do so, unless the underlying cause (eg H. pylori or NSAIDs) is removed. A long duration of symptoms prior to presentation is more likely to be associated with a poor response to medical therapy.

Acute complications can occur in patients with PUD of any aetiology. They include bleeding, perforation, penetration and gastric outlet obstruction. The frequency of complications varies geographically - comparison of figures from the US[815]and Nigeria[816] show almost a complete reversal in the frequency of complications:

	Haemorrhage	Perforation	Obstruction
US	73%	9%	3%
Nigerian	10%	30%	56%

It is suggested that some regional factors may account for these variations, in particular the varying rates of NSAID use, the incidence of H pylori infection and the distribution and extent of gastritis. Peptic ulcer bleeding is seen most commonly in older patients[817]. 60% of patients are above the age of 60 years and 20% are over the age of 80 years[818] [12]. This age distribution likely reflects increasing nonsteroidal anti-inflammatory drug (NSAID) use among older adults, combined with decreasing prevalence of H. pylori infection among younger patients. The use of NSAIDs is the most commonly identified risk factor for peptic ulcer bleeding and studies have found relative risks for bleeding ranging from 2.7 to 33.9[819]. Studies have also shown that the risk is drug-specific and dose-dependent: in a study of 2777 patients, the overall relative risk (RR) of bleeding associated with NSAID use was 5.3 (95% CI 4.5-6.2). However, the risk varied by drug and was lowest for aceclofenac (RR 3.1, 95% CI 2.3-4.2) and was highest for ketorolac (RR 14.4, 95% CI

[813] Hopkins RJ, Girardi LS, Turney EA. Relationship between Helicobacter pylori eradication and reduced duodenal and gastric ulcer recurrence: a review. Gastroenterology. 1996;110(4):1244.
[814] Wong GL, Wong VW, Chan Y, Ching JY, Au K et al. High incidence of mortality and recurrent bleeding in patients with Helicobacter pylori-negative idiopathic bleeding ulcers. Gastroenterology. 2009;137(2):525.
[815] Wang YR, Richter JE, Dempsey DT. Trends and outcomes of hospitalizations for peptic ulcer disease in the United States, 1993 to 2006. Ann Surg. 2010;251(1):51.
[816] Irabor DO. An audit of peptic ulcer surgery in Ibadan, Nigeria. West Afr J Med. 2005;24(3):242.
[817] Ohmann C, Imhof M, Ruppert C, Janzik U, Vogt C, Frieling T et al. Time-trends in the epidemiology of peptic ulcer bleeding. Scand J Gastroenterol. 2005;40(8):914.
[818] Sung JJ, Tsoi KK, Ma TK, Yung MY, Lau JY, Chiu PW. Causes of mortality in patients with peptic ulcer bleeding: a prospective cohort study of 10,428 cases. Am J Gastroenterol. 2010;105(1):84.
[819] Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC. Systematic review of the epidemiology of complicated peptic ulcer disease: incidence, recurrence, risk factors and mortality. Digestion. 2011;84(2):102.

5.2-39.9)[820]. The risk was higher in patients taking high-dose NSAIDs compared with those taking medium- or low-dose NSAIDs (RR 6.8, 95% CI 5.3-8.8 versus 4.0, 95% CI 3.2-5.0). There was also an increased risk of bleeding with aspirin use (RR 5.3) that again was dose-dependent (RR 7.5 with 500 mg per day versus 2.7 with 100 mg per day). The concurrent use of aspirin and NSAIDs conferred an even greater risk of bleeding than was seen with either agent alone (RR 12.7). Finally, the risk was highest in the first 30 days of NSAID use, with a RR of 7.6 (95% CI 6.0-9.5). The risk remained high between days 31 and 90 days (RR 7.3, 95% CI 4.0-13.2), but dropped after 91 days (RR 2.6, 95% CI 1.6-4.1). NSAIDs are also a risk factor for perforation[821]. Multiple studies have identified H. pylori infection as a risk factor for complicated PUD[822] and others have looked at the effect of H pylori infection alongside NSAID use with varying results. One meta analysis A meta-analysis that identified nine case-control studies that assessed the prevalence of H. pylori infection and NSAID use in patients with peptic ulcer bleeding suggested that the H. pylori infection combined with NSAID use increases the risk of bleeding above that seen with either risk factor alone[823]. The analysis found that individually, the odds ratios for bleeding peptic ulcers associated with H. pylori and NSAIDs use were 1.8 (95% CI 0.97-3.3) and 4.9 (95% CI 3.8-6.2), respectively, whereas the odds ratio increased to 6.1 when both H. pylori and NSAID were present (95% CI 3.9-9.6).

The initial treatment, investigation and follow up will depend on individual patient circumstances and local policy. The person should be declared temporarily unfit until a diagnosis is confirmed, treatment established with a documented plan for follow up and a resolution of symptoms seen. An individualized risk assessment should be carried out to include all factors and should be made in conjunction with a specialist assessment and report.

Reviewed 2015

17.11.3 HERNIAS

K 40-44	Hernias – Inguinal and Femoral. Likelihood of stragulation	T – Until surgically investigated to confirm no likelihood of strangulation and, if required, treated.	R – Untreated. Consider case by case assessment for near coastal waters	When satisfactorily treated or exceptionally when surgeon reports there is no likelihood of strangulation
	Hernias – umbilical, ventral Instability of abdominal wall on bending and lifting	Case-by-case assessment depending on severity of symptoms or impairment. Consider implications of regular	Case-by-case assessment depending on severity of symptoms or impairment. Consider implications of regular	Case-by-case assessment depending on severity of symptoms or impairment. Consider implications of regular

[820] Lanas A, García-Rodríguez LA, Arroyo MT, Gomollón F, Feu F et al. Risk of upper gastrointestinal ulcer bleeding associated with selective cyclo-oxygenase-2 inhibitors, traditional non-aspirin non-steroidal anti-inflammatory drugs, aspirin and combinations. Gut. 2006;55(12):1731.
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[822] Labenz J, Peitz U, Köhl H, Kaiser J, Malfertheiner P, Hackelsberger A, Börsch G. Helicobacter pylori increases the risk of peptic ulcer bleeding: a case-control study. Ital J Gastroenterol Hepatol. 1999;31(2):110.
[823] Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet. 2002;359(9300):14.

		heavy whole-body physical effort	heavy whole-body physical effort	heavy whole-body physical effort
	Hernias – diaphragmatic (hiatus) Reflux of stomach contents and acid causing heartburn, pain, triggered by bending and lifting	Case-by-case assessment based on severity of symptoms when lying down, sleeping, bending and lifting, and the impairment caused thereby.	Case-by-case assessment based on severity of symptoms when lying down, sleeping, bending and lifting, and the impairment caused thereby.	Case-by-case assessment based on severity of symptoms when lying down, sleeping, bending and lifting, and the impairment caused thereby.

17.11.3.1 INGUINAL AND FEMORAL HERNIAS

Groin hernias (inguinal and femoral) are estimated to have a prevalence in the US of 5- 10% and they are more common in men and in whites than non whites[824]. Men are eight times more likely to develop a hernia and 20 times more likely to need a hernia repair compared with women[825] . The lifetime risk of developing a groin hernia is about 25% in men but less than 5% in women and women tend to present later in life. The median age at presentation was 60 - 79 years of age for women compared with 50 to 69 years of age for men[826].

Groin hernias are classified anatomically as inguinal (indirect or direct) or femoral and in clinical reviews:

- Approximately 96% of groin hernias are inguinal and 4% are femoral[827].
- Indirect inguinal hernia is the most common groin hernia in both sexes. In the Swedish registry, indirect inguinal hernia accounted for 49% of repairs in women and 54% in men[828].
- Direct inguinal hernia accounts for 30 – 40% of groin hernias in men, but about 14 – 21% of groin hernias in women[829].
- Femoral hernias account for <10% of all groin hernias and only 2 - 4 % of all groin hernia repairs. Femoral hernias represent 20 – 31% of repairs in women[830][831] compared with only 1% in men[832]. Femoral hernias occur later in life than inguinal hernias - over the age of 70, femoral hernias represent 52% of repairs in women and 7% percent of repairs in men[833].

[824] McIntosh A, Hutchinson A, Roberts A, Withers H. Evidence-based management of groin hernia in primary care--a systematic review. Fam Pract. 2000;17(5):442.
[825] Bendavid, R. Femoral hernias in females. Facts, figures and fallacies. In: Abdominal wall hernias, Springer, New York 2001. p.639.
[826] Kark AE, Kurzer M. Groin hernias in women. Hernia. 2008 Jun;12(3):267-70. Epub 2008 Jan 24.
[827] Rutkow IM, Robbins AW. Demographic, classificatory, and socioeconomic aspects of hernia repair in the United States. Surg Clin North Am. 1993;73(3):413.
[828] Koch A, Edwards A, Haapaniemi S, Nordin P, Kald A. Prospective evaluation of 6895 groin hernia repairs in women. Br J Surg. 2005;92(12):1553.
[829] Koch A, Edwards A, Haapaniemi S, Nordin P, Kald A. Prospective evaluation of 6895 groin hernia repairs in women. Br J Surg. 2005;92(12):1553.
[830] Kark AE, Kurzer M. Groin hernias in women. Hernia. 2008 Jun;12(3):267-70. Epub 2008 Jan 24
[831] Dahlstrand U, Wollert S, Nordin P, Sandblom G, Gunnarsson U. Emergency femoral hernia repair: a study based on a national register. Ann Surg. 2009;249(4):672.
[832] Dahlstrand U, Wollert S, Nordin P, Sandblom G, Gunnarsson U. Emergency femoral hernia repair: a study based on a national register. Ann Surg. 2009;249(4):672.
[833] Arenal JJ, Rodríguez-Vielba P, Gallo E, Tinoco C. Hernias of the abdominal wall in patients over the age of 70 years. Eur J Surg. 2002;168(8-9):460-3.

Risk factors for hernia development include the following[834][835][836][837][838]:

- History of hernia or prior hernia repair (including childhood)
- Older age
- Male sex
- Caucasian race
- Chronic cough
- Chronic constipation
- Abdominal wall injury
- Smoking
- Family history of hernia

Groin hernias can present with a range of symptoms from an incidental finding on physical examination, to complaints of groin or pelvic discomfort or pain particularly when intraabdominal pressure is increased, such as with heavy lifting, straining, or prolonged standing, to an emergency presentation secondary to incarceration and strangulation.

The incidence of incarceration and strangulation is low, estimated at 0.3 – 3% per year. Risk factors include advancing age, femoral hernia and recurrent hernia[839][840][841]. Although all groin hernias can strangulate, femoral hernias appear more predisposed to do so[842].

The definitive treatment of any groin hernia is surgical repair[843]. Urgent or emergent repair is required for patients who develop bowel complications but for patients without complications the timing of surgery and the optimal technique remains controversial. Surgical repair has minimal short term morbidity and the risk of recurrence is estimated at 1 – 2%. Mortality within 30 days of groin hernia surgery for both sexes is 0.1% in elective settings, but increases significantly when emergency operation is needed, ranging from 2.8 - 3.1%[844][845], and are even higher when bowel resection is needed[846].

[834] McIntosh A, Hutchinson A, Roberts A, Withers H. Evidence-based management of groin hernia in primary care--a systematic review. Fam Pract. 2000;17(5):442.
[835] Rosemar A, Angerås U, Rosengren A, Nordin P. Effect of body mass index on groin hernia surgery. Ann Surg. 2010 Aug;252(2):397-401.
[836] Ruhl CE, Everhart JE. Risk factors for inguinal hernia among adults in the US population. Am J Epidemiol. 2007 May;165(10):1154-61. Epub 2007 Mar 20.
[837] Sorensen LT, Friis E, Jorgensen T, Vennits B, Andersen BR, Rasmussen GI, Kjaergaard J. Smoking is a risk factor for recurrence of groin hernia. World J Surg. 2002 Apr;26(4):397-400. Epub 2002 Jan 2.
[838] Akbulut S, Cakabay B, Sezgin A. A familial tendency for developing inguinal hernias: study of a single family. Hernia. 2010 Aug;14(4):431-4. Epub 2009 Aug 29.
[839] Gallegos NC, Dawson J, Jarvis M, Hobsley M. Risk of strangulation in groin hernias. Br J Surg. 1991;78(10):1171.
[840] Fitzgibbons RJ Jr, Giobbie-Hurder A, Gibbs JO, Dunlop DD, Reda DJ et al. Watchful waiting vs repair of inguinal hernia in minimally symptomatic men: a randomized clinical trial. JAMA. 2006;295(3):285.
[841] Abi-Haidar Y, Sanchez V, Itani KM. Risk factors and outcomes of acute versus elective groin hernia surgery. J Am Coll Surg. 2011;213(3):363.
[842] McIntosh A, Hutchinson A, Roberts A, Withers H. Evidence-based management of groin hernia in primary care--a systematic review. Fam Pract. 2000;17(5):442.
[843] Rosenberg J, Bisgaard T, Kehlet H, Wara P, Asmussen T, Juul P, Strand L, Andersen FH, Bay-Nielsen M, Danish Hernia Database. Danish Hernia Database recommendations for the management of inguinal and femoral hernia in adults. Dan Med Bull. 2011 Feb;58(2):C4243.
[844] Koch A, Edwards A, Haapaniemi S, Nordin P, Kald A. Prospective evaluation of 6895 groin hernia repairs in women. Br J Surg. 2005;92(12):1553.
[845] Arenal JJ, Rodríguez-Vielba P, Gallo E, Tinoco C. Hernias of the abdominal wall in patients over the age of 70 years. Eur J Surg. 2002;168(8-9):460-3.
[846] Nilsson H, Stylianidis G, Haapamäki M, Nilsson E, Nordin P. Mortality after groin hernia surgery. Ann Surg. 2007 Apr;245(4):656-60.

These excellent surgical outcomes, even among elderly individuals (particularly if local anesthesia can be used), have led to recommendations to offer surgical repair to patients with inguinal or femoral hernia who are symptomatic. However it may be reasonable to take a more conservative approach in men who have minimal or no symptoms[847]. Because women are at higher risk for hernia complications, in general, they should be offered repair when diagnosed.

The largest study evaluating watchful waiting (the WW trial) randomly assigned 720 men with an inguinal hernia to watchful waiting or open surgical repair (tension-free hernia repair)[848][849]. The men, who were mostly between the ages of 40 and 65, were asymptomatic or had only minimal symptoms, and the hernia remained easily reduced within 6 weeks of initial screening. The following results were noted:

- At two years follow-up, there were no differences between the groups for the primary end points of pain sufficient to limit activity, or change in physical health scores.
- 23% of patients in the watchful waiting group had surgery within two years and 31% at four years.
- With longer-term follow-up (maximum 11.5 years), the estimated cumulative crossover rates using Kaplan-Meier analysis was 68%. Crossover rates were higher for men older than 65 years compared with younger men (79 vs.62%). The most common reason for crossover was pain (54.%) not acute complications.
- Significant hernia complications did occur in patients being watched, but were rare with only 0.0018 hernia-related adverse events per patient-year.
- The rate of postoperative complications was not significantly different between patients who were assigned to and received surgical repair compared with those who were assigned to watchful waiting and then crossed over to receive surgical repair (21.7 vs. 27.9%). At the time of maximum follow-up, a total of three patients required an emergency operation, but there was no mortality.

These findings suggest that a strategy of watchful waiting rather than elective repair is an option for white, middle-aged male patients with asymptomatic or minimally symptomatic inguinal hernia, provided the patient is aware of the risk of potential hernia complications and understands the need for prompt medical attention if symptoms develop. However, there are insufficient data regarding the risk of watchful waiting in older patients who are at the greatest risk of strangulation, and the risk associated with emergency hernia repair [850]. In addition, it is not clear whether the above studies are generalizable to young individuals, women, other ethnic groups, or other types of hernia. In persons the risk of surgery must be weighed against the risk of

[847] Rosenberg J, Bisgaard T, Kehlet H, Wara P, Asmussen T, Juul P, Strand L, Andersen FH, Bay-Nielsen M, Danish Hernia Database. Danish Hernia Database recommendations for the management of inguinal and femoral hernia in adults. Dan Med Bull. 2011 Feb;58(2):C4243.

[848] Fitzgibbons RJ Jr, Giobbie-Hurder A, Gibbs JO, Dunlop DD, Reda DJ et al. Watchful waiting vs repair of inguinal hernia in minimally symptomatic men: a randomized clinical trial. JAMA. 2006;295(3):285

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[850] Hernández-Irizarry R, Zendejas B, Ramirez T, Moreno M, Ali SM, Lohse CM, Farley DR. Trends in emergent inguinal hernia surgery in Olmsted County, MN: a population-based study. Hernia. 2012 Aug;16(4):397-403. Epub 2012 Jun 14.

significant symptoms or complications developing during the validity period and the need for regular (6 monthly or annual follow up) if watchful waiting is the treatment option of choice.

Reviewed 2015

17.11.4 CROHNS DISEASE AND ULCERATIVE COLITIS

K 50, 51, 57, 58, 90	Non-infectious enteritis, colitis, Crohn's disease, diverticulitis, etc. Impairment and pain.	T – Until investigated and treated P – If severe or recurrent	R – Does not meet the requirements for unlimited medical certificate but rapidly developing recurrence unlikely. Near-coastal waters.	Case-by-case specialist assessment. Fully controlled with low likelihood of recurrence.

17.11.4.1 CROHNS DISEASE

Crohns disease is a disorder of uncertain aetiology that is characterized by transmural inflammation of the gastrointestinal tract. Crohns disease may involve the entire gastrointestinal tract from mouth to peri anal area:

- Approximately 80% have small bowel involvement, usually in the distal ileum with one third having ilieitis alone
- Approximately 50% have ileocolitis
- Approximately 20% have disease limited to the colon although in 50% of these the rectum will be spared
- Approximately one third of patients have peri anal disease
- Approximately 5 – 15% have predominant involvement of the moth or gastroduodenal area.

Systemic symptoms and a variety of extra intestinal manifestations can also occur and initial assessment and ongoing management of the patient with Crohns disease should be done by an appropriate specialist team.

The typical course in a patient with Crohns Disease involving the small and/or large intestine is one of intermittent exacerbation followed by periods of remission. Approximately 10 – 20% of patients experience a prolonged remission following initial presentation[851]. In another study 53% of patients developed a stricture or penetrating disease at 10 years[852]. Predictors of a severe course include age less than 40, the presence of perianal or rectal disease, smoking, low education level, and initial requirement for glucocorticoids[853] [854].

[851] Farmer RG, Whelan G, Fazio VW. Long-term follow-up of patients with Crohn's disease. Relationship between the clinical pattern and prognosis. Gastroenterology. 1985;88(6):1818.
[852] Solberg IC, Vatn MH, Høie O, Stray N, Sauar J, Jahnsen J, Moum B, Lygren I, IBSEN Study Group. Clinical course in Crohn's disease: results of a Norwegian population-based ten-year follow-up study. Clin Gastroenterol Hepatol. 2007;5(12):1430.

[853] Beaugerie L, Seksik P, Nion-Larmurier I, Gendre JP, Cosnes J. Predictors of Crohn's disease. Gastroenterology. 2006;130(3):650.
[854] Cosnes J, Bourrier A, Nion-Larmurier I, Sokol H, Beaugerie L, Seksik P. Factors affecting outcomes in Crohn's disease over 15 years. Gut. 2012 Aug;61(8):1140-5. Epub 2012 Mar 2.

