Guidelines on autopsy practice: Sudden death with likely cardiac pathology

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In accordance with the College’s pre-publications policy, this document was on the College website for consultation from 29 April to 20 May 2015 and 37 items of feedback were received. The authors considered them and amended the document as appropriate. Please email publishing@rcpath.org if you wish to see the responses and comments.

All other comments regarding this document should be sent to the College’s non-forensic autopsy pathology lead, via clinicaledgefectiveness@rcpath.org

This document replaces earlier editions and is part of the ‘Guidelines on autopsy practice’ series.

Dr David Bailey
Vice President for Communications
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Foreword

The autopsy guidelines published by The Royal College of Pathologists (RCP) are guidelines that enable pathologists to deal with non-forensic consent and Coroner’s post mortems in a consistent manner and to a high standard. The guidelines are systematically developed statements to assist the decisions of practitioners and are based on the best available evidence at the time the document was prepared. Given that much autopsy work is single observer and one-time only in reality, it has to be recognised that there is no reviewable standard that is mandated beyond that of the FRCP Part 2 examination. Nevertheless, much of this can be reviewed against ante-mortem imaging and other data. It may be necessary or even desirable to depart from the guidelines in the interests of specific patients and special circumstances. The medicolegal risk of departing from the guidelines should be assessed by the autopsy pathologist; just as adherence to the guidelines may not constitute defence against a claim of negligence, so a decision to deviate from them should not necessarily be deemed negligent. In all cases, clinical judgement should be used to tailor the post mortem to the needs of that specific case and the questions it raises.

There is a general requirement from the General Medical Council (GMC) to have continuing professional development (CPD) in all practice areas and this will naturally encompass autopsy practice. Those wishing to develop expertise in or specialise in cardiac pathology are encouraged to seek appropriate educational opportunities and participate in the UK Cardiac Pathology Network external quality assessment (EQA) scheme.

The guidelines themselves constitute the tools for implementation and dissemination of good practice.

The stakeholders consulted for this document were the Human Tissue Authority and the UK Cardiac Pathology Network.

The information used to develop this document was derived from current medical literature and a previous version of this guideline. Much of the content of the document represents custom and practice, and is based on the substantial clinical experience of the authors. All evidence included in this guideline has been graded using modified SIGN guidance (see Appendix B).

No major organisational changes or cost implications have been identified that would hinder the implementation of this guideline.

A formal revision cycle for all guidelines takes place on a five-year cycle. The College will ask the authors of the guideline to consider whether or not the guideline needs to be revised. A full consultation process will be undertaken if major revisions are required. If minor revisions or changes are required, a short note of the proposed changes will be placed on the College website for two weeks for members’ attention. If members do not object to the changes, the short notice of change will be incorporated into the guideline and the full revised version (incorporating the changes) will replace the existing version on the College website.

The guideline has been reviewed by the Death Investigation Group and the Clinical Effectiveness Department, prior to being placed on the College website for consultation with the membership from 29 April to 29 May 2015. All comments received from the membership were addressed by the author to the satisfaction of the Vice-President for Communications.

This guideline was developed without external funding to the writing group. The College requires the authors of guidelines to provide a list of potential conflicts of interest; this is monitored by the Clinical Effectiveness Department and is available on request. The authors of this document have declared that there are no conflicts of interest.
1 Introduction

This document was created to address the needs of the autopsy pathologist dealing with deaths reflecting cardiac disease, and indicates a technical approach and investigations that should prevent criticism of case analysis in medicolegal environments. It should also serve to protect the needs of the living (i.e. surviving siblings and relatives with genetic conditions) as well as society in general. The limitations of local Coronial practice and permissions are often unique to different cases and various parts of the UK, but this documentary guidance should be satisfactory for all cases. The document is designed to be a focused bench-top guide with step-by-step examination suggestions. It highlights matters for consideration.

The levels of evidence reflect published case reports, analyses of cardiac diseases and the current status of cardiac pathology discussed at meetings – as summarised in the first two references. In some areas this knowledge has been tested in legal settings and is cited as level D (see Appendix B), whereas most is given as good pathology practice.