In a study of 306 patients[855](45% with ileal disease, 32% with colonic disease,19% with ileocolonic disease), 81% had nonstricturing/nonpenetrating disease at baseline. The cumulative risk of developing either complication at 90 days was 19%, at 1 year was 22%, at 5 years was 34%, and at 20 years was 51%. Factors associated with complications included disease extent at baseline. Patients with ileal disease had a ninefold increased risk compared with those with colonic disease, and patients with ileocolonic disease had a sixfold increased risk. In addition, use of mesalamine or sulfasalazine in the first 90 days increased the risk of complications twofold. Finally, perianal disease increased the risk of complications, though the results were of borderline significance.

The following observations were made from a systematic review of the literature and guideline published by the American College of Gastroenterology[856]:

- Patients who are in remission for one year have an 80% chance of remaining in remission for the subsequent years.
- Patients who have active disease within the past year have a 70% chance of remaining active in the forthcoming year and a 50% chance of being in remission within the ensuing three years.
- Overall, 13% of patients will have a relapse-free course, while 20% have annual relapses, and 67% have a combination of years in relapse and years in remission within the first eight years after initial diagnosis.
- Fewer than 5% will have a continuous course of active disease.
- Recurrence of perianal fistulas after medical or surgical therapy is common (59 to 82%).

Many patients with CD ultimately require surgical intervention with intestinal resection because of intractability of symptoms, obstruction, or perforation. Some patients tend to follow a pattern of either recurrent obstruction or recurrent perforations; the latter group has been reported to have a more aggressive form of the disease, leading to earlier postoperative recurrence and the need for more surgery.

17.11.4.2 ULCERATIVE COLITIS

Ulcerative colitis is characterized by recurring episodes of inflammation limited to the mucosal layer of the colon. It commonly involves the rectum and may extend in a proximal and continuous fashion to other parts of the colon. Patients usually present with diarrhea which may be associated with blood and other abdominal symptoms. The severity of the disease ranges from mild to severe based on the number of stools passed per day and other abdominal symptoms[857]:

[855] Thia KT, Sandborn WJ, Harmsen WS, Zinsmeister AR, Loftus EV Jr. Risk factors associated with progression to intestinal complications of Crohn's disease in a population-based cohort. Gastroenterology. 2010;139(4):1147.
[856] Lichtenstein GR, Hanauer SB, Sandborn WJ, Practice Parameters Committee of American College of Gastroenterology. Management of Crohn's disease in adults. Am J Gastroenterol. 2009;104(2):465.
[857] Silverberg MS, Satsangi J, Ahmad T, Arnott ID, Bernstein CN, Brant SR, Caprilli R, Colombel JF, Gasche C et al. Toward an integrated clinical, molecular and serological classification of inflammatory bowel disease: report of a Working Party of the 2005 Montreal World Congress of Gastroenterology. Can J Gastroenterol. 2005;19 Suppl A:5A.

- Mild: four or fewer stools per day, with or without blood and only mild abdominal symptoms. No systemic involvement and a normal ESR.
- Moderate: > 4 loose, bloody stools per day with mild anaemia (not requiring transfusion) and abdominal pain that is not severe. Minimal systemic toxicity including a low grade fever but adequate nutrition is maintained and no significant weight loss.
- Severe: > 6loose, bloody stools per day with severy cramps and evidence of systemic toxicity and possible rapid weight loss.

Most patients present with a mild attack at presentation, 27% have moderate disease and just 1% have severe disease[858]. Acute complications of Ulcerative Colitis include severe bleeding (in up to 10% of patients), fulminant colitis and toxic megacolon and perforation (most commonly associated with toxin megacolon). Extraintestinal manifestations can also occur and again care should be managed by an appropriate specialist team.

The attacks of bloody diarrhea characteristic of the disease tend to last for weeks or months. With treatment the disease typically consists of intermittent exacerbations followed long periods of complete symptomatic remission. However a small percentage have ongoing symptoms[859]. Overall patients who present with proctitis have a more benign course and frequently respond to topical therapy, whereas those who present with more extensive disease require systemic therapy and have a higher risk of colectomy.

Approximately 67% of patients have at least one relapse 10 years following the diagnosis[860] and the risk of relapse depends on the age at initial diagnosis[861], disease flare within two years of the diagnosis, the presence of fever or weight loss at diagnosis, and active disease in the preceding year increase the risk of subsequent relapse[862].

Approximately 20 – 30% of patients with Ulcerative Colitis will require colectomy for acute complications or for medically intractable disease. The likelihood and timing of colectomy depends on the extent of the disease and severity at presentation. As an example, for patients with pancolitis, the rate of colectomy is approximately 19% after 10 years, whilst 5% of patients who present with proctitis alone have undergone colectomy after 10 years[863].

[858] Langholz E, Munkholm P, Nielsen OH, Kreiner S, Binder V. Incidence and prevalence of ulcerative colitis in Copenhagen county from 1962 to 1987. Scand J Gastroenterol. 1991 Dec;26(12):1247-56.
[859] Solberg IC, Lygren I, Jahnsen J, Aadland E, Høie O, Cvancarova M, Bernklev T, Henriksen M, Sauar J, Vatn MH, Moum B, IBSEN Study Group. Solberg IC, Lygren I, Jahnsen J, Aadland E, Høie O, Cvancarova M, Bernklev T, Henriksen M, Sauar J, Vatn MH, Moum B, IBSEN Study Group. Scand J Gastroenterol. 2009;44(4):431-40.
[860] Höie O, Wolters F, Riis L, Aamodt G, Solberg C, Bernklev T, Odes S, Mouzas IA, Beltrami M, Langholz E, Stockbrügger R, Vatn M, Moum B, European Collaborative Study Group of Inflammatory Bowel Disease (EC-IBD). Ulcerative colitis: patient characteristics may predict 10-yr disease recurrence in a European-wide population-based cohort. Am J Gastroenterol. 2007 Aug;102(8):1692-701. Epub 2007 Jun 6.
[861] Ha CY, Newberry RD, Stone CD, Ciorba MA. Patients with late-adult-onset ulcerative colitis have better outcomes than those with early onset disease. Clin Gastroenterol Hepatol. 2010;8(8):682.
[862] Langholz E, Munkholm P, Davidsen M, Binder V. Course of ulcerative colitis: analysis of changes in disease activity over years. Gastroenterology. 1994;107(1):3.
[863] Solberg IC, Lygren I, Jahnsen J, Aadland E, Høie O, Cvancarova M, Bernklev T, Henriksen M, Sauar J, Vatn MH, Moum B, IBSEN Study Group. Clinical course during the first 10 years of ulcerative colitis: results from a population-based inception cohort (IBSEN Study). Scand J Gastroenterol. 2009;44(4):431-40.

Assessment of the person with Crohns disease or Ulcerative Colitis must include a specialist report including current disease status, current requirement for medication and any significant side effects that may impair the person's ability to perform his routine or emergency duties, the risk of recurrence of disease or a complication, the need for follow up and any specific dietary requirements the person may have. A thorough history and examination must be undertaken by the seafarer's doctor and an individualized risk assessment carried out.

17.11.4.3 DIVERTICULAR DISEASE

Colonic diverticulosis refers to herniation of mucosa and submucosa through the muscular layer of the colonic wall and may be the result of colonic smooth muscle over-activity. Diverticular disease may be defined as any clinical state caused by symptoms pertaining to colonic diverticula and includes a wide-ranging spectrum from asymptomatic to severe and complicated disease. Diverticulitis indicates inflammation of a diverticulum or diverticula and may be caused by infection. Other complications of diverticular disease include segmental colitis, lower gastrointestinal bleeding, infection, abscess, perforation, peritonitis, and fistula formation.

The exact incidence of diverticular disease is difficult to estimate as many patients are asymptomatic and many of the studies are retrospective. It is known that the incidence increases with age (<10% under 40 years of age, approximately 50% at 50 years of age and incresaing to 50 – 66% at 80 years of age in developed countries. It is lower in vegetarians and right sided disease (associated with meat consumption) is more common in Asia[864]. One study has reported an overall prevalence of 12 – 49%[865] and whilst there is no overall sex difference in the prevalence, in older adults it is more common in females[866].

The aetiology of diverticular disease is multi factorial with both genetic and environmental factors, especially a low dietary fibre intake (deemed as the most predominant factor in Western populations)[867]. Other factors include[868]:

- Decreased physical activity
- Obesity
- Increased red meat consumption
- Excessive alcohol and caffeine intake
- Steroids
- NSAIDS

[864] Lin OS, Soon MS, Wu SS, et al. Dietary habits and right-sided colonic diverticulosis. Dis Colon Rectum. 2000;43:1412-1418.
[865] Delvaux M. Diverticular disease of colon in Europe: epidemiology, impact on citizen health and prevention. Aliment Pharmacol Ther. 2003;18(suppl 3):71-74.
[866] Parks TG. Natural history of diverticular disease of the colon. Clin Gastroentrol. 1975;4:53-69.
[867] Painter NS, Burkitt DP. Diverticular disease of the colon: a deficiency disease of Western civilization. Br Med J. 1971;2:450-454.
[868] Andersen JC, Bundgaard L, Elbrønd H, et al. Danish national guidelines for treatment of diverticular disease. Dan Med J. 2012;59:C4453.

The management of diverticular disease depends on the severity of symptoms with asymptomatic disease requiring no active treatment, just simple dietary advice[869]. Mild symptoms may again only require dietary modification and improved hydration[870] with oral antibiotics if necessary. More severe disease may require admission to hospital for further investigation of complications and appropriate treatment including surgery if necessary[871].

In cases of simple disease most patients recover with medical treatment and do not require surgical intervention. However in one third of patients there will be a relapse of symptoms within 5 years[872]. Recurrent disease is associated with high mortality and the response to therapy is less favourable. Approximately 25% of all patients following surgical treatment will continue to remain symptomatic and require ongoing therapy and follow up[873]. Complications can include abscess, perforation and strictures.

The assessment of a person with diverticular disease should include specialist input with reference to the severity of the disease, treatment required, ongoing symptoms, the need for ongoing follow up and the risk of relapse of the disease in the setting of limited access to medical care. An individualized risk assessment should then be performed.

Reviewed 2016

17.11.5 ANAL CONDITIONS

K 60, I 84	Anal conditions: Piles (haemorrhoids), fissures, fistulae Likelihood of episode causing pain and limiting activity.	T – If piles prolapsed, bleeding repeatedly or causing symptoms. If fissure or fistula painful, infected, bleeding repeatedly or causing faecal incontinence. P – Consider if not treatable or recurrent	Case-by-case assessment of untreated cases for near coastal duties	When satisfactorily treated

17.11.5.1 HAEMORRHOIDS

Haemorrhoidal cushions are normal anatomical structures located within the anal canal, usually occupying the left lateral, right anterior and posterior positions. As they enlarge, they can protrude outside the anal canal causing symptoms. The true prevalence of haemorrhoids is difficult to assess due to different levels of access to healthcare and appropriate investigation. In a large cross-sectional US study the prevalence of self reported haemorrhoids was 4.4% , they

[869] Marlett JA, McBurney MI, Slavin JL; American Dietetic Association. Position of the American Dietetic Association: health implications of dietary fiber. J Am Diet Assoc. 2002;102:993-1000.
[870] Unlu C, Daniels L, Vrouenraets BC, et al. A systematic review of high-fibre dietary therapy in diverticular disease. Int J Colorectal Dis. 2012;27:419-427.
[871] Andersen JC, Bundgaard L, Elbrønd H, et al. Danish national guidelines for treatment of diverticular disease. Dan Med J. 2012;59:C4453.
[872] Young-Fadok TM, Roberts PL, Spencer MP, et al. Colonic diverticular disease. Curr Prob Surg. 2000;37:459-514.
[873] Munson KD, Hensien MA, Jacob LN, et al. Diverticulitis: a comprehensive follow-up, Dis Colon Rectum. 1996;39:318-322.

are more common in white patients than black, there is no difference in prevalence between men and women and presentation peaks between the ages of 45 – 65 years.

Approximately 40% of individuals are asymptomatic , patients with symptoms usually complain of passing blood per rectum, pain associated with a thrombosed haemorrhoid, perianal pruritis or faecal soilage. The majority of patients with symptomatic haemorrhoids can be managed successfully with conservative measures although surgery will provide immediate pain relief for thrombosed haemorrhoids. Elective surgery should be reserved for patients with ongoing symptoms despite appropriate medical treatment and lifestyle changes.

The risk assessment of a person with haemorrhoids must include the effect of any underlying risk factors (eg diarrheoa, pregnancy, pelvic tumours, prolonged sitting, straining and chronic constipation) on the person's ability to perform his/her regular and emergency duties, current symptoms and management and the likely need for follow up or surgery during the validity period.

17.11.5.2 ANAL FISSURE

An anal fissure is a split in the skin of the distal anal canal characterised by pain on defecation and rectal bleeding. An acute fissure heals within six weeks whilst a chronic fissure fails to heal with conservative management and requires a more aggressive, surgical approach[874] [875]. A fissure is primary or secondary dependent on it's aetiology – primary are caused by local trauma eg passage of hard stool, prolonged diarrhoea, vaginal delivery or anal sex, whilst secondary fissures are a complication of an underlying disease process eg inflammatory bowel disease, granulomatous disease, communicable diseases or malignancy.[876]

It is a common condition in young to middle-aged adults and may occur in 1 in 350 people in the European Union[877]. It is equally common in men and women and often affects adults aged 15 – 40 years although may be seen in older adults and in children due to poor toileting[878].

Most primary anal fissures respond to conservative management and are self limiting with healing achieved in 6 – 8 weeks. A further 20% will heal after a course of topical treatment. However some may recur and around 30% will require a surgical option[879].

A person with symptoms of an acute anal fissure should probably be declared temporarily unfit until symptoms have resolved although a restricted certificate may be appropriate. If symptoms persist a specialist report should be sought outlining the treatment plan and likely need for

[874] Zaghiyan KN, Fleshner P. Anal fissure. Clin Colon Rectal Surg. 2011 Mar;24(1):22-30.
[875] Madalinski MH. Identifying the best therapy for chronic anal fissure. World J Gastrointest Pharmacol Ther. 2011 Apr;2(2):9-16.
[876] Oh C, Divino CM, Steinhagen RM. Anal fissure. 20-year experience. Dis Colon Rectum. 1995 Apr;38(4):378-82.
[877] Ayatunde AA, Debrah SA. Current concepts in anal fissures. World J Surgery. 2006;30:2246-60.
[878] Simpson J, Lund JN, Thompson RJ, et al. The use of glyceryl trinitrate (GTN) in the treatment of chronic anal fissure in children. Med Sci Monit. 2003;9:PI123-PI126.
[879] Collins EE, Lund JN. A review of chronic anal fissure management. Tech Coloproctol. 2007;11:209-223.

additional treatment and follow up. The impact of any underlying disease must also be taken into consideration.

17.11.5.3 PERIANAL/ANORECTAL ABSCESS

A perianal/anorectal abscess is an infection of the soft tissues around the anus and is estimated to affect 0.18% of the general population[880]. They occur two to three times more commonly in men and the mean age for presentation is 40 years (range 20 – 60 years)[881] [882]. Adequate drainage of the abscess should result in the immediate resolution of symptoms although the person should not be declared fit until the surgical wound has healed. Recurrence of the abscess occurs in up to 2% of patients unless there is an associated anal fissure, as is the case in 37%[883] [884]. A specialist opinion should be sought with reference to any underlying pathology and therefore the risk of the need for further treatment during the certificate validity period.

17.11.5.4 ANAL FISTULAE

An anorectal fistula is the chronic manifestation of the acute perirectal process that forms an anal abscess. When the abscess ruptures or is drained, an epithelialized track can form that connects the abscess in the anus or rectum with the perirectal skin[885]. The incidence of an anal fistula developing from an anal abscess ranges from 26 – 38%[886] [887]. The mean age for presentation of anal abscess and fistula disease is 40 years (range 20 to 60 years)[888]. Adult males are twice as likely to develop an abscess and/or fistula compared with women[889]. The most common aetiology is a perianal abscess although other causes include but are not limited to Crohn's disease, lymphogranuloma venereum and rectal foreign bodies. Patients with an anal fistula experience symptoms that are not likely to be compatible with life at sea and their ability to perform their routine and emergency duties. Surgery is the main stay of treatment and it is likely that this should be performed before a certificate is granted. However a restricted and time limited certificate may be appropriate whilst waiting for surgery and this must be assessed on a case by case basis.

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[880] Gilliland R, Wexner SD. Complicated anorectal sepsis. Surg Clin North Am. 1997;77:115-153.
[881] Abcarian H. Anorectal infection: Abscess-fistula. Clinics in colon and rectal surgery 2011. 24:14.
[882] Nelson RL, Abcarian H, Davis FG, Persky V. Prevalence of benign anorectal disease in a randomly selected population. Dis Colon Rectum. 1995;38(4):341.
[883] Hamalainen KP, Sainio AP. Incidence of fistulas after drainage of acute anorectal abscesses. Dis Colon Rectum. 1998;41:1357-1361.
[884] Vasilevsky CA, Gordon PH. The incidence of recurrent abscesses or fistula-in-ano following anorectal suppuration. Dis Colon Rectum. 1984;27:126-130.
[885] Whiteford MH, Kilkenny J 3rd, Hyman N, Buie WD, Cohen J, Orsay C, Dunn G, Perry WB, Ellis CN, Rakinic J, Gregorcyk S, Shellito P, Nelson R, Tjandra JJ, Newstead G, Standards Practice Task Force, American Society of Colon and Rectal Surgeons. Practice parameters for the treatment of perianal abscess and fistula-in-ano (revised). Dis Colon Rectum. 2005;48(7):1337.
886 Scoma JA, Salvati EP, Rubin RJ. Incidence of fistulas subsequent to anal abscesses. Dis Colon Rectum. 1974;17(3):357.
887 Vasilevsky CA, Gordon PH. The incidence of recurrent abscesses or fistula-in-ano following anorectal suppuration. Dis Colon Rectum. 1984;27(2):126.
[888] Nelson RL, Abcarian H, Davis FG, Persky V. Prevalence of benign anorectal disease in a randomly selected population. Dis Colon Rectum. 1995;38(4):341.
[889] Sainio P. Fistula-in-ano in a defined population. Incidence and epidemiological aspects. Ann Chir Gynaecol. 1984;73(4):219.

Reviewed 2015

17.11.6 CIRRHOSIS OF THE LIVER

K 70, 72	Cirrhosis of the liver Liver failure. Bleeding oesophageal varices.	T – Until satisfactorily investigated P – If severe or complicated with ascites or oesophageal varices	R, L – Case-by-case specialist assessment	Not applicable

17.11.6.1 CIRRHOSIS OF THE LIVER

Cirrhosis of the liver is a diffuse pathological process characterised by fibrosis and conversion of the normal liver architecture to strucuturally abnormal nodules. It can arise from a variety of causes but patients may be asymptomatic for many years and hence its prevalence and incidence in the general population is difficult to ascertain with any accuracy. In 2010 cirrhosis accounted for 49,500 deaths and was the eighth leading cause of death in the US[890]. The major complications of liver cirrhosis include the following and once these are determind to be present the disease is said to be decompensated.

- Variceal hemorrhage
- Ascites
- Spontaneous bacterial peritonitis
- Hepatic encephalopathy
- Hepatocellular carcinoma
- Hepatorenal syndrome
- Hepatopulmonary syndrome

Other complications include portal vein thrombosis and cardiomyopathy although alone these do not indicate decompensated cirrhosis.

The 10 year survival rate in patients with compensated cirrhosis is approximately 90% and the likelihood of transitioning to decompensated cirrhosis within 10 years is 50%[891]. However the course of the disease will depend on many factors including but not limited to bleeding, infection, alcohol intake, medications, dehydration, constipation and obesity[892] [893] [894] [895]. The natural history of the underlying disease process must also be taken into consideration as must the likely progression of any one of the complications itself. The median survival time of a patient with decompensated cirrhosis is approximately 2 years. Using scores such as the Child-

[890] Murray CJ, Atkinson C, Bhalla K, Birbeck G, Burstein R, Chou D et al. The state of US health, 1990-2010: burden of diseases, injuries, and risk factors. JAMA. 2013;310(6):591

[891] D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol. 2006;44:217-231.

[892] Liao WC, Hou MC, Chang CJ, Lee FY, Lin HC, Lee SD. Potential precipitating factors of esophageal variceal bleeding: a case-control study. Am J Gastroenterol. 2011;106(1):96.

[893] Mumtaz K, Ahmed US, Abid S, Baig N, Hamid S, Jafri W. Precipitating factors and the outcome of hepatic encephalopathy in liver cirrhosis. J Coll Physicians Surg Pak. 2010;20(8):514.

[894] Sundaram V, Shaikh OS. Hepatic encephalopathy: pathophysiology and emerging therapies. Med Clin North Am. 2009;93(4):819.

[895] Berzigotti A, Garcia-Tsao G, Bosch J, Grace ND, Burroughs AK. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology. 2011;54(2):555.

Pugh-Turcotte and the Model for End-Stage Liver Disease score, four clinical stages of cirrhosis have been identified and each is associated with a different prognosis:

Stage 1 - Patients without gastro-oesophageal varices or ascites have a mortality of approximately 1% per year.

Stage 2 - Patients with gastro-oesophageal varices (but no bleeding) and no ascites have a mortality of approximately 4% per year.

Stage 3 - Patients with ascites with or without gastro-oesophageal varices (but no bleeding) have a mortality of approximately 20% per year.

Stage 4 - Patients with GI bleeding due to portal hypertension with or without ascites have a 1-year mortality of 57%.

It is important to note that these are only figures looking at actual mortality and the risk of significant medical events over the certificate validity period must also be taken into consideration. Patients with cirrhosis should be monitored every 6 – 12 months with appropriate investigations and any person with suspected or diagnosed liver disease seeking a fitness certificate must have a specialist report outlining the underlying disease and it's likely progression, the need for follow up and the presence or absence of any liver impairment, cirrhosis and complications including a statistical estimate of the risk of developing any of the above. Only then can an individualised risk assessment be made.

OESOPHAGEAL VARICES

Oesophageal varices are dilated collateral blood vessels that develop as a complication of portal hypertension, usually in the setting of cirrhosis. In the US and Europe the major cause is alcoholic liver disease although world wide hepatitis B and C are the major causes of cirrhosis.

Gastro oesophageal varices are present in almost 50% of patients at the time of the diagnosis of cirrhosis. The 1 year incidence of bleeding is 5% with small varices to 15% with large varices[896]. Development and growth of varices each occur at approximately 7% patients per year. Other important predictors of haemorrhage are decompensated cirrhosis and the endoscopic finding of red wale marks[897].

[896] North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. A prospective multicenter study. N Engl J Med. 1988;319:983-989.
[897] Garcia-Tsao G, Sanyal AJ, Grace ND, et al. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Hepatology. 2007;46:922-938.

The follow up recommendations for patients with oesophageal varices depend on the size of the varices and whether or not they have bled[898][899]:

- Patients with cirrhosis and no varices should have surveillance endoscopy every 2 to 3 years, or yearly if they develop decompensated cirrhosis.
- Patients who have cirrhosis and small varices should have repeat endoscopy every 1 to 2 years.
- Patients on beta-blocker treatment for prevention of variceal bleeding do not need surveillance endoscopy.
- Endoscopic surveillance schedule after variceal eradication by banding ligation is 3 months, then after 6 months, and then yearly.

The 6 week mortality for variceal bleeding is approximately 10% and the 1 year recurrence rate of bleeding is 60% if no preventative treatment is given[900]. Overall prognosis depends on the aetiology of the underlying portal hypertension and on the status of the liver function. Patients with oesophageal variceal bleeding have an overall 1 year mortality of 30% - 40% whilst patients with varices but no bleeding or ascites have a mortality rate of 3.4% per year[901].

In conducting the medical examination of a person with known oesophageal varices a full specialist assessment must be sought which should include but is not limited to underlying aetiology and it's prognosis, current status of liver function and likely course of deterioration, bleeding history, size of varices, likelihood of bleeding or other complication of liver diseas and the requirments for monitoring. Only then can a decision on fitness be made.

17.11.6.2 ACUTE LIVER FAILURE

Acute liver failure is a rare syndrome defined by a rapid decline in hepatic function characterised by jaundice, coagulopathy and hepatic encephalopathy in patients with no evidence of prior liver disease. The time course is usually accepted to be less than 26 weeks.

The incidence in the US is approximately 2000 cases per year yet it accounts for up to 6% of all liver related deathsand is responsible for 6% of liver transplants in the US[902]. In figures from the last 20 years it is shown that the majority of cases were women (67%) and the mean age was 38 years (range 17-79 years). Mortality without liver transplantation was 45%; 25% of cases received a transplant and the overall mortality was 30%[903].

[898] de Franchis R; Baveno V Faculty. Revising consensus in portal hypertension: report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2010;53:762-768.
[899] Tripathi D, Stanley AJ, Hayes PC, et al. UK guidelines on the management of variceal haemorrhage in cirrhotic patients. Gut. 2015;64:1680-1704.
[900] Garcia-Tsao G, Bosch J. Management of varices and variceal hemorrhage in cirrhosis. N Engl J Med. 2010;362:823-832.
[901] D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol. 2006;44:217-231.
[902] 2007 Annual Report of the U.S. Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients (OPTN/SRTR): Transplant Data 1997-2006. Rockville, MD: Health Resources and Services Administration, Healthcare Systems Bureau, Division of Transplantation; 2007.
[903] Lee WM, Squires RH Jr, Nyberg SL, et al. Acute liver failure: summary of a workshop. Hepatology. 2008;47:1401-1415.

This same prospective study identifies the causes of acute liver failure as follows:

- Paracetamol toxicity (46%). Other studies have shown that this is equally divided between intentional and unintentional cases[904]. Paracetamol may also be associated with many cases that are indeterminate[905]
- Idiosyncratic drug induced liver injury (11%). In total over half of all ALF cases are associated with a drug reaction and in cases of non paracetamol liver injury reports have noted a rise in cases resulting from complementary medicines in contrast with prescription medicines[906]
- Acute hepatitis B (8%)
- Autoimmune hepatitis (6%)
- Shock liver (4%)
- Acute hepatitis A (3%)
- Indeterminate (14%)

Retrospective studies elsewhere have demonstrated acute viral hepatitis is the most common cause of ALF, particularly hepatitis A and E[907]. The managememt of ALF depends on intensive care monitoring and supportive measures to treat the underlying aetiology. Liver transplantation should also be considered.

Any person who recovers spontaneously from ALF of any aetiology will require ongoing monitoring and specialist input. Depending on the cause they may have ongoing chronic liver disease requiring long term therapy. If a liver transplant has been performed they will require intensive post operative monitoring including management of their immunopsuppression. Patients with ALF who undergo liver transplantation appear to have a higher risk of death within the first 3 montjs following transplant and more commonly require re-transplantation compared with elective cases. Howeer the 1 year post transplant survival rate in the US has improved over the last 10 years form 73% to 82%[908]. Other studies have shown survival rates of 87% at 1 year and 78% at 3 years.

It should be noted that survival rates post ALF and post transplant vary between centres and local specialist input will be necessary in the assessment of all of these patients wishing to return to a career at sea. A time limited and restricted certificate may well be appropriate to allow sufficient ongoing care and monitoring, if the person is declared fit at all.

Reviewed 2015

[904] Larson AM, Polson J, Fontana RJ, et al. Acetaminophen-induced acute liver failure: results of a United States multicenter, prospective study. Hepatology. 2005;42:1364-1372.
[905] Davern TJ 2nd, James LP, Hinson JA, et al. Measurement of serum acetaminophen-protein adducts in patients with acute liver failure. Gastroenterology. 2006;130:687-694.
[906] NewHillman L, Gottfried M, Whitsett M, et al. Clinical features and outcomes of complementary and alternative medicine induced acute liver failure and injury. Am J Gastroenterol. 2016 Apr 5 [Epub ahead of print].
[907] Lee WM. Etiologies of acute liver failure. Semin Liver Dis. 2008;28:142-152.
[908] Liou IW, Larson AM. Role of liver transplantation in acute liver failure. Semin Liver Dis. 2008;28:201-209

17.11.7 BILIARY TRACT DISEASE

K 80-83	Biliary tract disease Likelihood of biliary colic from gallstones, cirrhosis of liver, liver failure	T – Biliary colic until definitely treated P – Advanced liver disease, recurrent or persistent impairing symptoms	R, L – Case-by-case specialist assessment. Does not meet requirements for unlimited medical certificate. Sudden onset of biliary colic unlikely.	Case-by-case specialist assessment. Very low likelihood of recurrence or worsening in next two years.

17.11.7.1 GALLSTONES

Gallstones are common, particularly in Western populations and occurs in 10 – 15% of adults in the US and Europe[909] and risk factors include age, obesity and female sex hormones[910]. 90% of gallstones are composed of cholesterol and form in the gallbladder[911].Despite it's high prevalence gallstones are generally asymptomatic in over 80% of people, however biliary pain will develop annually in 1 – 2% of individuals who were previously symptom free[912][913]. Those with a history of biliary colic are more likely to experience recurrent pain and are at increased risk of other complications. Major complications eg acute cholecystitis, cholangitis and acute pancreatitis occure at an annual rate of 0.1 – 0.3% among asymptomatic individuals harbouring stones[914].

Symptomatic gallstones require surgical cholecystectomy and any person undergoing such a procedure will need time to recover before being declared fit to return to work at sea. Asymptomatic stones do not require any treatment however if there is thought to be a heightened risk of developing gallbladder carcinoma (gallstones > 3cm or a calcified, 'porcelain' gallbladder) or when the risk of gallstone formatiomn and its complications are very high, elective cholecystectomy may be performed. For a person with asymptomatic gallstones a thorough, individualised risk assessment with specialist input as to the risk of complications arising must be undertaken prior to a fitness decision being made.

17.11.7.2 JAUNDICE

Jaundice, or icterus, is the result of the accumulation of bilirubin in the bloodstream and subsequent deposition in the skin, sclera and mucous membranes. The normal range for total bilirubin varies between laboratories but it is worth noting that jaundice may not be clinically evident until the serum level of bilirubin exceeds 51 micromol per litre (3mg7dl). For clinical

[909] Stinton LM, Shaffer EA. Epidemiology of gallbladder disease: cholelithiasis and cancer. Gut Liver. 2012;6:172-187.
[910] Sun H, Tang H, Jiang S, et al. Gender and metabolic differences of gallstone diseases. World J Gastroenterol. 2009;15:1886-1891.
[911] Diehl AK. Epidemiology and natural history of gallstone disease. Gastroenterol Clin North Am. 1991;20:1-19
[912] Freidman GD, Raviola CA, Fireman B. Prognosis of gallstones with mild or no symptoms: 25 years of follow-up in a health maintenance organization. J Clin Epidemiol. 1989;42:127-136.
[913] Gracie WA, Ransohoff DF. The natural history of silent gallstones: the innocent gallstone is not a myth. N Engl J Med. 1982;307:798-800.
[914] Friedman GD. Natural history of asymptomatic and symptomatic gallstones. Am J Surg. 1993;165:399-404

purposes the predominant type of bile pigments in the plasma can be used to classify hyperbilirubinaemia into two major categories:

- Plasma elevation of predominantly unconjugated bilirubin due to the overproduction of bilirubin, impaired uptake by the liver or abnormalities of bilirubin conjugation.
- Plasma elevation of both unconjugated and conjugated bilirubin due to hepatocellular disease, impaired canalicular excretion and biliary obstruction.

A detailed history and examination along with appropriate investigations is necessary to determine the cause of the jaundice and to identify any other abnormalities eg anaemia, further abnormal liver function, clotting abnormalities. Specialist input will be necessary and a detailed, individualised risk assessment must be performed.

It should be noted that survival rates post ALF and post transplant vary between centres and local specialist input will be necessary in the assessment of all of these patients wishing to return to a career at sea. A time limited and restricted certificate may well be appropriate to allow sufficient ongoing care and monitoring, if the person is declared fit at all.

Reviewed 2016

17.11.8 PANCREATITIS

K 85-86	Pancreatitis Likelihood of recurrence	T – Until resolved P – If recurrent or alcohol related, unless confirmed abstention. See alcohol abuse.	Case-by-case assessment based on specialist reports.	Case-by-case assessment based on specialist reports. Very low likelihood of recurrence.

Acute pancreatitis is an inflammatory condition of the pancreas characterized by abdominal pain and elevated levels of pancreatic enzymes in the blood. Incidence varies from 4.5 – 79.8 per 100000 per year in different countries. This variation is due to different diagnostic criteria, geographical factors and changes over time[915]. Approximately 210 000 patients are admitted to hospital each year with pancreatitis and approximately 20% of these meet the criteria for severe pancreatitis alone in the US[916]. Several aetiological factors have been described for acute pancreatitis but in 10% - 20% of cases a cause cannot be identified[917] although 80% of these cases are thought to be related to biliary sludge.

The most common cause world wide is alcohol consumption and in the US 80 – 90% of cases are caused by gallstones or alcohol[918]. Other causes include:

- Hypertriglyceridaemia
- Hyper calcaemia

[915] Kingsnorth A, O'Reilly D. Acute pancreatitis. BMJ. 2006;332:1072-1076.
[916] Swaroop VS, Chari ST, Clain JE. Severe acute pancreatitis. JAMA. 2004;291:2865-2868.
[917] Whitcomb DC. Clinical practice. Acute pancreatitis. N Engl J Med. 2006;354:2142-2150.
[918] van Brummelen SE, Venneman NG, van Erpecum KJ, et al. Acute idiopathic pancreatitis: does it really exist or is it a myth? Scand J Gastroenterol Suppl. 2003;239:117-122.

- Pancreatic malignancy
- Post endoscopic retrograde cholangiopancreatography (ERCP) (2 -3%)
- Trauma
- Infections
- Drugs
- Autoimmunce conditions
- Hereditary

The majority of patients with acute pancreatitis will improve within 3 to 7 days of conservative management. Care is supportive and should take into account the underlying cause. In gallstone pancreatitis a cholecystectomy should be considered before discharge in mild cases and scheduled for a few months post recovery in patients with severe symptoms. If a patient is not suitable for surgery ERCP should be considered.

The mortality rate is influenced by the severity of the disease and many prognostic factors have been investigated and documented. In milder disease the mortality of the acute disease is 1%, however this increase in severe pancreatitis to 10% with sterile and 25% with infected pancreatic necrosis[919]. Long term prognosis is based on the underlying cause and the patient's compliance with lifestyle modifications. The disease usually resolves and leaves normal pancreatic function however it can become chronic in the event of recurrent alcohol intake, pancreas divisum or cystic fibrosis.

Any assessment of a person who has suffered an attack of acute pancreatitis must include a specialist report and information on the aetiology, severity, treatment and lifestyle advice given along with an estimate of the risk of recurrence with and without adherence to advice given. The need for any further treatment eg cholecystectomy must also be documented.

Reviewed 2016

17.11.9 STOMA

Y 83	Stoma (ileostomy, colostomy) Impairment if control is lost – need for bags, etc. Potential problems during prolonged emergency	T – Until stabilised P – Poorly controlled	R – Case-by-case assessment	Case-by-case specialist assessment.

Ileostomy or colostomy creation may be required temporarily or permanently for the management of a variety of pathological conditions including congenital anomalies, colon obstruction, inflammatory bowel disease, intestinal trauma and gastrointestinal malignancies. The anatomic location and the type of stoma construction have an impact on management – loop colostomies tend to be larger and somewhat more difficult to manage than end

[919] Nirula R. Chapter 9: Diseases of the pancreas. High yield surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2000.

colostomies and the type and volume of output is determined by the location of the stoma relative to the ileocecal valve. Ileostomies, cecostomies and ascending colonostomies typically produce more than 500mls of effluent in 24 hours that contains digestive enzymes and is irritant to the skin. Descending/sigmoid colostomies produce stool that tis formed and does not contain digestive enzymes[920]. Ileostomy patients need to pay particular attention to fluid balance as fluid and electrolyte loss can be an issue, particularly in times of increased output or heavy perspiration. These patients may also have improper absorption of some minerals and vitamins and should avoid time released or enteric coated medication as these are likely to be incompletely absorbed.

Post stoma formation most of the activities of daily living can be resumed with minimal modification. Most sport activities can also be resumed, with the exception of contact sports which may damage the stoma. However ostomy pouches should not be exposed to extreme temperatures which may cause issues in some places and in some positions e.g. working in the engine room in the Middle East or the tropics. In considering the fitness of a person with a stoma the seafarer's doctor must ensure that the person can perform all of their routine and emergency duties for the required period of time and that they are well educated in the care of their stoma and how to handle a prolonged emergency situation.

Persons with a stoma will need to ensure that they have sufficient supplies of pouches etc. for their planned contract at sea as it may not be possible to obtain the supplies they need in other parts of the world. When travelling to and from ships by air they should be advised to take all of their medical supplies as hand luggage.

The incidence of stomal complications ranges from 14 – 79%[921][922] and these can occur in the early post operative period or many years following the construction of the stoma. Early complications occurring less than 30 days after surgery tend to be related to technical issues and include stomal necrosis, stomal bleeding, stoma retraction and mucocutaneous separation[923][924]. The most common late stomal complications (after 30 days) are parastomal hernia, stomal prolapse and stoma stenosis[925]. Complications vary with the type of stoma, with loop ileostomies have the highest rate. The most common problems of end and loop

[920] Colwell J. Principles of stoma management. In: Colwell, J, Goldberg, M, Carmel, J, Fecal and Urinary Diversions: Management Principles (Ed), Mosby, St. Louis 2004. p.240.
[921] Arumugam PJ, Bevan L, Macdonald L, Watkins AJ, Morgan AR, Beynon J, Carr ND; A prospective audit of stomas--analysis of risk factors and complications and their management. Colorectal Dis. 2003;5(1):49.
[922] Robertson I, Leung E, Hughes D, Spiers M, Donnelly L, Mackenzie I, Macdonald A; Prospective analysis of stoma-related complications. Colorectal Dis. 2005;7(3):279.
[923] Persson E, Berndtsson I, Carlsson E, Hallén AM, Lindholm E; Stoma-related complications and stoma size - a 2-year follow up. Colorectal Dis. 2010;12(10):971.
[924] Cottam J, Richards K, Hasted A, Blackman A; Results of a nationwide prospective audit of stoma complications within 3 weeks of surgery. Colorectal Dis. 2007;9(9):834.
[925] Shabbir J, Britton DC; Stoma complications: a literature overview. Colorectal Dis. 2010;12(10):958.

ileostomies are dehydration, skin irritation and small bowel obstruction; parastomal hernias are the most common complication for end and loop ileostomies and colostomies[926].