1.1 Target users of this guideline

The target primary users of this guideline are established consultants performing medicolegal and consent autopsies. The recommendations will also be of value to trainees, particularly those approaching the Certificate of Higher Autopsy Training (CHAT) examination and the FRCPath Part 2 in forensic pathology. In addition, these recommendations may be of use in other types of post-mortem examination (e.g. forensic post mortems) where the possibility of a cardiac cause of death is being entertained.

2 The role of the autopsy

- To establish whether death is related to cardiac disease or another process.
- To establish the nature of the cardiac disease, if present.
- To consider whether the cardiac disease identified is related to systemic disease.
- To consider whether any cardiac disease is likely to be inherited.
- To consider whether the cardiac disease could have been treated.
- To consider whether the cardiac disease is related to illicit activity (e.g. drug taking).

[Level of evidence: Grade D – the evidence has been taken from reviews of various texts/case reports and other presented cases, in medical and legal settings.]

3 Other (not primarily cardiac) pathology to be considered at the autopsy

Many cases of apparent sudden ‘cardiac’ death have no relevance to myocardial disorders. Indeed, fatal non-cardiac pathology (with cardiac-like symptoms) may be often encountered:

- pulmonary embolism
- pneumonia
- pancreatitis
- peptic ulceration/peritonitis
- abdominal/aortic aneurysm dissection/rupture.

Deaths occurring suddenly in epilepsy (SUDEP) need to exclude co-existent cardiac disease.
Exclusion of co-existent illicit and/or therapeutic psychiatric drugs should be considered. Even standard non-cardiac medications may have bearing upon cardiac electrical and contraction function.

Alcohol may be associated with sudden death, often as a consequence of inebriation. One should also be aware of alcoholic cardiomyopathy/sudden death in association with alcohol misuse (SUDAM) and other cardiac functional abnormalities. Alcohol and drug interactions must also be considered.

4 Causes of sudden death involving cardiac disease

Coronary artery disease/ischaemic heart disease
- Atheroma
- Coronary artery anomaly
- Kawasaki disease (paediatric)
- Vasculitis
- Myocardial bridging
- Coronary artery dissection
- Aortitis and secondary atherosclerosis
- Embolism into coronary arteries
- Fibromuscular dysplasia of the coronary arteries
- Regional coronary artery spasm.

Valve disease
- Aortic stenosis (senile/bicuspid)
- Mitral valve prolapse
- Rheumatic valve disease
- Infective endocarditis
- Tricuspid and pulmonary valve disease, in setting of congenital heart disease or other systemic disease.

Myocardial disease
- Myocarditis (lymphocytic, eosinophilic, neutrophilic, giant cell, sarcoïd/ granulomatous)
- Cardiomyopathies (HCM, DCM, ARVC, etc.)
- Left ventricle hypertrophy and hypertension
- Obesity-associated cardiomyopathy
- Idiopathic myocardial fibrosis
- Cardiac amyloid
- Storage disorders
- Connective tissue disorders (rheumatoid disease, lupus (SLE), sickle cell (HbSS), endocrine; hypothyroid/hyperthyroid states).

Congenital heart disease (+/- corrected), also known as GUCH (grown-up congenital heart disease).
Cardiac tumour
This is mostly cardiac myxoma, but rarely may be a primary malignant lesion (sarcoma). Metastasis from other tumour sites should not be missed as a terminal event promoting cardiac dysfunction and dysrhythmias.

Structural abnormalities of the conduction system
- Absence of part of the atrioventricular node
- Damage to the His bundle
- Inflammation (e.g. sarcoid)
- Cystic tumour of the atrioventricular node
- Wolfe-Parkinson-White syndrome and other aberrant pathways.

Note: It is impractical to serial section the whole conduction system and diagnoses such as Wolfe-Parkinson-White syndrome are made clinically and electrophysiologically. Nonetheless, targeted blocks of areas of interest may be pertinent such as in cases where sampling may be useful to assess for fibrous replacement of the AV node and bundle of His.

Drug toxicity
Cocaine, amphetamines/ecstasy, solvents marijuana and antidepressants/antipsychotics are the drugs most likely to be implicated in cardiac-related deaths but others may be relevant.

[Level of evidence: GPP.]

No morphological abnormalities (sudden arrhythmic death syndrome, SADS)
- Channelopathies
- Metabolic disease.

Pregnancy
- Ischaemic heart disease
- Congenital heart disease
- Cardiomyopathy
- Coronary artery dissection
- SADS.