Risk factors for complications include[927]:

- Sub optimal stoma site
- Height of stoma <10mm
- Comorbid medical illness e.g. obesity, Crohn disease, inflammatory bowel disease, diabetes
- Tobacco usage
- Obesity is also an independent risk factor

When examining a person with a stoma for a fitness certificate the seafarer's doctor must consider the physical and mental status of the person, the impact of the stoma on his/her abilities to perform their routine and emergency duties, the risk of complications of the stoma and the likely disease progression and monitoring/follow up requirements of any underlying disease process. All in the context of potentially limited access to medical care. An individualised risk assessment must be carried out on each occasion with appropriate specialist input.

Reviewed 2016

[926] Robertson I, Leung E, Hughes D, Spiers M, Donnelly L, Mackenzie I, Macdonald A; Prospective analysis of stoma-related complications. Colorectal Dis. 2005;7(3):279.

[927] Cottam J, Richards K, Hasted A, Blackman A; Results of a nationwide prospective audit of stoma complications within 3 weeks of surgery. Colorectal Dis. 2007;9(9):834.

17.12 SKIN DISEASES

17.12.1 SKIN INFECTIONS

L 00-08	Skin infections Recurrence, transmission to others	T – Until satisfactorily treated P – Consider for catering staff with recurrent problems	R, L – Based on nature and severity of infection	Cured with low likelihood of recurrence

17.12.1.1 IMPETIGO

Impetigo is a contagious superficial bacterial infection most commonly seen in children although people of any age may be affected. Primary impetigo involves the direct bacterial invasion of previously normal skin whereas secondary impetigo is the infection of sites of minor skin trauma or underlying skin conditions e.g. eczema. Clinically the infection is classified to bullous or non bullous impetigo or ecthyma with lesions extending through the epidermis and into the dermis. The infection usually occurs in warm, humid conditions and is easily spread among individuals in close contact – risk factors include poverty, crowding, poor hygiene and underlying scabies[928]. Whilst the disease is usually self limiting in adults, rapid and effective treatment is necessary to reduce the spread of infection, hasten the resolution of discomfort and improve the cosmetic appearance[929]. Any person presenting for a medical examination with evidence of acute impetigo should be declared temporarily unfit until the lesions have resolved. Carriage of group A Streptococcus (GAS) and Staphylococcus aureus predisposes to subsequent impetigo[930] and it may be necessary to investigate this further, and instigate appropriate treatment.

17.12.1.2 HERPES SIMPLEX

Infection with Herpes Simplex Virus 1 (HSV 1) or Herpes Simplex Virus 2 (HSV 2) can cause genital, oral or ocular ulcers. Whilst oral herpes is always caused by HSV 1, either can cause genital lesions and both may cause both primary and recurrent episodes, some of which are asymptomatic. For both HSV 1 and HSV 2 asymptomatic shedding can occur in the absence of lesions and hence cause transmission to others during asymptomatic shedding. For persons with acute HSV infections due consideration should be made of their fitness to return to sea, additional advice given regarding the spread of the infection and appropriate investigations recommended e.g. HIV test at the time of diagnosis of primary genital herpes infection.

[928] Lejbkowicz F, Samet L, Belavsky L, Bitterman-Deutsch O. Impetigo in soldiers after hand-to-hand combat training. Mil Med. 2005;170(11):972.
[929] Koning S, van der Sande R, Verhagen AP, van Suijlekom-Smit LW, Morris AD, Butler CC, Berger M, van der Wouden JC. Interventions for impetigo. Cochrane Database Syst Rev. 2012;1:CD003261.
[930] Dajani AS, Ferrieri P, Wannamaker LW. Natural history of impetigo. II. Etiologic agents and bacterial interactions. J Clin Invest. 1972;51(11):2863.

17.12.1.3 VARICELLA ZOSTER

Varicella Zoster virus (VZV) causes two clinically distinct forms of disease:

- Varicella/chickenpox results from primary infection and produces a diffuse vesicular rash
- Herpes zoster/shingles results from reactivation of latent infection within the sensory dorsal root ganglion and produces a painful, unilateral vesicular eruption in a restricted dermatomal distribution

VARICELLA INFECTION (CHICKENPOX)

VZV is found worldwide and is extremely contagious. Over 90% of unimmunised people become infected during their lifetime but infection occurs at different ages in different parts of the world: in the UK, US and Japan, 80% of people have been affected by the age of 10 years and by the age of 20 to 30 years in India, South East Asia and the Caribbean[931] [932] [933]. The disease itself is usually self limiting although Varicella mortality data from the US (1990 – 1994) indicate that although less than 5% of varicella cases occur among adults aged over 20 years, 55% of varicella related deaths occur in this age group[934]. In another study, adults with chickenpox had a 25-fold higher risk of complications compared to children[935]. The most common complication in an immunocompetent adult is pneumonia –this accounts for the majority of morbidity and mortality and has a reported incidence of 1:400 cases[936]. Risk factors for the development of varicella pneumonia include cigarette smoking[937], pregnancy[938]and male sex[939], it typically develops within one to six days after the rash has appeared and its mortality is 10 – 30%[940].

Others complications are far rarer in adults but include:

- Skin/soft tissue infection e.g. cellulitis, myositis, necrotising fasciitis, toxic shock syndrome
- Neurological complications e.g. encephalitis (acute cerebellar ataxia or diffuse encephalitis), transient focal defects, aseptic meningitis, transverse myelitis, vasculitis and hemiplegia[941]
- Pharyngitis and otitis media

[931] Lee BW. Review of varicella zoster seroepidemiology in India and Southeast Asia. Trop Med Int Health. 1998;3:886-890.
[932] Kowitdamrong E, Pancharoen C, Thammaborvorn R, et al. The prevalence of varicella-zoster virus infection in normal healthy individuals aged above 6 months. J Med Assoc Thai. 2005;88(suppl 4):S7-S11.
[933] Garnett GP, Cox MJ, Bundy DA, et al. The age of infection with varicella-zoster virus in St Lucia, West Indies. Epidemiol Infect. 1993;110:361-372.
[934] Centers for Disease Control and Prevention. Varicella-related deaths among adults - United States, 1997. MMWR Morb Mortal Wkly Rep. 1997;46:409-412
[935] Guess HA, Broughton DD, Melton LJ 3rd, Kurland LT. Chickenpox hospitalizations among residents of Olmsted County, Minnesota, 1962 through 1981. A population-based study. Am J Dis Child. 1984;138(11):1055.
[936] Hockberger RS, Rothstein RJ. Varicella pneumonia in adults: a spectrum of disease. Ann Emerg Med. 1986;15(8):931.
[937] Fairley CK, Miller E. Varicella-zoster virus epidemiology--a changing scene? J Infect Dis. 1996;174 Suppl 3:S314.
[938] Esmonde TF, Herdman G, Anderson G. Chickenpox pneumonia: an association with pregnancy. Thorax. 1989;44(10):812.
[939] WEBER DM, PELLECCHIA JA. VARICELLA PNEUMONIA: STUDY OF PREVALENCE IN ADULT MEN. JAMA. 1965;192:572.
[940] Triebwasser JH, Harris RE, Bryant RE, Rhoades ER. Varicella pneumonia in adults. Report of seven cases and a review of literature. Medicine (Baltimore). 1967;46(5):409.
[941] Straus SE, Ostrove JM, InchauspéG, Felser JM, Freifeld A, Croen KD, Sawyer MH. NIH conference. Varicella-zoster virus infections. Biology, natural history, treatment, and prevention. Ann Intern Med. 1988;108(2):221.

The introduction of the varicella vaccine has led to a marked decrease in morbidity and mortality however vaccination schedules and efficacy vary between countries so the effect cannot be said to be worldwide.

VZV is also highly contagious with secondary household attack rates of > 90% in susceptible individuals[942]. The average incubation period for varicella infection is 14 to 16 days, although this interval can range from 10 to 21 days[943] and the period of infectivity is generally considered to last from 48 hours prior to the onset of rash until all skin lesions have fully crusted. It had been thought that second episodes of varicella infection in immunocompetent individuals occurred only rarely but post vaccination surveillance programmes have suggested that it may be as high as 13%[944]. Any person presenting with the signs and symptoms of acute varicella zoster infection should be declared temporarily unfit until all the lesions are crusted and there is no evidence of any complications.

HERPES ZOSTER/SHINGLES

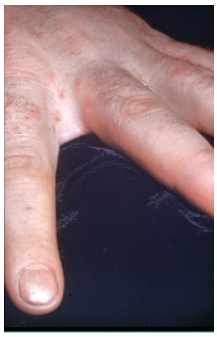
Following clinical resolution of VZV infection the virus may lie dormant in the sensory dorsal root ganglia of the spinal cord. Reactivation of this neurotropic virus leads to herpes zoster or shingles, a painful, unilateral vesicular eruption in a restricted dermatomal distribution. In the US it is estimated that 32% of people will suffer from zoster in their lifetime, with a cumulative lifetime incidence of 10 – 20%[945]. Incidence rates increase with age[946] and risk factors appear to be age, recent history of physical trauma[947], underlying malignancy, disorders of cell mediated immunity and chronic lung or kidney disease[948]. Therefore any person who is seen with zoster should be referred back to their primary care physician for a thorough history and examination to exclude any underlying causes. Dermatomal pain and a rash are the most common symptoms and less than 20% of patients have significant systemic symptoms e.g. headache, fever, malaise. The most common complication of herpes zoster is post herpetic neuralgia (7.9% of cases) and the risk of this increases with age. Secondary bacterial infection can also occur (2.3%) along with other rarer complications e.g. ocular complications, meningitis[949]. Whilst herpes zoster is less contagious than varicella it can spread person to person and can cause chickenpox in a previously uninfected person. Again the period of infectivity is from the appearance of the rash until all of the lesions have developed crusts. Hence any person with acute zoster infection

[942] Wharton M. The epidemiology of varicella-zoster virus infections. Infect Dis Clin North Am. 1996;10(3):571
[943] Heininger U, Seward JF. Varicella. Lancet. 2006;368(9544):1365.
[944] Hall S, Maupin T, Seward J, Jumaan AO, Peterson C, Goldman G, Mascola L, Wharton M. Second varicella infections: are they more common than previously thought? Pediatrics. 2002;109(6):1068
[945] Straus SE, Ostrove JM, InchauspéG, Felser JM, Freifeld A, Croen KD, Sawyer MH. NIH conference. Varicella-zoster virus infections. Biology, natural history, treatment, and prevention. Ann Intern Med. 1988;108(2):221
[946] Ragozzino MW, Melton LJ 3rd, Kurland LT, Chu CP, Perry HO. Population-based study of herpes zoster and its sequelae. Medicine (Baltimore). 1982;61(5):310.
[947] Zhang JX, Joesoef RM, Bialek S, Wang C, Harpaz R. Association of physical trauma with risk of herpes zoster among Medicare beneficiaries in the United States. J Infect Dis. 2013;207(6):1007
[948] McDonald JR, Zeringue AL, Caplan L, Ranganathan P, Xian H, Burroughs TE, Fraser VJ, Cunningham F, Eisen SA. Herpes zoster risk factors in a national cohort of veterans with rheumatoid arthritis. Clin Infect Dis. 2009;48(10):1364.
[949] Galil K, Choo PW, Donahue JG, Platt R. The sequelae of herpes zoster. Arch Intern Med. 1997;157(11):1209

should be declared temporarily unfit until the rash has completely crusted over and any underlying disease has been excluded or assessed thoroughly. If a person shows any complications of zoster an individual risk assessment should be carried out to determine his/her fitness to return to sea.

17.12.1.4 SCABIES

Scabies is caused by infestation with the ectoparasite, Sarcoptes scabiei, that causes an intensely pruritic eruption with a characteristic distribution pattern, particularly on the extremities. As many as 3 million people may be affected worldwide however this varies geographicallyand in come communities, particularly sub tropical and developing regions, prevalence may approach 50%[950]. Crowded conditions also increase the prevalence of scabies and scabies can occur in epidemics in institutions [951]. Transmission is usually from person to person by direct contact[952] but may also be through wearing or handling heavily contaminated clothing or sleeping in an unchanged bed recently occupied by an infested individual. Symptoms usually appear three to six weeks after primary infestation, however this may be reduced to one to three days in patients who have been previously exposed[953] [954]. The distribution of scabies usually involves the sides and webs of the fingers, the flexor aspects of the wrists, the extensor aspects of the elbows, anterior and posterior axillary folds, the skin immediately adjacent to the nipples (especially in women), the periumbilical areas, waist, male genitalia (scrotum, penile shaft, and glans), the extensor surface of the knees, the lower half of the buttocks and adjacent thighs, and the lateral and posterior aspects of the feet. The back is relatively free of involvement, and the head is spared except in very young children. Treatment with topical or oral medication is recommended and is also usually given to close contacts[955]. A person with scabies should be treated appropriately and may be declared temporarily unfit until one treatment has been given. An individual risk assessment should then be performed and the person must be advised on ways to minimise transmission to others.



Reviewed 2015

17.12.2 OTHER SKIN DISEASES

L10-99	Other skin diseases, e.g. eczema, dermatitis, psoriasis. Recurrence, sometimes occupational cause	T – Until investigated and satisfactorily treated	Case-by-case decision R – If aggravated by heat, or substances at work	Stable, not impairing

[950] Steer AC, Jenney AW, Kado J, et al. High burden of impetigo and scabies in a tropical country. PLoS Negl Trop Dis. 2009;3:467.
[951] Chosidow O. Clinical practices. Scabies. N Engl J Med. 2006;354(16):1718.
[952] Fuller LC. Epidemiology of scabies. Curr Opin Infect Dis. 2013 Apr;26(2):123-6.
[953] Chosidow O. Scabies and pediculosis. Lancet. 2000;355(9206):819.
[954] Vorou R, Remoudaki HD, Maltezou HC. Nosocomial scabies. J Hosp Infect. 2007;65(1):9.
[955]

17.12.2.1 ATOPIC DERMATITIS/ECZEMA

Atopic dermatitis is an inflammatory, pruritic skin disease with a chronic, relapsing course. Whilst it usually presents in childhood (with 70 – 85% of cases diagnosed by 5 years of age[956]) the disease persists into adult life in up to 30% of patients with many more experiencing relapses in later life[957]. The prevalence of atopic dermatitis is increasing in many industrialised nations[958] whilst children living in less hygienic environments in resource poor communities have a lower prevalence.

The aim of treatment is to reduce symptoms, prevent recurrences and minimize therapeutic risks. Topical treatment regimes with emollients plus/minus corticosteroids provide the main stay of treatment in mild and moderate disease[959] . Adults with severe disease may require the use of phototherapy and systemic immunosuppressants for adequate disease control[960]. Treatment should be ongoing even if the disease process is controlled to prevent recurrences and hence patient education and understanding is key. The elimination of exacerbating factors may also play a large role in preventing recurrences and reducing disease severity. Exacerbating factors that disrupt an already abnormal epidermal barrier include:

- Excessive bathing without subsequent moisturization
- Low humidity environments
- Emotional stress
- Dry skin
- Overheating of skin
- Exposure to solvents and detergents

Whilst no specific follow up regimes are in place, regular review to monitor the side effects of any medication may be necessary and the person will also need access to appropriate medication in case of recurrence or a complication such as infection. An individualised risk assessment should be carried out for each person suffering with atopic dermatitis and should take into account such factors as the severity of the disease, the need and possibility to avoid/eliminate exacerbating factors, the requirements for follow up and the necessary medication for the disease.

[956] Williams H. Atopic Dermatitis. N Engl J Med. 2005;352:2314-2324
[957] Spergel JM, Paller AS. Atopic dermatitis and the atopic march. J Allergy Clin Immunol. 2003;112:118-127
[958] Hurwitz S, Paller AS, Mancini J. Hurwitz clinical pediatric dermatology: a textbook of skin disorders of childhood and adolescence. 3rd ed. Philadelphia, PA; Edinburgh: Elsevier Saunders, 2006.
[959] Eichenfield LF, Tom WL, Berger TG, Krol A, Paller AS et al. Guidelines of care for the management of atopic dermatitis: section 2. Management and treatment of atopic dermatitis with topical therapies. J Am Acad Dermatol. 2014;71(1):116
[960] Sidbury R, Davis DM, Cohen DE, Cordoro KM, Berger TG, Bergman JN. Guidelines of care for the management of atopic dermatitis: section 3. Management and treatment with phototherapy and systemic agents. J Am Acad Dermatol. 2014;71(2):327

17.12.2.2 CONTACT DERMATITIS

Contact dermatitis is any dermatitis arising from direct skin exposure to a substance. It's prevalence in the US in 2004 was estimated at 24 400 per 100 000 per year[961]It may be allergic, causing a generalised allergic response or irritant where the irritant contact with the skin directly damages the skin. Allergic contact dermatitis is a significant cause of absenteeism in industry[962] and contact dermatitis in general is an important cause of occupational disability[963].

ALLERGIC CONTACT DERMATITIS (ACD)

This accounts for 20% of cases of contact dermatitis and occurs when a particular substance evoke a delayed, Type 4, hypersensitivity reaction. ACD usually presents with an intensely pruritic rash which can occur up to two weeks before the dermatitis appears. Hence identification of the trigger can difficult. Common sensitisers in the US include[964]:

- Plant oleoresin urushiol in poison ivy, poison oak, skin of mangoes
- Nickel in jewellery
- Formaldehyde
- Preservatives in topical medicines amd cosmetics
- Rubber
- Chemicals in shoes[965]

Typical physical findings include a papular erythematous rash distributed in the area of exposure. The extent of the dermatitis reflects the source of exposure (eg, cosmetics on the face, nickel where jewelry is worn, rubber where gloves are worn or elastic bands contact the skin, and points of shoe contact on the feet). Remote sites may less commonly be affected due to transfer of the allergen by the hands.

Treatment of the acute phase of ACD depends on the severity of the dermatitis. In mild or moderate cases the use of topical corticosteroids[966] for a limited course is successful, with the use of wet/semi wet dressings if necessary. In severe cases or where more than 10% of the total body surface area is involved treatment with systemic steroids or antihistamines may be required. Avoidance of the offending substance will usually result in control of the dermatitis within two to four weeks although as mentioned previously, such identification may be difficult, even with patch testing.

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[961] Bickers DR, Lim HW, Margolis D, et al. The burden of skin diseases: 2004 a joint project of the American Academy of Dermatology Association and the Society for Investigative Dermatology. J Am Acad Dermatol. 2006;55:490-500.
[962] Rosen RH, Freeman S. Occupational contact dermatitis in New South Wales. Australas J Dermatol. 1992;33(1):1.
[963] Belsito DV. Occupational contact dermatitis: etiology, prevalence, and resultant impairment/disability. J Am Acad Dermatol. 2005;53(2):303.
[964] Templet JT, Hall S, Belsito DV. Etiology of hand dermatitis among patients referred for patch testing. Dermatitis. 2004;15(1):25.
[965] Warshaw EM, Schram SE, Belsito DV, DeLeo VA, Fowler JF Jr, Maibach HI, Marks JG Jr, Mathias CG, Pratt MD, Rietschel RL, Sasseville D, Storrs FJ, Taylor JS, Zug KA. Shoe allergens: retrospective analysis of cross-sectional data from the north american contact dermatitis group, 2001-2004. Dermatitis. 2007;18(4):191.
[966] Saary J, Qureshi R, Palda V, DeKoven J, Pratt M, Skotnicki-Grant S, Holness L. A systematic review of contact dermatitis treatment and prevention. J Am Acad Dermatol. 2005;53(5):845.

Persons with an acute episode of ACD may need to be declared temporarily unfit until the precipitant has been identified and the acute symptoms are controlled. Identification of the allergen may be necessary to ensure that future avoidance is possible, particularly when there is thought to be an occupational aspect to the disease. If and when the person is declared fit to work at sea due consideration must be given to opportunities to avoid the allergen and treatment strategies for any acute episodes. A specialist dermatologist opinion should be sought in most cases and an individual risk assessment carried out in all cases.

IRRITANT CONTACT DERMATITIS (ICD)

ICD results from exposure to substances that cause physical, mechanical or chemical irritation of the skin. It is usually caused by common exposures that occur repeatedly on a daily basis eg soapy water, cleansers or rubbing alcohol. Some irritants eg bleach, strong acids or alkalis can cause severe ICD after one exposure. Mild irritants produce erythema, chapped skin, dryness and fissuring with varying degrees of pruritis whilst more severe cases present with oozing, oedema and tenderness. The hands are the usual site for ICD especially the web spaces of the fingers. Treatment is aimed at restoring a normal epidermal barrier and then protecting it from the irritant. In many cases decreasing exposure to the likely irritant and increasing the use of emollients is sufficient to control symptoms although in more severe cases topical corticosteroids may be required[967].

As with ACD any person with acute symptoms should be considered as temporarily unfit whilst symptoms are controlled and the precipitating factor identified. Again specialist input may be required and before the person returns to work a plan for avoiding and, if necessary, treating future acute episodes must be agreed with the person. An individualised risk assessment should be carried out.

Reviewed 2015

17.13 DISEASES OF THE MUSCULOSKELETAL SYSTEM

17.13.1 OSTEOARTHRITIS

M10-23	Osteoarthritis, other joint diseases and subsequent joint replacement. Pain and mobility limitation affecting normal or emergency duties. Replacement	T – Full recovery of function and specialist advice required before return to sea after hip or knee replacement P – For advanced and severe cases	R – Case-by-case assessment based on job requirements and history of condition. Consider emergency duties and evacuation from ship. Should meet	Case-by-case assessment if able to fully meet routine and emergency duty requirements. Very low likelihood of worsening such that duties could not be undertaken.

[967] Saary J, Qureshi R, Palda V, DeKoven J, Pratt M, Skotnicki-Grant S, Holness L. A systematic review of contact dermatitis treatment and prevention. J Am Acad Dermatol. 2005;53(5):845.

	joint: Possibility of infection or dislocation. Limited life of replacement joints.		general fitness requirements.	

17.13.1.1 OSTEOARTHRITIS (OA)

Osteoarthritis is the result of mechanical and biological events that destabilize the normal process of degradation and synthesis of articular cartilage, chondrocytes, extracellular matrix and subchondral bone. It most commonly presents over the age of 40 years with pain of the affected joint that is typically exacerbated by activity and relieved by rest. Stiffness is also a common symptom particularly in the mornings (lasting up to 30 minutes) or after a period of inactivity. The most commonly affected joints are:

- Cervical and lumbar spine
- First carpometacarpal joint
- Proximal interphalangeal joint
- Distal interphalangeal joint
- Hip
- Knee
- Subtalar joint
- First metatarsophalangeal joint

Patients with OA experience a range of severity of symptoms and the progression of the disease is difficult to predict. There is no cure but a combination of different treatments can provide adequate pain relief and preserve function and quality of life for many patients. Treatment options include non pharmacological therapies eg education, exercise, use of a brace and pharmacological treatments such as topical or intra articular local analgesia, systemic analgesics and anti inflammatories (NSAIDS or COX-2 inhibitors). The side effects of such treatments themselves will need to be monitored and treated where necessary. If such treatments are inadequate joint replacement may be advised.

17.13.1.2 RHEUMATOID ARTHRITIS (RA)

Rheumatoid arthritis is a chronic, symmetric, inflammatory, peripheral poly arthritis of unknown aetiology. It affects around 1% of the population making it the most common inflammatory arthritis. Patients are usually in their 50s when diagnosed and there is a slight female preponderance, particularly in younger patients when it is 2:1[968]. Approximately 40% of patients with RA will develop involvement of other parts of the musculoskeletal system apart from joints and other organs eg skin, eye, heart, lung, kidneys[969]. These manifestations are more common in severe disease, in those of an increasing age, with the presence of rheumatoid factor and

[968] Lee DM, Weinblatt ME. Rheumatoid arthritis. Lancet. 2001;358:903-911.
[969] Turesson C, O'Fallon WM, Crowson CS, Gabriel SE, Matteson EL. Extra-articular disease manifestations in rheumatoid arthritis: incidence trends and risk factors over 46 years. Ann Rheum Dis. 2003;62(8):722.

other antibodies and in patients with early disability and who smoke. The scope and clinical course of such disease manifestations is outside the scope of this guidance but extra articular involvement must be considered in any patient with RA and it's presence or absence should form part of an individualised risk assessment.

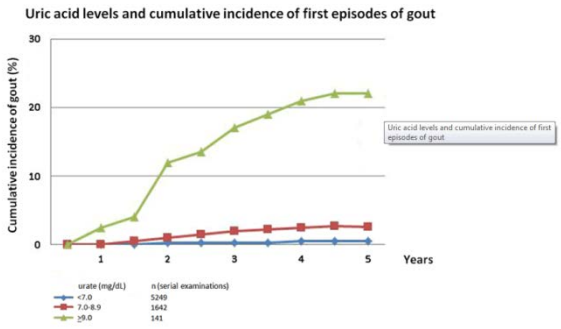
The aim of treatment and the modalities used are similar to those discussed above in patients with OA. However studies have shown that RA is associated with increased mortality and morbidity and that delay in treatment of the disease process itself contributed greatly to both[970]. Hence disease modifying drugs eg methotrexate, leflunomide, sulphasalazine and hydroxychloroquine tend to be introduced in patients with mild to moderate disease. For more severe disease biological agents eg Tumour Necrosis Factor may also be used.

All of these disease modifying drugs require specific investigations prior to commencement and regular monitoring of their effect and side effects. Hence a restricted or time limited certificate may well be appropriate if the person is considered fit to work at sea at all.

17.13.1.3 GOUT

Gout is a syndrome characterized by hyperuriceamia and deposition of urate crystals causing acute attacks of acute inflammatory arthritis, topho around the joints, possible joint destruction, renal disease and uric acid renal calculi. It can affect any joint but most commonly presents in the first toe, foot, ankle, knee, fingers, wrist and elbow. The incidence increases with age and the annual incidence in people over 50 years in the US is 1,6 per 1000 in men and 0,3 per 1000 in women[971]. The prevalence in developed countries is about 1% with a male:female ratio of 7:1 – 9:1 although there are geographical and racial variations. The incidence of gout not due to diuretic use has doubled over the past 20 years and this may be due to lifestyle changes, increased obesity and other comorbidities including diabetes[972].

There is a causal relationship between hyperuriceamia and gout. Hyperuriceamia does not always lead to gout but the incidence of gout increases with the urate level. Risk factors for hyperuricaemia include dietary factors eg consumption of seafood, meat and alcohol[973]; increased endogenous production of urate due



[970] Pincus T, Callahan LF. Taking mortality in rheumatoid arthritis seriously--predictive markers, socioeconomic status and comorbidity. J Rheumatol. 1986;13:841-845.
[971] Abbott RD, Brand FN, Kannel WB, et al. Gout and coronary heart disease: the Framingham Study. J Clin Epidemiol. 1988;41:237-242.
[972] Arromdee E, Michet CJ, Crowson CS, et al. Epidemiology of gout: is the incidence rising? J Rheumatol. 2002;29:2403-2406.
[973] Choi HK, Atkinson K, Karlson EW, et al. Purine-rich foods, dairy and protein intake and the risk of gout in men. N Engl J Med. 2004;350:1093-1103.

to a high cell turnover eg haematological cancer; drugs eg diuretics; obesity; insulin resistance and hypertension[974].

The aim of short term treatment is to the rapid resolution of pain and the preservation of joint function. This is usually achieved with non steroidal anti inflammatory drugs or colchicine[975] although corticosteroids may be used if other options are contraindicated. An acute attack is painful and self limiting and may severly impact on a persons ability to conduct their routine or emergency duties. Therefore all persons suffering from an acute episode of gout at the time of medical examination should be declared temporarily unfit until the acute episode has been controlled and further assessment undertaken. The aim of long term management is to prevent recurrent attacks and chronic joint destruction. Management includes dietary modifications and weight loss where indicated, although evidence is lacking[976]. Prophylactic drug therapy eg allopurinol, probenecid, febuxostat is indicated by:

- Recurrent attacks (>3 per year)
- Tophaceous gout
- Radiographic changes and chronic destructive joint disease
- Urate nephrolithiasis
- Patient preference because of severe and debilitating polyarticular attacks.

Again all of these treatment options carry a significant risk of side effects and appropriate monitoring is required. This may mean that a restricted or time limited certificate is appropriate.

Whilst an acute attack is self limiting the risk of recurrence is high without long term treatment: 62%, 78% and 84% during the first, second and third years respectively[977]. Also, if untreated about 2% of patients will develop severe debilitating arthritis typically 20 years after the first attack[978]. Untreated gout and hyperuriceamia is also associated with renal insufficiency. In a veteran population of gout sufferers the rate of incidence of kidney disease was lower in men with controlled serum urate levels than with high serum urate levels: 2% vs. 4% at year 1, 3% vs. 6% at year 2, and 5% vs. 9% at year 3, respectively[979].

[974] Choi HK, Atkinson K, Karlson EW, et al. Obesity, weight change, hypertension, diuretic use, and risk of gout in men: the Health Professionals Follow-up Study. Arch Intern Med. 2005;165:742-748.
[975] Terkeltaub R, Furst RE, Bennett K, et al. Colchicine efficacy assessed by time to 50% reduction of pain is comparable in low dose and high dose regimens: secondary analyses of the AGREE trial. Abstract presented at: American College of Rheumatology Scientific Meeting; October 2009; Philadelphia, PA.
[976] Moi JH, Sriranganathan MK, Edwards CJ, et al. Lifestyle interventions for chronic gout. Cochrane Database Syst Rev. 2013;(5):CD010039.
[977] Yu TF, Gutman AB. Efficacy of colchicine prophylaxis in gout. Prevention of recurrent gouty arthritis over a mean period of five years in 208 gouty subjects. Ann Intern Med. 1961;55:179-192.
[978] Hench PS. The diagnosis of gout and gouty arthritis. J Lab Clin Med. 1936;220:48.
[979] Krishnan E, Akhras KS, Sharma H, et al. Serum urate and incidence of kidney disease among veterans with gout. J Rheumatol. 2013;40:1166-1172.

17.13.1.4 JOINT REPLACEMENT

Joint replacement may be the only option for relief of pain and restoration of function in a joint if conservative management has not been successful. Any plan for joint replacement should be discussed in detail with an appropriately qualified and experienced Orthopaedic surgeon with due consideration given to the likelihood of the person returning to work and being able to carry out his/her routine and emergency duties. Post operatively a specialist report must be obtained detailing the person's physical capability and functional ability, the likelihood of dislocation, infection and the need for future further surgery before a person is considered fit to return to work. A restricted or time limited certificate may be appropriate to allow monitoring and rapid access to medical care in case of complications.

Any decision to issue a medical certificate to a person with joint disease must take into consideration the person's physical capability and functional ability for both his/her routine and emergency duties, the need for symptom management and the risks and monitoring requirements of both the underlying disease and any side effects of the medication required.

Reviewed 2015

17.13.2 RECURRENT INSTABILITY OF SHOULDER OR KNEE JOINTS

M24.4	Recurrent instability of shoulder or knee joints Sudden limitation of mobility, with pain	T – Until satisfactorily treated	R – Case-by-case assessment of occasional instability	Treated; very low risk of recurrence

The multiple causes of joint disease and loss of function, stability and possibly mobility are beyond the scope of this text. Any person with a history of significant joint injury with loss of function and physical capability must be assessed by an Orthopaedic surgeon prior to being issued with a fitness certificate. Due consideration must be given to the risk of recurrence of the problem, including dislocation, locking etc and the impact on his/her ability to perform their routine and emergency duties and meet the physical capability requirements. Once specialist information is obtained an individual risk assessment must be carried out.

Reviewed 2015

17.13.3 BACK PAIN

M54.5	Back pain Pain and mobility limitation. Likelihood of acute exacerbation.	T – In acute stage P – If recurrent or incapacitating	Case-by-case assessment	Case-by-case assessment

It is estimated that up to 84% of adults have low back pain at some time in their lives[980], the prevalence increases with increasing age and annual incidence ranges from 4% to 93%[981]. Women tend to have a slightly higher incidence than men[982]. Musculoskelatal lower back pain is defined as pain, stiffness and or soreness below the 12th rib and above the gluteal folds persisting for up to 12 weeks. An exclusion diagnosis this is made by eliminating specific causes eg neurological compromise, neoplasia, inflammatory arthritis, fracture and referred pain. Usually a good history and physical examination is sufficient and imaging is rarely necessary in the initial evaluation of lower back pain of less than 4 weeks duration. The exact nature of the pain is often very difficult to identify but arises from any combination of pathology involving the discs, vertebrae, facet joints, ligaments and/or muscles[983]. Risk factors associated with back pain complaints include smoking, obesity, age, female gender, physically strenuous work, sedentary work, psychologically strenuous work, low educational attainment, Workers' Compensation insurance, job dissatisfaction, and psychological factors such as somatization disorder, anxiety, and depression[984] [985] [986] [987] [988].

Treatment is aimed at reducing pain and restoring functional status and physical capability. Options include:

- Lifestyle measures including a return to usual activities as soon as possible
- Pharmacological therapies eg non steroidal anti inflammatory drugs or analgesics
- Physiotherapy
- Other therapies eg spinal manipulation

Approximately 90% of acute non-specific lower back pain improves substantially within 4 – 6 weeks although one study has estimated that only approximately 25% of patients have recovered fully at 1 year[989]. The rates of recurrence are also significant with 50% – 59% experiencing some pain and 20% – 35% having functionally disabling back pain between 6 – 22

[980] Deyo RA, Tsui-Wu YJ. Descriptive epidemiology of low-back pain and its related medical care in the United States. Spine (Phila Pa 1976). 1987;12(3):264.
[981] Rubin DI. Epidemiology and risk factors for spine pain. Neurol Clin. 2007;25:353-371.
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[983] Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med. 2007;147:478-491.
[984] Skovron ML, Szpalski M, Nordin M, Melot C, Cukier D. Sociocultural factors and back pain. A population-based study in Belgian adults. Spine (Phila Pa 1976). 1994;19(2):129.
[985] Croft PR, Papageorgiou AC, Ferry S, Thomas E, Jayson MI, Silman AJ. Psychologic distress and low back pain. Evidence from a prospective study in the general population. Spine (Phila Pa 1976). 1995;20(24):2731.
[986] Croft PR, Papageorgiou AC, Thomas E, Macfarlane GJ, Silman AJ. Short-term physical risk factors for new episodes of low back pain. Prospective evidence from the South Manchester Back Pain Study. Spine (Phila Pa 1976). 1999;24(15):1556.
[987] Macfarlane GJ, Thomas E, Papageorgiou AC, Croft PR, Jayson MI, Silman AJ. Employment and physical work activities as predictors of future low back pain. Spine (Phila Pa 1976). 1997;22(10):1143.
[988] Steffens D, Ferreira ML, Latimer J, Ferreira PH, Koes BW, Blyth F, Li Q, Maher CG. What triggers an episode of acute low back pain? A case-crossover study. Arthritis Care Res (Hoboken). 2015 Mar;67(3):403-10.
[989] Croft PR, Macfarlane GJ, Papageorgiou AC, et al. Outcome of low back pain in general practice: a prospective study. BMJ. 1998;316:1356-1359.

months after the acute presentation[990]. If pain does not settle within 4 – 6 weeks or is recurrent a specialist opinion should be sought.

When issuing a certificate for a person with back pain the seafarer's doctor should ensure that he/she has all the information they require with regards to reports and the results of imaging where appropriate in order to conduct an individualised risk assessment. This should include but is not limited to: degree of pain, treatment requirments, need for follow up, physical capability, functional ability and the risk of recurrence requiring treatment and/or impacting on the person's ability to perform their routine and emergency duties.

Reviewed 2015

17.13.4 LIMB PROSTHESIS

Y 83.4 Z 97.1	Limb prosthesis Mobility limitation affecting normal or emergency duties	P – If essential duties cannot be performed	R – If routine and emergency duties can be performed but there are limitations on specific non essential activities	If general fitness requirements (C – Physical capability requirements) are fully met. Arrangements for fitting prosthesis in emergency must be confirmed.

Any person with a limb prosthesis must be able to fulfil his/her emergency duties and be able to fit the prosthesis in an emergency situation without assistance if necessary. Consideration must also be given to any other physical limitations or underlying medical condition eg diabetes, peripheral vascular disease. Specialist input should be obtained and a detailed individualised risk assessment completed.

Reviewed 2015

17.14 GENITO-URINARY CONDITIONS

N00, N17	Acute nephritis Renal failure, hypertension	P – Until resolved	Case-by-case assessment if any residual effects	Full recovery with normal kidney function and no residual damage
N03-05, N18-19	Sub-acute or chronic nephritis or nephrosis. Renal failure, hypertension.	T – Until investigated	R, L – Case-by-case assessment by specialist, based on renal function and likelihood of complications.	Case-by-case assessment by specialist, based on renal function and likelihood of complications.

[990] Carey TS, Garrett JM, Jackman A, et al. Recurrence and care seeking after acute back pain: results of a long-term follow-up study. Med Care. 1999;37:157-164.

17.14.1.1 ACUTE NEPHRITIS

ACUTE INTERSTITIAL NEPHRITIS (AIN)

AIN is a pattern of acute renal inflammation localised to the renal interstitium, usually triggered by medications, particularly antibiotics. Incidence and prevalence are largely under reported as a definitive diagnosis can only be made on renal biopsy and this may not be clinically indicated. The distribution of causes of AIN have been reported as :

- Medication – 70 – 75% (with antibiotics responsible for 30 – 49% of these cases)
- Infections (multiple organisms) – 4 – 10%
- Tubulointerstititial nephritis and uveitis (TINU) syndrome – 5 – 10%
- Systemic disease eg sarcoidosis, Sjogren's syndrome, SLE – 10 – 20%.

Any drug can cause AIN although only a few are reported with any frequency. AIN was particularly common with methicillin although this has now been withdrawn in many countries. The common drug causes of AIN now include:

- Antibiotics: virtually all penicillins and cephalosporins, as well as many sulfonamides, rifampicin, and some quinolones; beta-lactam antibiotics are the most common cause of AIN .
- Diuretics (several classes) .
- Non-steroidal anti-inflammatory drugs: virtually all NSAIDS trigger a unique reaction consisting of AIN with a concurrent nephrotic syndrome and are the most common cause in the elderly .
- Proton-pump inhibitors .
- Antihistamines: cimetidine and ranitidine .
- Other medications: allopurinol, phenindione, phenytoin, sulfadiazine, mesalazine, and warfarin .