Note: This list is not exhaustive and all-encompassing, but highlights the common lesions to be considered.

5 Specific health and safety aspects
Generally there are no specific infection hazards in this arena of cardiac tissue examination, beyond standard autopsy health and safety realities. However, it should be remembered that many patients have cardiac pacemakers, which may be relevant to the investigation/cause of death. Furthermore, it is stressed that some pacemakers (defibrillators) require deactivation prior to body examination. Finally, the presence of some cardiac/vascular devices may be composed of metal and may pose a hazard with sharp edges when cut for extraction.³

[Level of evidence: GPP.]
6 Relevant information required before the autopsy commences

- Circumstances of death
- Witness statements
- Previous medical history
- Medical therapy regime (current and prior)
- Previous surgical operations and other interventions
- Alcohol usage +/- illicit drug use
- Family history
- ECG, enzyme results and other pathological data
- Serum lipid profiles and other biochemical tests.

[Level of evidence: Stage D – the evidence has been taken from reviews of various texts/case reports and other presented cases, in medical and legal settings.]

7 The autopsy procedure

- A standard autopsy is required but with particular emphasis upon the cardiac and vascular tissues.
- Body mass is important to cross-compare with the heart weight.
- Examination of the cranial, lung, liver and kidney tissues must be done to give a balanced case analysis.

Staged dissection is recommended with consideration of any devices and their site, orientation and functionality. If necessary, the whole device should be retained for subsequent analysis. Care should be taken to deactivate any device prior to commencing the autopsy if it poses any risk to those conducting the autopsy. (See RCPath document, Guidance for pathologists conducting post-mortem examinations on individuals with implanted electronic medical devices, June 2015.)

- Photography is supported.
- The position of vascular access lines, tubes for ventilation, ECG pads, defibrillator units, etc., should be recorded.

[Level of evidence: GPP.]

8 The standard examination of the heart

The heart should be dissected in line with standard text guidelines, but is summarised here.

- Check pericardium for adhesion, effusions, exudates and blood accumulation.
- Transverse sections of the aorta, both venae cavae to release heart, making sure that transection is 1 cm above the right superior vena cava/atrium interface to preserve the sino-atrial node.
• Transverse section/internal palpation of the pulmonary artery to check for pulmonary emboli.

• Check the two coronary artery origins and distribution on the heart surface to exclude congenital anomalies.

• Examine any vein/internal mammary artery grafts. Check these bypass grafts and their patency. Possible reservation of graft/native vessel interface/anastomosis for histology.

• Examine any devices applied externally.

• Transverse section of the coronary arteries sequentially to exclude significant lumen obstruction by atheroma or other disease process. The evaluation of any stenosis is accepted as not absolute, but most pathologists are competent at assessing the grade of narrowing. Absolute definition of the lumen would require histology.

• Arteries with stents can be opened longitudinally with sharp scissors, removed and checked for thrombus macroscopically. An alternative would be to use a scalpel to cut down onto the stented vessel to inspect the stent lumen. Thrombus filling the lumen can be removed for standard histology. A stented vessel may occasionally be removed intact, fixed in resin and cut with special saws for resin embedded sections in specialist centres. Radiology may be of use in stented areas and compared with ante-mortem imaging.

• Transverse section across the ventricles up to the midsection of the ventricular tissues. This preserves the chordae and valvular tissues.

• Consider the chamber lumens and wall thickness. If felt necessary, take measurements at mid-ventricular level or 1 cm below valves in right and left ventricular outflow tracts, and record these in report.

• Opening the rest of the heart should be generally in the style of the ‘flow of blood’.

• The posterior right atrium and posterior aspect of the right ventricle are opened, showing the atrial/ventricular junction. Check the intact tricuspid valve before cutting open.

• The anterior right ventricle is opened into the pulmonary outflow tract and across the pulmonary valve.

• The left atrium is opened across the four veins. Check the intact mitral valve for prolapse up into the left atrium. Then a cut is made down the back of the left atrium and down across the back of the left ventricle to examine the mitral valve and atrial/ventricular junction.

• Check up into left ventricular outflow tract to see intact aortic leaflets or else view via cut aorta from above. The anterior aspect of the left ventricle is opened alongside the left anterior descending artery and across the aortic valve, passing behind the left main stem in front of the left atrial appendage. This allows examination of the aortic valve and coronary openings.