Treatment depends on the underlying cause and if patients do not respond rapidly advice should be sought from a nephrologist. Corticosteroids may be necessary along with other supportive care aimed at maintaining fluid and electrolyte balance. The prognosis for AIN is good and most patients with drug induced AIN will have significant improvement in renal function once the offending medication is discontinued . Only a few require dialysis and this is often only in the short term. However renal function remains abnormal in a few patients and tubular-interstitial fibrosis on biopsy remains a long term consequence of AIN . Some patients have multiple relapses and require repeat or even long term use of corticosteroids. Regular testing of renal function and at least annual review of blood pressure is required to detect any progression of renal disease.

ACUTE GLOMERULONEPHRITIS (GN)

GN denotes glomerular injury and applies to a group of diseases that are generally, but not always, characterised by inflammatory changes in the glomerular capillaries and the glomerular basement membrane (GBM). The injury can involve a part or all of the glomeruli or the glomerular tuft and the inflammatory changes are mostly immune mediated. Again incidence and prevalence are likely to be under reported and it is estimated that for every patient with clinically apparent glomerulonephritis, approximately 5 – 10 patients have undiagnosed subclinical disease . Even with that, in the US and Europe, GN is the third most common cause of

end stage renal disease after diabetes and hypertension. Worldwide it is thought to be the commonest cause of end stage renal disease due to the various infectious agents in developing countries. The disease can result from renal-limited glomerulopathy or from glomerulopathy-complicating systemic disease eg SLE and rheumatoid arthritis. It is often idiopathic but other causes include :

- Infections (group A beta-haemolytic Streptococcus, respiratory and GI infections, hepatitis B and C, endocarditis, HIV, toxaemia, syphilis, schistosomiasis, malaria, and leprosy)
  Systemic inflammatory conditions such as vasculitides (SLE, rheumatoid arthritis, and antiglomerulobasement disease, Wegener's granulomatosis, microscopic polyarteritis nodosa, cryoglobulinaemia, Henoch-Schonlein purpura, scleroderma, and haemolytic uraemic syndrome)
- Drugs (penicillamine, gold sodium thiomalate, NSAIDs, captopril, heroin, mitomycin C, and ciclosporin)
- Metabolic disorders (diabetes mellitus, hypertension, thyroiditis)
- Malignancy (lung and colorectal cancer, melanoma, and Hodgkin's lymphoma)•
- Hereditary disorders (Fabry's disease, Alport's syndrome, thin basement membrane disease, and nail-patella syndrome)
- Deposition diseases (amyloidosis and light chain deposition disease).

Treatment is patient specific and aimed at reversing renal damage and preserving renal function. It is directed at the underlying aetiology and management of any complications eg hypertension, hypervolaemia and hyperlipidaemia.

Patients with post-streptococcal GN and IgA nephropathy have a low incidence of developing chronic kidney disease as long as the underlying disease is treated. Most patients eventually have complete clinical recovery from the initial episode. However for other glomerular diseases, the long-term prognosis tends to be better in patients who present with asymptomatic haematuria and proteinuria and who have focal, rather than diffuse, glomerular involvement on renal biopsy. Principal determinants of a relatively poor renal outcome include more severe renal dysfunction at presentation, more severe proteinuria, lack of response to initial treatment, and an enhanced amount of fibrotic changes, such as interstitial fibrosis and glomerulosclerosis on initial renal biopsy. Over 10 – 15 years, end-stage renal disease eventually occurs in up to 50 - 60% of untreated patients with membranoproliferative disease , and in approximately 20 - 25% of patients with Wegener's granulomatosis .

All patients will require regular and frequent monitoring of renal function, specific antibodies, blood pressure etc as determind by the underlying aetiology, the specialist managing the case and any signs of recurrence.

In assessing any person with an acute nephritis for sea service the seafarer’s doctor must perform an individualised risk assessment including but not limited to prognosis, treatment requirements and follow up arrangments for the underlying disease/precipitating cause and the acute nephritis itself, access to medical care, the risk of recurrence and the likelihood/presence of complications eg hypertension, ongoing renal dysfunction. Specialist input is essential.

17.14.1.2 RENAL FAILURE

Chronic renal failure/chronic kidney disease (CKD) is defined by either a pathological abnormality of the kidney eg proteinuria and/or haematuria or a reduction in the GFR of less than 60ml/minute/1.73 metres squared for more than 3 months, regardless of the cause[991]. This is a relatively common condition, often unrecognised until the advanced stages, and it is estimated that 10% of the worldwide adult population will have CKD[992]. The incidence is likely to increase due to an ageing population, a higher incidence of diseases such as Diabetes Mellitus (accounting for 40% of patients on renal replacement therapy) and hypertension (accounting for 33% of patients on renal replacement therapy) which are the most common causes in the adult population and also an increased incidence of glomerular disorders eg focal segmental glomerulosclerosis. Other causes of CKD include[993]:

- cystic disorders of the kidney (polycystic kidney disease)
- obstructive uropathy
- glomerular nephrotic and nephritic syndromes such as focal segmental glomerulosclerosis
- membranous nephropathy
- lupus nephritis
- amyloidosis
- rapidly progressive glomerulonephritis

In addition, individuals with a history of an episode of acute kidney injury are most likely to develop CKD or end stage renal disease in the future.

All aetiologies are progressive and the main goal of treatment is to slow the progressive loss of renal function and delay/prevent the need for renal replacement therapy or kidney transplantation. It is essential that treatment is started early in the course of CKD so risk factors can be managed appropriately eg optimal glycaemic control in DM, treatment of hypertension and complications eg volume overload, hyperkalaemia, metabolic acidoses, anaemia, secondary hyperparathyroidism can be treated.

All patients with CKD should be managed in a specialist unit and will require monitoring on a regular basis as determind by the underlying disease process, stage of disease and presence of complications. CKD will eventually lead to end stage renal disease and the need for renal replacement therapy although the rate of decline is variable and difficult to predict. CKD is also a strong cardiovascular risk factor and the majority of patients with CKD will die prior to reaching end stage renal disease.

[991] KDOQI Advisory Board Members. Clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Part 4. Definition and classification of stages of chronic kidney disease. Am J Kidney Dis. 2002;39:S46-S75.
[992] Hamer RA, El Nahas AM. The burden of chronic kidney disease. BMJ. 2006;332:563-564.
[993] National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. United States Renal Data System, USRDS 2006 annual data report: Atlas of end-stage renal disease in the United States. 2006. http://www.usrds.org (last accessed 27 July 2014).

The assessment of a person with chronic kidney disease must include specialist input and full consideration of the underlying disease, treatment required, monitoring schedule and the risk of complications with access to appropriate medical care. It is likely that a time limited or restricted certificate will be apporpriate if the person is fit at all and an individual risk assessment must be done on a regular basis.

Reviewed 2015

17.14.2 RENAL OR URETERIC CALCULUS

N20-23	Renal or ureteric calculus Pain from renal colic	T – Until investigated and treated P – Recurrent stone formation	R – Consider if concern about ability to work in tropics or under high temperature conditions. Case-by-case assessment for near-coastal waters.	Case-by-case assessment by specialist with normal urine and renal function without recurrence.

Renal and ureteric stones are a common problem and the lifetime prevalence is estimated to be between 5 – 12% with the probability varying according to age, gender (male:female 2 – 3:1[994]) and geographical location[995] [996]. Stone occurrence is relatively uncommon before the age of 20 years and peaks in the fourth to sixth decade of life[997]. It also has a higher prevalence in hot, arid or dry climates. Most people (80%) of patients with nephrolithiasis form calcium stones – other types include uric acid, struvite and cystine stones.

Risk factors for stone formation include:

- Dietary factors eg high animal protein intake, high sodium/low calcium/high oxalate intake and low fluid intake
- History of previous stones – the rate of stone recurrence is quoted at 10 – 30% at 3 – 5 years with idiopathic calcium oxalate stones[998] [999]. Much higher rates of recurrence (15% at one year, 35 – 40% at 5 years and 50% at 10 years) have been found in a separate study[1000] although this may be due to differences in imaging.
- Positive family history[1001]
- Enhanced enteric oxalate absorption eg gastric bypass surgery, bariatric surgery, short bowel syndrome[1002]
- Frequent upper urinary tract infections and the use of medication that may crystallize in the urine[1003]

[994] Hiatt RA, Dales LG, Friedman GD, et al. Frequency of urolithiasis in a prepaid medical care program. Am J Epidemiol. 1982;115:255-265.
[995] Norlin A, Lindell B, Granberg PO, et al. Urolithiasis. A study of its frequency. Scand J Urol Nephrol. 1976;10:150-153.
[996] Scales CD Jr, Smith AC, Hanley JM, et al. Prevalence of kidney stones in the United States. Eur Urol. 2012;62:160-165.
[997] Marshall V, White RH, De Saintonage M, et al. The natural history of renal and ureteric calculi. Br J Urol. 1975;47:117-124.
[998] Hiatt RA, Ettinger B, Caan B, Quesenberry CP Jr, Duncan D, Citron JT. Randomized controlled trial of a low animal protein, high fiber diet in the prevention of recurrent calcium oxalate kidney stones. Am J Epidemiol. 1996;144(1):25.
[999] Kocvara R, Plasgura P, Petrík A, LouzenskýG, BartonícковáK, Dvorácek J. A prospective study of nonmedical prophylaxis after a first kidney stone. BJU Int. 1999;84(4):393.
[1000] Uribarri J, Oh MS, Carroll HJ. The first kidney stone. Ann Intern Med. 1989;111(12):1006.
[1001] Curhan GC, Willett WC, Rimm EB, Stampfer MJ. Family history and risk of kidney stones. J Am Soc Nephrol. 1997;8(10):1568.
[1002] Asplin JR, Coe FL. Hyperoxaluria in kidney stone formers treated with modern bariatric surgery. J Urol. 2007;177(2):565.
[1003] Kopp JB, Miller KD, Mican JA, Feuerstein IM, Vaughan E, Baker C, Pannell LK, Falloon J. Crystalluria and urinary tract abnormalities associated with indinavir. Ann Intern Med. 1997;127(2):119.

- Hypertension - the risk of stone formation is increased two fold[1004]

The treatment of renal and ureteric calculi in the acute stage is based on analgesia and adequate fluid intake and the condition is usually self limiting. However urgent urological assessment may be required in the case of urosepsis, anuria, ongoing symptoms and acute renal failure. Patients should also be requested to sieve their urine and collect any stones that are passed as this may help in future management decisions.

Whilst it may be difficult to predict the first renal or ureteric calculus, all persons who experience nephrolithiasis should be assessed by a specialist to determine the risk of further stone formation. Many approaches/protocols can be used but the following groups are considered to be at moderate to high risk for stone recurrence:

- Middle-aged, white males with a family history of stones
- African Americans, a group in which stone formation is less common[1005]
- Patients with chronic diarrheal states and/or malabsorption, history of bowel surgery or bariatric surgery, pathologic skeletal fractures, osteoporosis, urinary tract infection, and/or gout
- Those with stones known to be composed of cystine, uric acid, or struvite
- Obese patients, or patients with diabetes, who have an increased incidence of uric acid stones[1006] [1007]

Advice should be given with regards to lifestyle modifications and any underlying risk factors eg hypertension should be managed appropriately. The assessment of a person's fitness to return to sea with a history of renal or ureteric calculi must be made on an individual basis with specialist input, including the risk of recurrence and the probability of complications and access to appropriate medical care.

ASYMPTOMATIC NEPHROLITHIASIS

It is common to find that people have asymptomatic kidney stone(s) and several studies have examined the natural history of asymptomatic calculi:

- A cohort of 110 patients with 160 asymptomatic kidney stones was followed with active surveillance (using renal ultrasound performed every 6 to 12 months)[1008]. During a mean follow-up of 3.4 years, 28% of stones produced symptoms and 17% required surgery for these symptoms; an additional 3% caused silent obstruction that required intervention. Lower pole stones were less likely to cause symptoms or pass spontaneously.

[1004] Cappuccio FP, Strazzullo P, Mancini M. Kidney stones and hypertension: population based study of an independent clinical association. BMJ. 1990;300(6734):1234.
[1005] Sarmina I, Spirnak JP, Resnick MI. Urinary lithiasis in the black population: an epidemiological study and review of the literature. J Urol. 1987;138(1):14.
[1006] Pak CY, Sakhaee K, Moe O, Preminger GM, Poindexter JR, Peterson RD, Pietrow P, Ekeruo W. Biochemical profile of stone-forming patients with diabetes mellitus. Urology. 2003;61(3):523.
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[1008] Dropkin BM, Moses RA, Sharma D, Pais VM Jr. The natural history of nonobstructing asymptomatic renal stones managed with active surveillance. J Urol. 2015;193(4):1265.

- Another study monitored 107 such patients for a mean of 32 months[1009]. The likelihood of developing symptoms was approximately 32% at 2.5 years and 49% at 5 years; the risk was lowest in patients who had no history of previous stones. Roughly 50% of symptomatic patients required a procedure (such as shockwave lithotripsy) for removal of the stone, while the remaining symptomatic patients passed the stone spontaneously.

In addition to these findings, a number of studies have found that patients with residual stones following shockwave lithotripsy or percutaneous stone removal are at increased risk for symptomatic stone episodes. However, these investigations also suggest that appropriate medical stone management can significantly reduce recurrent stone formation or growth of existing stones[1010] [1011] [1012]. Thus, certain asymptomatic patients should undergo evaluation and treatment, based upon their occupation (airline pilots, frequent business travelers) or complexity (neurologic disease, anatomic abnormalities of the urinary tract, such as urinary diversion or solitary kidney) but the decision to do this in a person should be made on an individual basis with full awareness of the limited access to medical care and the impact an acute episode may have on the person, his colleagues and the ship.

Active surveillance may be a reasonable approach in asymptomatic patients with small, non-infected calculi, without evidence of obstruction. A time limited or restricted certificate may be appropriate. However in patients with intermittently symptomatic calculi or in those individuals "at risk" for stone episodes (solitary kidney, urinary tract reconstruction, immunosuppression, etc), minimally invasive stone removal (SWL, ureteroscopy or percutaneous nephrolithotomy) is warranted. A metabolic evaluation and appropriate medical therapy should still be considered, regardless of the decision concerning removal of the present stone.

Reviewed 2015

17.14.3 PROSTATIC ENLARGEMENT/URINARY OBSTRUCTION

N33, N40	Prostatic enlargement/urinary obstruction Acute retention of urine	T – Until investigated and treated P – If not remediable	R – Case-by-case assessment for near-coastal duties.	Successfully treated. Low likelihood of recurrence.

Urinary tract obstruction is a common problem but a relatively rare cause of significant acute kidney injury. It can occur anywhere along the urinary tract, be acute or chronic, unilateral or bilateral and partial or complete. It is readily reversible if treated quickly but undiagnosed can predispose to urinary tract infection, urosepsis and eventually end stage renal disease. The causes vary with the age, gender, race and nationality of the patient. Common causes include:

- Renal stones – see notes above

[1009] Glowacki LS, Beecroft ML, Cook RJ, Pahl D, Churchill DN. The natural history of asymptomatic urolithiasis. J Urol. 1992;147(2):319.
[1010] Fine JK, Pak CY, Preminger GM. Fine JK, Pak CY, Preminger GM. J Urol. 1995;153(1):27.
[1011] Maloney ME, Springhart WP, Marguet CG, et AL. Appropriate medical treatment after percutaneous nephrolithotomy can control active stone disease in the presence of residual calculi. Journal of Urology. 2004; 171:302.
[1012] Osman MM, Alfano Y, Kamp S, Haecker A, Alken P, Michel MS, Knoll T. 5-year-follow-up of patients with clinically insignificant residual fragments after extracorporeal shockwave lithotripsy. Eur Urol. 2005;47(6):860

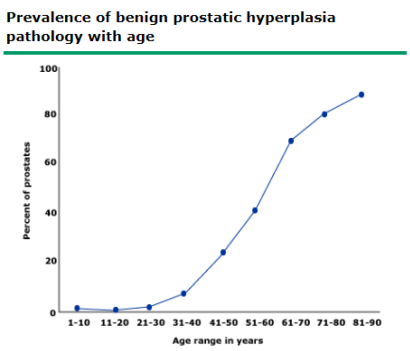
- Benign prostatic hypertrophy
- Prostate cancer
- Bladder tumours

Less commonly urinary tract obstruction can be caused by:

- Ureteropelvic junction obstruction
- Cystocoele
- Herniation of the bladder into the inguinal canal
- Iatrogenic injury during surgery
- Pelvic malignancy

The treatment of unilateral obstruction is aimed at the underlying cause, most commonly renal calculi. If the obstruction is bilateral a catheter will usually be placed to relieve the obstruction and appropriate additional therapy instigated. Ongoing treatment and the frequency and type of monitoring required will also depend on the cause and the initial treatment required. An individual risk assessment of fitness to work at sea must be performed for all persons with a history of urinary tract obstruction.

17.14.3.1 BENIGN PROSTATIC HYPERPLASIA (BPH)



BPH is a common problem amongst men, particularly with increasing age. Although the lack of a common definition has led to a difficulty in comparing the prevalence of BPH it is estimated to increase from 8% in men aged 31 – 40 years to 80% in men over 80 years (see diagram). Race may have some influence on the severity of BPH and the need for surgery with black men under the age of 65 years more likely to require surgical intervention than their white counterparts[1013]. In a separate study of over 34 000 men Asians had the lowest risk of symptoms, diagnosis and surgery with risks similar for blacks and whites[1014].

Treatment is aimed at the reduction in lower urinary tract outflow symptoms and may be either medical or surgical, depending on the severity of symptoms and response to medication. With or without treatment patients with diagnosed BPH should undergo monitoring of their clinical symptoms on a regular basis and all should be assessed with regards to the efficacy of treatment. In general patients started on alpha blockade will see an improvement in their symptoms after 1 -2 weeks, patients commenced on 5-alpha-reductase inhibitors will begin to see improvement in 4 – 6 months and patitents who have undergone surgery should be

[1013] Sidney S, Quesenberry CP Jr, Sadler MC, Guess HA, Lydick EG, Cattolica EV. Incidence of surgically treated benign prostatic hypertrophy and of prostate cancer among blacks and whites in a prepaid health care plan. Am J Epidemiol. 1991;134(8):825.
[1014] Kang D, Andriole GL, Van De Vooren RC, Crawford D, Chia D, Urban DA, Reding D, Huang WY, Hayes RB. Risk behaviours and benign prostatic hyperplasia. BJU Int. 2004;93(9):1241.

evaluated for response at 6 weeks following the procedure. If felt to be appropriate patients between the ages of 40 – 75 years may undergo screening for prostate cancer on an annual basis.

Clinic al progression of BPH itself occurs in approximately 20% of patients[1015] and the likelihood of any complication of BPH is low but include urinary tract infection, renal insufficiency, acute retention of urine and an overactive bladder. Approximately 2.5% of patients will develop acute urinary retention and another 6% will require invasive therapy over a 5-year time-frame.

All persons with BPH should be assessed on an individual basis and consideration given to the need for ongoing care, the requirements for follow up and the risk of complications. Specialist input is recommended.

17.14.3.2 ACUTE RETENTION OF URINE (ARU)

ARU is the sudden inability to voluntarily pass urine. It is the most common urological emergency[1016]. In men it is usually secondary to BPH and it's incidence increases with age[1017]. It is rare in women with a male:female incidence of 13:1 and it is estimated that there are 3 cases of AUR per 100 000 women per year[1018]. Any person who has experienced an episode of acute retention of urine should be investigated to determine the underlying cause. If this is/was found to be reversible eg urinary tract infection, medication then no further evaluation is needed. However if the aetiology is BPH or the aetiology of the episode is/was unknown further specialist assessment is warranted. This should include optimal management of the underlying condition and an estimate of the likelihood of recurrence, and this information must be included in an individualised risk assessment of the persons fitness to work at sea.

Reviewed 2015

17.14.4 GYNAECOLOGICAL CONDITIONS

N70-98	Gynaecological conditions – heavy vaginal bleeding, severe menstrual pain, endometriosis, prolapse of genital organs or other. Impairment from pain or bleeding.	T – If impairing or investigation needed to determine cause and remedy it	R – Case-by-case assessment if condition is likely to require treatment on voyage or affect working capacity	Fully resolved with low likelihood of recurrence

[1015] McConnell JD, Roehrborn CG, Bautista O, et al. The long-term effect of doxazosin, finasteride, and combination therapy on the clinical progression of benign prostatic hyperplasia. N Engl J Med. 2003;349:2387-2398.
[1016] Marshall JR, Haber J, Josephson EB. An evidence-based approach to emergency department management of acute urinary retention. Emerg Med Pract. 2014;16(1):1.
[1017] Jacobsen SJ, Jacobson DJ, Girman CJ, Roberts RO, Rhodes T, Guess HA, Lieber MM. Natural history of prostatism: risk factors for acute urinary retention. J Urol. 1997;158(2):481
[1018] Klarskov P, Andersen JT, Asmussen CF, Brenøe J, Jensen SK, Jensen IL, Lund P, Schultz A, Vedel T. Acute urinary retention in women: a prospective study of 18 consecutive cases. Scand J Urol Nephrol. 1987;21(1):29.

17.14.4.1 ABNORMAL UTERINE BLEEDING (AUB)

AUB refers to menstrual bleeding that is abnormal in quantity, duration or schedule. It is a common gynaecological complaint and accounts for 33% of out patient visits to Gynaecologists[1019]. Given the age range of most women persons we will concentrate on the causes, management and risks of AUB in the non pregnant woman of reproductive age. Causes, management and risks are different in pregnant females and post menopausal women.

AUB is common in women of 18 – 50 years and one US study has reported a prevalence of 53 per 1000 women[1020]. The importance of AUB relates to it's impact on quality of life and productivity and of course the female person's ability to manage such symptoms at sea and remain able to perform her routine and emergency duties. Each woman with a history of AUB should be thoroughly investigated in the appropriate setting as the causes are many and a detailed history and examination outside of the scope of the person medical examination is required. This should ensure the underlying aetiology is discovered and allow an individualized risk assessment based on the cause, necessary treatment and follow up, risks of complications and of course the person's current physiological status and physical capability.

17.14.4.2 DYSMENNORHOEA

Dysmennorhoea is estimated to affect 50 – 90% of reproductive aged women[1021] [1022] [1023] although the incidence decreases with increasing age[1024]. When severe it interferes with daily life and is a cause of abseentism from work or other commitments. There are often no risk factors for the disorder although a systemic review found that age < 30 years, BMI <20, smoking, menarche <12 years, longer menstrual cycles/duration of bleeding and history of sexual assault were associated with the disorder[1025]. In women complaining of severe pain associated with menstruation a detailed clinical history and examination, plus further investigations as appropriate, should be carried out to exclude significant pelvic pathology eg endometriosis, pelvic inflammatory disease, fibroids and confirm a diagnosis of primary dysmennorhoea. Treatment is aimed at providing adequate pain relief to allow the woman to perform most, if not all, of her usual duties. In the female person consideration must be given to her ability to perform routine and emergency duties when in pain, ensuring a sufficient supply

[1019] Spencer CP, Whitehead MI. Endometrial assessment re-visited. Br J Obstet Gynaecol. 1999 Jul;106(7):623-32.
[1020] Kjerulff KH, Erickson BA, Langenberg PW. Chronic gynecological conditions reported by US women: findings from the National Health Interview Survey, 1984 to 1992. Am J Public Health. 1996 Feb;86(2):195-9.
[1021] Burnett MA, Antao V, Black A, Feldman K, Grenville A, Lea R, Lefebvre G, Pinsonneault O, Robert M. Prevalence of primary dysmenorrhea in Canada. J Obstet Gynaecol Can. 2005;27(8):765.
[1022] Ortiz MI. Primary dysmenorrhea among Mexican university students: prevalence, impact and treatment. Eur J Obstet Gynecol Reprod Biol. 2010 Sep;152(1):73-7. Epub 2010 May 15.
[1023] Hillen TI, Grbavac SL, Johnston PJ, Straton JA, Keogh JM. Primary dysmenorrhea in young Western Australian women: prevalence, impact, and knowledge of treatment. J Adolesc Health. 1999;25(1):40.
[1024] Sundell G, Milsom I, Andersch B. Factors influencing the prevalence and severity of dysmenorrhoea in young women. Br J Obstet Gynaecol. 1990;97(7):588.
[1025] Latthe P, Mignini L, Gray R, Hills R, Khan K. Factors predisposing women to chronic pelvic pain: systematic review. BMJ. 2006;332(7544):749.

of analgesia for her contract and the side effects of any medication eg drowsiness. An individualized risk assessment must be performed.

17.14.4.3 ENDOMETRIOSIS

Endometriosis is the presence of endometrial glands and stroma outside of the endometrial cavity and uterine musculature. These implants of endometrium are usually in the pelvis but can occur anywhere in the body. It occurs during the active reproductive period (women aged 25 – 35 years)[1026] and is uncommon in pre or post menarchal girls and post menopausal women who are not taking oestrogen. General prevalence is difficult to estimate due to the range and diversity of symptoms although it is estimated at up to 50% in patients undergoing laparoscopy for pelvic pain or infertility[1027] [1028]. In a survey of 940 women with endometriosis, approximately 75% of symptomatic patients experienced pelvic pain and/or dysmenorrhea[1029]. Other presenting symptoms or findings were:

- Dysmenorrhea (79%)
- Pelvic pain (69%)
- Dyspareunia (45%)
- Bowel upset (eg, constipation, diarrhea) (36%)
- Bowel pain (29%)
- Infertility (26%)
- Ovarian mass/tumor (20%)
- Dysuria (10%)
- Other urinary problems (6%)

Endometriosis is a chronic and relapsing condition and it's natural history, untreated shows that at 6 – 12 months after diagnostic laparoscopy[1030] [1031]

- 22 – 29% had disease regression
- 29 – 45 had disease progression
- 33 – 42% had stable disease

Treatment can be with medication or surgery but should be individualized and aimed at the relief of symptoms experienced by an individual. Likewise there are no specific monitoring guidelines and follow up should be based on an individual basis and the primary complaint.

[1026] Olive DL, Schwartz LB. Endometriosis. N Engl J Med. 1993;328(24):1759.
[1027] Sangi-Haghpeykar H, Poindexter AN 3rd. Epidemiology of endometriosis among parous women. Obstet Gynecol. 1995;85(6):983.
[1028] Missmer SA, Hankinson SE, Spiegelman D, Barbieri RL, Marshall LM, Hunter DJ. Incidence of laparoscopically confirmed endometriosis by demographic, anthropometric, and lifestyle factors. Am J Epidemiol. 2004;160(8):784.
[1029] Sinaii N, Plumb K, Cotton L, Lambert A, Kennedy S, Zondervan K, Stratton P. Differences in characteristics among 1,000 women with endometriosis based on extent of disease. Fertil Steril. 2008;89(3):538.
[1030] Sutton CJ, Pooley AS, Ewen SP, Haines P. Follow-up report on a randomized controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal to moderate endometriosis. Fertil Steril. 1997;68(6):1070.
[1031] Abbott J, Hawe J, Hunter D, Holmes M, Finn P, Garry R. Laparoscopic excision of endometriosis: a randomized, placebo-controlled trial. Fertil Steril. 2004;82(4):878.

17.14.4.4 UTERINE LEIOMYOMAS (FIBROIDS)

Fibroids are benign tumours of the uterus and are the most common pelvic tumour in women[1032] [1033]. A hysterectomy study found myomas in 77% of uterine specimens[1034]. One study has shown the crude incidence of uterine fibroids to be about 1% per year and the incidence was shown to be significantly increased with advancing age, black race (3-fold), increased BMI, history of infertility, and current alcohol consumption[1035]. Fibroids usually present with heavy or prolonged menstrual bleeding, pelvic pressure or pain or reproductive dysfunction. The natural history of fibroids in pre menopausal women is variable and prospective studies have shown that between 7 – 40% of fibroids regress over 6 months to 3 years[1036] [1037]. Treatment is aimed at the amelioration of symptoms whilst addressing any future fertility desires and wishes regarding uterine preservation. For patients with no or minimal symptoms who elect for non surgical intervention annual follow up is sufficient. Surgical options include myomectomy and uterine artery embolization although hysterectomy remains the most successful treatment of symptomatic uterine fibroids. When assessing a female person with a diagnosis of fibroids full consideration must be given to the symptoms, necessary follow up and treatment required in an individualized risk assessment.

17.14.4.5 PELVIC ORGAN PROLAPSE (POP)

POP is the herniation of the pelvic organs to or beyond the vaginal wall. It is a common condition although prevalence is difficult to estimate due to differences in diagnostic criteria and the wide range and diversity of symptoms. However a large managed health care population study in the US estimated that American women have an 11.1% risk of POP and urinary incontinence surgery before the age of 80 years with nearly 30% having more than one procedure[1038]. In the UK, the Oxford Family Planning Association Study, of 17,032 women between the ages of 25 and 39 years, revealed that the incidence of patients admitted to hospital with prolapse was 2.04 per 1000 person-years of risk[1039]. Risk factors include:

- Parity – the risk of POP increases with increasing parity

[1032] Baird DD, Dunson DB, Hill MC, Cousins D, Schectman JM. High cumulative incidence of uterine leiomyoma in black and white women: ultrasound evidence. Am J Obstet Gynecol. 2003;188(1):100.
[1033] Buttram VC Jr, Reiter RC. Uterine leiomyomata: etiology, symptomatology, and management. Fertil Steril. 1981;36(4):433.
[1034] Cramer SF, Patel A. The frequency of uterine leiomyomas. Am J Clin Pathol. 1990;94(4):435.
[1035] Marshall LM, Spiegelman D, Barbieri RL, et al. Variation in the incidence of uterine leiomyoma among premenopausal women by age and race. Obstet Gynecol. 1997;90:967-973.
[1036] Peddada SD, Laughlin SK, Miner K, Guyon JP, Haneke K, Vahdat HL, Semelka RC, Kowalik A, Armao D, Davis B, Baird DD. Growth of uterine leiomyomata among premenopausal black and white women. Proc Natl Acad Sci U S A. 2008;105(50):19887.
[1037] DeWaay DJ, Syrop CH, Nygaard IE, Davis WA, Van Voorhis BJ. Natural history of uterine polyps and leiomyomata. Obstet Gynecol. 2002;100(1):3.
[1038] Brubaker L Bump R, Jacquetin B, et al. Pelvic organ prolapse. In: Abrams P, Cardozo L, Khoury S, et al, eds. Incontinence: 2nd international consultation on incontinence. Plymouth, UK: Health Publication Ltd; 2002:243-265.
[1039] Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. Br J Obstet Gynaecol. 1997;104:579-585.

- Advancing age – older women are at increased risk and one study showed an increased risk of 40% with every additional 10 years[1040]
- Obesity – women with a BMI >25 have a two fold increase in the risk of POP
- Race and ethnicity – African – American women have a lower prevalence of POP than other racial or ethnic groups in the US[1041]

Patients with POP may present with symptoms related specifically to the prolapsed structures, such as a bulge or vaginal pressure or with associated symptoms including urinary, defecatory or sexual dysfunction[1042]. The severity of symptoms does not correlate well with the degree of proplase and many women are asymptomatic. Prolapse has traditionally been regarded as a progressive disease, with mild prolapse inexorably leading to more advanced disease. However, data suggest that the course is progressive until menopause, after which the degree of prolapse may follow a course of alternating progression and regression. Prolapse regression was demonstrated in a prospective cohort study of 249 women who were followed over a three-year period[1043]. Prolapse increased by at least 2 cm in 11% of women and regressed by the same amount in 3% of women.

Treatment is often surgical although some women may be managed conservatively with physiotherapy and/or the use of a pessary[1044]. These women are likely to require follow up every 3 – 6 months although woman who are being observed as asymptomatic may only require annual follow up. Post surgery follow up is usually undertaken at 3 and 12 months. The risk of POP after surgery is not uncommon and nearly 30% of women undergoing one procedure have at least one more[1045]. Specialist input is required in the fitness assessment of any female person with POP and a risk assessment including symptoms and impact on activity, treatment and follow up and the likelihood of progression or complications must be undertaken.

Reviewed 2015

17.14.5 PROTEINURIA, HAEMATURIA, GLYCOSURIA

R31, 80, 81, 82	Proteinuria, haematuria, glycosuria or other urinary abnormality Indicator of kidney or other diseases	T – If initial findings clinically significant P – Serious and non-remediable underlying cause, e.g. impairment of kidney function	L – When repeat surveillance required R, L – When uncertainty about cause but no immediate problem	Very low likelihood of serious underlying condition

[1040] Swift S, Woodman P, O'Boyle A, Kahn M, Valley M, Bland D, Wang W, Schaffer J. Pelvic Organ Support Study (POSST): the distribution, clinical definition, and epidemiologic condition of pelvic organ support defects. Am J Obstet Gynecol. 2005;192(3):795.
[1041] Whitcomb EL, Rortveit G, Brown JS, Creasman JM, Thom DH, Van Den Eeden SK, Subak LL. Racial differences in pelvic organ prolapse. Obstet Gynecol. 2009;114(6):1271.
[1042] Jelovsek JE, Maher C, Barber MD. Pelvic organ prolapse. Lancet. 2007;369(9566):1027.
[1043] Bradley CS, Zimmerman MB, Qi Y, Nygaard IE. Natural history of pelvic organ prolapse in postmenopausal women. Obstet Gynecol. 2007;109(4):848.
[1044] Hagen S, Stark D. Conservative prevention and management of pelvic organ prolapse in women. Cochrane Database Syst Rev. 2011;(12):CD003882.
[1045] Olsen AL, Smith VJ, Bergstrom JO, et al. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol. 1997;89:501-506.

17.14.5.1 PROTEINURIA

Average daily urinary protein excretion in adults is 80 mg/day, with normal excretion considered to be <150 mg/day. Albumin represents approximately 15% of the daily urinary protein excretion in healthy people. Proteinuria varies in amount and may be transient or persistent[1046]. The presence of proteinuria is an independent risk factor for cardiovascular disease, death, and end-stage renal disease in the general population, and in patients with chronic kidney disease[1047] [1048] [1049]. Proteinuria is often diagnosed incidentally on routine dipstick test - it is common and prevalence increases with kidney disease progression. It is also likely to be more common in black people and those with an increased BMI[1050]. The sensitivity of the urinary dipstick for albumin ranges from 83% to 98% with a specificity of 59% to 86% [1051]and qualitative values can be estimated.

negative	0 mg/dL
trace	15-30 mg/dL
1+	30-100 mg/dL
2+	100-300 mg/dL
3+	300-1000 mg/dL
4+	>1000 mg/dL

Dipstick proteinuria ranges

It is important to distinguish between transient and persistent proteinuria by repeating the test after 1 – 2 weeks - the presence of dipstick positive proteinuria on two successive samples warrants further accurate quantification and further assessment[1052].

Transient proteinuria is common and is reported in 8 – 12% of young men[1053]. Common causes of transient proteinuria include fever, recent strenuous exercise, dysuria, urgency, frequency, foul-smelling/cloudy urine, and/or trauma. Persistent proteinuria will usually require referral to a nephrologist with further clinical assessment and the measurement of renal function as required. The decision on a person's fitness in this situation will depend upon but is not limited to the underlying cause, requirements for treatment and monitoring, likely disease progression, the risk of complications and the access to appropriate medical care if necessary. Specialist input is essential.

[1046] Viswanathan G, Upadhyay A. Assessment of proteinuria. Adv Chronic Kidney Dis. 2011;18:243-248.
[1047] van der Velde M, Matsushita K, Coresh J, et al. Lower estimated glomerular filtration rate and higher albuminuria are associated with all-cause and cardiovascular mortality. A collaborative meta-analysis of high-risk population cohorts. Kidney Int. 2011;79:1341-1352.
[1048] Gansevoort RT, Matsushita K, van der Velde M, et al. Lower estimated GFR and higher albuminuria are associated with adverse kidney outcomes. A collaborative meta-analysis of general and high-risk population cohorts. Kidney Int. 2011;80:93-104.
[1049] British Medical Journal. Low eGFR and high albuminuria predict end stage kidney disease and death at all ages. BMJ. 2012;345:e7478.
[1050] Kawar B, Bello AK, El Nahas AM. High prevalence of microalbuminuria in the overweight and obese population: data from a UK population screening programme. Nephron Clin Pract. 2009;112:c205-c212.
[1051] White SL, Yu R, Craig JC, et al. Diagnostic accuracy of urine dipsticks for detection of albuminuria in the general community. Am J Kidney Dis. 2011;58:19-28.
[1052] National Kidney Foundation. KDOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002;39(suppl 1):S1-S266.
[1053] Park YH, Choi JY, Chung HS, Koo JW, Kim SY, Namgoong MK, Park YS, Yoo KH, Lee KY, Lee DY, Lee SJ, Lee JE, Chung WY, Hah TS, Cheong HI, Choi Y, Lee KS. Hematuria and proteinuria in a mass school urine screening test. Pediatr Nephrol. 2005 Aug;20(8):1126-30. Epub 2005 Jun 10.

17.14.5.2 HAEMATURIA

Haematuria that is not explained by an underlying obvious condition is fairly common and in many cases, particularly in young adults, it is transient and of no consequence[1054]. However there is an appreciable risk of malignancy in adults over the age of 35 years with haematuria, even if it is transient[1055]. Haematuria can be grossly visible as red/brown urine or microscopic and found on incidental urine dipstick. Dipsticks are very sensitive for heam and so false negatives are unusual, however false positives may be due to:

- Semen in the urine after ejaculation may cause a positive haem reaction on the dipstick
- Alkaline urine with a pH greater than 9 or contamination with oxidizing agents used to clean the perineum
- Presence of myoglobinuria

Hence any positive dipstick result must be confirmed with microscopic examination of the urine. Common causes of haematuria include:

- Menstruation in women
- Urinary tract infection
- Pyelonephritis
- Nephrolithiasis
- Acute prostatitis
- Benign prostatic hyperplasia
- Trauma

More uncommon but clinically relevant causes include:

- Renal cell carcinoma
- Transient cell carcinoma
- Cystic kidney disease
- Renal infarction
- Renal vein thrombosis and many others

All persons with haematuria should be assessed further with a thorough clinical history and examination and investigations as indicated. The results of this assessment in a primary care or specialist setting will largely determine the person's fitness to serve at sea and whether or not any restrictions or time limitations are appropriate. Even if a cause for haematuria is not identified on this occasion patients will generally require follow up with urinalysis, cytology, blood pressure monitoring and possibly imaging – the need and timing of this follow up will depend on whether or not the haematuria was transient or persistent and upon the risk for malignancy.

[1054] Froom P, Ribak J, Benbassat J. Significance of microhaematuria in young adults. Br Med J (Clin Res Ed). 1984;288(6410):20.
[1055] Khadra MH, Pickard RS, Charlton M, Powell PH, Neal DE. A prospective analysis of 1,930 patients with hematuria to evaluate current diagnostic practice. J Urol. 2000;163(2):524.

17.14.5.3 GLYCOSURIA

The presence of glycosuria may be due to either the inability of the kidney to reabsorb filtered glucose in the proximal tubule despite normal plasma glucose levels (renal glycosuria) or urinary spillage because of abnormally high plasma glucose concentrations. Ascorbic acid can produce a false-negative test for glycosuria[1056]. Any patient with glycosuria should be investigated with other screening tests for diabetes eg fasting plasma glucose, oral glucose tolerance test and glycated haemoglobin. Further assessment of fitness will depend on the results of these investigations and a thorough clinical history and examination.

When glycosuria occurs with a normal plasma glucose, a primary defect of proximal tubule reabsorption needs to be considered and appropriate further clinical assessment and measurement of renal function undertaken . Glycosuria may coexist with additional manifestations of proximal tubular dysfunction, including phosphaturia (leading to hypophosphatemia), uricosuria, renal tubular acidosis, and aminoaciduria. This constellation is called the Fanconi syndrome and may result from a variety of disorders, including multiple myeloma, heavy metal exposure, and treatment with certain medications including tenofovir, lamivudine, cisplatin, valproic acid, and aminoglycosides[1057] . Glycosuria may also be an isolated defect (isolated renal glycosuria) associated with genetic mutations affecting renal glucose transport.

In all cases a detailed individual risk assessment of the person must be completed with appropriate specialist input before a certificate can be issued.

Reviewed 2015

17.14.6 REMOVAL OF ONE KIDNEY OR ONE NON-FUNCTIONING KIDNEY

Z90.5	Removal of kidney or one non-functioning kidney Limits to fluid regulation under extreme conditions if remaining kidney not fully functional.	P – Any reduction of function in remaining kidney in new person. Significant dysfunction in remaining kidney of serving person	R – No tropical or other heat exposure. Serving person with minor dysfunction in remaining kidney.	Remaining kidney must be fully functional and not liable to progressive disease. Based on renal investigations and specialist report.

In general most people with a single functioning kidney have few problems. However over time (25 years) there may be some decrease in kidney function, increased proteinuria, reduced glomerular filtration rate and the risk of hypertension. It is important to maintain appropriate hydration, to follow a sensible diet and to avoid situations where the remaining, functioning kidney may be injured eg contact sports. Follow up should be at least annually and may need to

[1056] Brigden ML, Edgell D, McPherson M, Leadbeater A, Hoag G. High incidence of significant urinary ascorbic acid concentrations in a west coast population--implications for routine urinalysis. Clin Chem. 1992;38(3):426.
[1057] Haque SK, Ariceta G, Batlle D. Proximal renal tubular acidosis: a not so rare disorder of multiple etiologies. Nephrol Dial Transplant. 2012 Dec;27(12):4273-87.

be more regular based on kidney function etc. A person with only one functioning kidney should have an individualised risk assessment including specialist input before a decision on fitness is made and a time limited certificate may be appropriate.

Reviewed 2015

17.15 PREGNANCY, CHILD BIRTH AND THE PUERPERIUM

O00-99	Pregnancy Complications, late limitations on mobility. Potential for harm to mother and child in the event of premature delivery at sea.	T – Late stage of pregnancy and early postnatal period. Abnormality of pregnancy requiring high level of surveillance.	R, L – Case-by-case assessment if minor impairing effects. May consider working until later in pregnancy in near-coastal waters.	Uncomplicated pregnancy with no impairing effects. Normally until 24th week. Pregnancy should be declared at an early stage so that necessary assessments can be made.

Ideally pregnancy should be declared by the person as soon as it is confirmed so arrangements for appropriate ante natal care and any necessary adjustments to the work place can be made. The schedule of recommended antenatal care varies between nations and the individual person should receive care equivalent to that they would have access to if they were working on shore. If there are no complications, appropriate antenatal care can be accessed and the working environment on board is suitable for the changing shape, mass and physiology of pregnancy, a pregnant person can usually continue to work at sea until week 24 of pregnancy. After this point the child may survive if born in a centre that can offer appropriate neonatal care and this is certainly not the case at sea. There may also be risks to the mother in the case of delivery and the pregnant person is unlikely to be physically capable of performing her routine or emergency duties at this point. Restriction to near coastal waters and short voyages may be considered depending on the role of the person. In all cases an individualised risk assessment must be carried out with input from a specialist and company doctor where appropriate.

Reviewed 2016

17.16 ICD 10 CONDITIONS NOT STATED ELSEWHERE

17.16.1 SPEECH DISORDERS

R47, F80	Speech disorders Limitations to communication ability	P – If incompatible with reliable performance of routine and emergency duties.	R – If assistance with communication/aids is needed to ensure reliable performance of routine and emergency duties. Specify assistance/aid.	Disorder does not impair reliable performance of routine and emergency duties.

A communication disorder refers to ‘an impairment in the ability to receive, send, process and comprehend concepts or verbal, non verbal and graphic symbol systems’ . Two major types of communication disorder are

- speech disorders – impairment in articulation, fluency (stuttering) and/or voice

- language disorders – impaired comprehension and/or use of spoken, written and/or other symbol systems.

17.16.1.1 SPEECH DISORDERS

These may be classified into

- Articulation disorders – difficulties with the production of speech sounds may be secondary to:
- Hearing impairment
- Neurological problems – dysarthria due to neuromuscular impairment secondary to a cerebrovascular accident, brain tumour etc
- Apraxia
- Structural defects eg cleft lip and palate, complete or partial glossectomy
- Fluency disorders – developmental stuttering is the most common fluency disorder and usually begins as a child. It is more common in males and has a high familial incidence.
- Voice disorders – are related to misuse or organic changes of the vocal mechanism. Causes include ulcers, vocal nodules, cancer, granuloma, infection etc. They also include resonance disorders with hyper- or hypo-nasality caused by structural defects of the palate or nasopharynx.

17.16.1.2 LANGUAGE DISORDERS

These may be separated to

- Developmental language impairment – a variety of developmental disorders, including those with cognitive impairment, in which speech and language are also affected.
- Specific language impairment – a developmental disorder that occurs in the absence of intellectual disability, hearing loss, motor disorder, socioemotional dysfunction or frank neurological deficit .
- Language disorders may be acquired or developmental and acquired causes include:
- Degenerative neurologic disorders
- Infection
- Neglect and abuse
- Head injury

Any assessment of the person with a communication difficulty should primarily be based on his/her ability to perform their routine and emergency duties safely and effectively. Specialist input should be obtained and an individual risk assessment carried out in each case. The need for aids, any exacerbating factors of the disorder eg stress and the presence of any underlying or associated disease process must be taken into consideration when making a fitness decision.

Reviewed 2015

17.16.2 ALLERGIES

T78 Z88	Allergies (other than allergic dermatitis and asthma) Likelihood of recurrence and increasing severity of response. Reduced ability to reliably perform routine and emergency duties.	T – Until fully investigated by specialist P – If life-threatening response reasonably foreseeable.	Case-by-case assessment of likelihood and severity of response, management of the condition and access to medical care. R – Where response is impairing rather than life-threatening, and	Where response is impairing rather than life-threatening, and effects can be fully controlled by long-term non-steroidal self-medication or by lifestyle modifications that are practicable at sea with

			reasonable adjustments can be made to reduce likelihood of recurrence.	no safety-critical adverse effects.

An allergic reaction to a substance can cause a wide range of symptoms from a localised swelling and discomfort to anaphylaxis – an acute, severe, life threatening allergic reaction in pre sensitised individuals with involvement of at least two organs. Allergic dermatitis and asthma are other manifestations of an allergic reaction and these have been discussed elsewhere. Whilst a local allergic reaction may be uncomfortable and prevent the person carrying out there duties for a short time it is usually self limiting and resolves quickly. It may however be a sign of a more severe reaction to come so should be documented and followed up as appropriate.

17.16.2.1 ANAPHYLAXIS

Due to the previous lack of a useful clinical definition and consensus criteria it is likely that anaphylaxis has been under reported and figures with regards to incidence and prevalence give a wide range. In the US the prevalence is estimated as 1% - 17% where 0,02% of the population may die from an anaphylactic reaction . Other studies from Europe, North America and Australia describe a lifetime prevalence of between 0,05% - 2%. Incidence and prevalence also differ for specific allergens. Common allergens include drugs, foods and insect stings but exercise may also trigger the condition.

Individuals with previous reactions are at higher risk for recurrence however the severity of the previous reaction does not necessarily predict the severity of a subsequent reaction . Persons must be educated with regards to recognition of the early symptoms of anaphylaxis, allergen avoidance, the need to carry any recommended medication eg adrenaline, chlorpheniramine, steroids and the requirement for early and effective treatment.

Any decision with regards to a person's fitness following an allergic reaction of any severity must include an individualised risk assessment with specialist input where appropriate. This should include but not be limited to the likelihood of exposure, the probability of a severe or life threatening reaction and the access to medical care.

Reviewed 2015

17.16.3 TRANSPLANTS

Z 94	Transplants – kidney, heart, lung, liver. (for prosthetics, i.e. joints, limbs, lenses, hearing aids, heart valves, etc. see condition-specific sections) Possibility of rejection. Side effects of medication.	T – Until effects of surgery and anti-rejection medication stable P – Case-by-case assessment, with specialist advice.	R, L – Case-by-case assessment, with specialist advice.	Not applicable.

Transplantation is the treatment of choice for end stage disease in many organs eg heart, liver, lungs and kidneys. Whilst it may offer an improved quality of life and survival rates are improving these patients require close follow up after transplantation with regards to their immunosuppression and the consequent risk of infection, malignancy and cardiovascular disease. In addition transplant patients often have co-morbidities as the cause of or due to the underlying disease and these diseases must be optimally managed. Patients will require regular medical review and investigations eg blood tests, urinalysis, Echo cardiography in addition to the rapid identification and treatment of any complications that may develop eg infection, renal dysfunction, new onset diabetes after transplantation and worsening of the graft function. Specialist input is vital to any individual risk assessment and fitness decision and if a person is considered fit at all a time limited and restricted certificate should be given.

Reviewed 2016

17.16.4 PROGRESSIVE CONDITIONS

Classify by condition	Progressive conditions, which are currently within criteria, e.g. Huntington's chorea (including family history) and keratoconus.	T – Until investigated and satisfactorily treated if indicated P – Consider at pre-sea medical if other choice of profession is more appropriate.	Case-by-case specialist assessment. Such conditions are acceptable if, within validity period of medical certificate, progression to a degree that impairs ability to perform routine and emergency duties is judged unlikely.	Case-by-case assessment, with specialist advice. Such conditions are acceptable if, within validity period of medical certificate, progression to a degree that impairs ability to perform routine and emergency duties is judged unlikely.

For any progressive condition it is important to document the current disease status, the requirement for medication and the impact both of these may have on the person's ability to perform his/her routine and emergency duties. A risk assessment based on this, the likelihood of complications or deterioration of the condition over the certificate validity period, the need for medical follow up and the effect of these on the person's ability to safely perform their routine and emergency duties at any time must be made on an individual, case by case basis.

17.16.4.1 HUNTINGTON'S CHOREA

Huntington's Disease (HD) is characterised by chorea, psychiatric illness and depression. A 2012 study made the following observations[1058]:

- Worldwide prevalence was 2.7 per 100 000
- In studies from Europe, North America and Australia the prevalence was 5.7 per 100 000
- In studies from Asia the prevalence was lower at 0.4 per 100 000

[1058] Pringsheim T, Wiltshire K, Day L, Dykeman J, Steeves T, Jette N. The incidence and prevalence of Huntington's disease: a systematic review and meta-analysis. Mov Disord. 2012 Aug;27(9):1083-91. Epub 2012 Jun 12.

- Worldwide incidence was 0.38 per 100 000

The disease affects men and women equally and typical onset is 35 – 45 years of age with a range from 2 – 80 years[1059]. Onset before the age of 20 years is classified as juvenile or Westphal variant of HD and accounts for less than 10% of cases[1060]. It is important to note that symptoms often begin insidiously with movement abnormalities, psychiatric disorder and/or cognitive features. Hence it is vital to listen to any concerns raised by family members or colleagues as patients may often not recognise/acknowledge their symptoms.

Motor symptoms and signs

Chorea is the defining symptom at diagnosis and may initially be mild and incorporated into purposeful movement by the patient. One study suggests that 50% of patients with motor signs were unaware of them at diagnosis[1061]. Gradually the chorea becomes more florid and widespread, interfering with movement and in later stages also affecting the diaphragm, pharynx and larynx. The inability to sustain certain simple voluntary acts is another common manifestation of HD, as is hypotonia with hyperreflexia and dystonia may be seen in the hands with such activities as walking. Abnormal eye movements may be seen and with disease progression motor function slowly deteriorates.

Psychiatric symptoms

Common symptoms associated with HD include depressed mood, irritability, apathy, and anxiety, with prevalences ranging from 33 - 76%[1062]. Other, less frequently observed symptoms are obsessive – compulsive disorder (10 – 52%) and psychosis (3 – 11%). Depression, paranoia, delusions, and hallucinations can develop at any point in the illness[1063] but Psychiatric symptoms do not correlate with duration of disease, repeat length, the presence of dementia or motor symptoms [1064] and may be present for a number of years before the onset of chorea[1065] [1066].

[1059] Hayden MR. Huntington's chorea. New York, NY: Springer; 1981.
[1060] Seneca S, Fagnart D, Keymolen K, Lissens W, Hasaerts D, Debulpaep S, Desprechins B, Liebaers I, De Meirleir L. Early onset Huntington disease: a neuronal degeneration syndrome. Eur J Pediatr. 2004;163(12):717.
[1061] McCusker EA, Gunn DG, Epping EA, Loy CT, Radford K, Griffith J, Mills JA, Long JD, Paulsen JS, PREDICT-HD Investigators of the Huntington Study Group. Unawareness of motor phenoconversion in Huntington disease. Neurology. 2013 Sep;81(13):1141-7. Epub 2013 Aug 21.
[1062] van Duijn E, Kingma EM, van der Mast RC. Psychopathology in verified Huntington's disease gene carriers. J Neuropsychiatry Clin Neurosci. 2007;19(4):441-8.
[1063] Shiwach R. Psychopathology in Huntington's disease patients. Acta Psychiatr Scand. 1994;90(4):241.
[1064] Zappacosta B, Monza D, Meoni C, Austoni L, Soliveri P, Gellera C, Alberti R, Mantero M, Penati G, Caraceni T, Girotti F. Psychiatric symptoms do not correlate with cognitive decline, motor symptoms, or CAG repeat length in Huntington's disease. Arch Neurol. 1996;53(6):493.
[1065] van Duijn E, Kingma EM, van der Mast RC. Psychopathology in verified Huntington's disease gene carriers. J Neuropsychiatry Clin Neurosci. 2007;19(4):441-8.
[1066] Kirkwood SC, Su JL, Conneally P, Foroud T. Progression of symptoms in the early and middle stages of Huntington disease. Arch Neurol. 2001;58(2):273.

Dementia

Cognitive decline is inevitable although the onset and rate of decline are variable. The main effects are a reduced ability to make decisions, multi-task and switch from one set of cognitive goals to another, so called executive dysfunction.

Patients suffering with HD should be reviewed every 6 – 12 months or sooner if the condition changes. This need for regular follow up will usually dictate that a time limited certificate is given if the person is considered fit enough to work at sea. Whilst some symptoms may be controlled with medication, HD is a chronic, progressive disease whatever the age of onset[1067]. The slow but relentless deterioration in cognitive and motor function causes significant morbidity and early mortality with a life expectancy from diagnosis of 10 – 30 years. At what point a person with HD becomes permanently unfit for sea service will depend on many factors and an individualised risk assessment, including detailed input from a specialist, must be done in each case and on each occasion that the person presents for examination.

17.16.4.2 KERATOCONUS

Keratoconus is a progressive, non-inflammatory disorder of the cornea with unknown aetiology characterised by progressive thinning of the cornea leading to a loss of visual acuity. Patients present in puberty or early adulthood and prevalence is estimated to range from 50 – 230 per 100 000 and there is no difference in incidence or prevalence between men and women [1068] [1069] [1070]. Unfortunately there are few large observational cohorts describing expected rates of progression. One study reported a 12% rate of keratoplasty over eight years of follow-up, factors associated with a higher likelihood of progression included younger age, corneal scaring, and worse visual acuity[1071]. The person must have appropriate visual acuity to meet the standards for fitness and a specialist report should be obtained outlining the likely rate of deterioration over the certificate validity period.

Reviewed 2015

[1067] Dorsey ER, Beck CA, Darwin K, Nichols P, Brocht AF, Biglan KM, Shoulson I, Huntington Study Group COHORT Investigators. Natural history of Huntington disease. JAMA Neurol. 2013 Dec;70(12):1520-30.
[1068] Krachmer JH, Feder RS, Belin MW. Keratoconus and related noninflammatory corneal thinning disorders. Surv Ophthalmol. 1984;28(4):293.
[1069] Kennedy RH, Bourne WM, Dyer JA. A 48-year clinical and epidemiologic study of keratoconus. Am J Ophthalmol. 1986 Mar;101(3):267-73.
[1070] Rabinowitz YS. Keratoconus. Surv Ophthalmol. 1998 Jan;42(4):297-319.
[1071] Gordon MO, Steger-May K, Szczotka-Flynn L, Riley C, Joslin CE, Weissman BA, Fink BA, Edrington TB, Olafsson HE, Zadnik K, Clek Study Group. Baseline factors predictive of incident penetrating keratoplasty in keratoconus. Am J Ophthalmol. 2006 Dec;142(6):923-30. Epub 2006 Sep 1.

17.16.5 CONDITIONS NOT SPECIFICALLY LISTED

Classify by condition	Conditions not specifically listed	T – Until investigated and satisfactorily treated if indicated P – If permanently impaired ability to reliably perform routine and emergency duties	Use analogy with related conditions as a guide. Consider likelihood of sudden incapacity, of recurrence or progression and limitations on performing normal and emergency duties. If in doubt, obtain advice and consider restriction	Use analogy with related conditions as a guide. Consider likelihood of sudden incapacity, of recurrence or progression and limitations on performing normal and emergency duties. If in doubt, obtain advice or consider restriction

There are many conditions that are not covered directly by the regulations and/or discussed in this document. It is hoped that having attended the taught course and used these guidelines for other conditions, the seafarer's doctor will be familiar with the process of medical selection and risk assessment. By using the same principles as are demonstrated throughout the course and this guidance the seafarer's doctor should feel comfortable to make and document their thought process and decision. Further assistance can be sought from the NMA/appellate body if and when necessary.

At all times the seafarer's doctor should bear in mind the purpose of the regulations as outlined in Section 1

"These Regulations shall ensure that the person is medically fit for service on board, is not suffering from a medical condition likely to be aggravated by service at sea or to endanger the health and safety of other persons on board."

Reviewed June 2018