• Cardiac weight can now be assessed, once all blood/clot is removed. This should be compared against the body mass and/or standard cardiac weight tables.

• Any zones of fresh or previous infarction should be identified and quantitated.
• Fat replacement and scarring should be looked for carefully in the myocardium of both the right and left ventricle.

• Infiltrates are more difficult to identify, but general thickening of the myocardium and irregular stiffness of the tissues may be a clue to infiltrative diseases.

[Level of evidence: GPP.]

9 Histological examination

Clinical judgement must be used to assess the need for histology in each individual case. Where a pathologist is satisfied that the cause of death has been identified based on the balance of probability, there may be no need for histology to be taken. However, when this is not the case and the cause of death is felt likely to lie within the cardiovascular system, there should be a very low threshold for taking the relevant histological samples. Any sampling must be within the limits of consent in the case of a consented autopsy or within the limits of the relevant medicolegal legislation and guidelines, if the case is of a medicolegal nature. When the cause of death is felt to lie within the heart, but in which no obvious lesion is identified, sampling for histology is strongly advised. In such cases, the extent of sampling will be governed by the clinical judgement of the pathologist performing the autopsy. However where possible as a minimum, it is recommended to take mapped blocks of anterior, lateral and posterior right and left ventricle and septum from a representative mid-ventricular transverse slice and right ventricular outflow tract. Alternatively retention of the whole heart and sending it intact to a specialist centre for expert opinion should be considered.

The role of histology in cardiac tissue evaluation is paramount, and blocks should ideally be taken to illustrate any cardiac disease – such as one block from an infarcted area.

Coronary artery tissues may similarly be blocked or sectioned, according to need. It should be remembered that high-grade stenosis may require evaluation, following decalcification.

If there is no apparent focal lesion, multiple blocks should be taken across right ventricle and left ventricle/septum, or potentially large blocks should be taken to encompass the entire ventricular ring/septal tissues.

Samples of the right and left atria are generally not needed unless there is a focal lesion.

On occasion (i.e. not routinely), the sino-atrial node can be sampled in cases of sudden cardiac death or where there is no apparent pathology macroscopically. Likewise, the atrio-ventricular node can be blocked to show the nodal tissue, His bundle and branches.

Additional histochemical stains may be of use to assist general stains. A connective tissue stain (elastic van Gieson or Masson’s trichrome) is advisable. Congo/Sirius red (for amyloid), Perl’s Prussian blue (for iron) and PAS/ABPAS (for storage disorders), immunohistochemistry for CD3, CD20, CD68, etc. (myocarditis) should be done as required.

In cases where the cause of death is felt to be cardiac but in which no obvious abnormality is identified, it may be prudent to also sample background lung as well as kidney and liver or other relevant tissues as determined by the observed pathology.

In all cases, the histological sampling required must be guided by the clinical judgement of the pathologist conducting the case, guided by the specific requirements of that case.

[Level of evidence: GPP.]
10 Other samples that may be relevant

- If illicit drug use is suspected, peripheral blood should be sent for analysis.\(^4\)

- If acute anaphylaxis is suspected, peripheral blood should be sent for analysis (if possible, spun peripheral blood serum should be taken but it is recognised that this may not be possible in many mortuaries).\(^5\)

- If myocarditis is suspected, if possible a piece of fresh heart tissue should be retained for microbiological and viral studies.

- If a potentially inheritable disorder of the heart is suspected, fresh blood and/or a 2 cm cube of fresh spleen should be reserved if feasible. The authority/consent for this retention must, however, be ensured by the pathologist conducting the post mortem – be that through obtaining consent from someone in a suitable qualifying relationship to the deceased or through confirmation that such retention lies within the jurisdiction of the Coroner responsible for that case (as Coronial practice varies widely with respect to this issue). Under no circumstances should any tissue or sample be retained without confirmation that is legal to do so. If possible, the sample should be frozen or put directly into RNA later, pending onward referral for genetic testing. Failing that, the sample should be refrigerated and sent at the earliest opportunity, as fast as possible, to the genetic centre where longer-term storage can be addressed.

- In paediatric cases, the possibility of metabolic problems should be considered and appropriate samples (guided by clinicians and chemical pathologists) should be reserved.

[Level of evidence: GPP.]

11 Organ retention

Ideally the heart should be retained until all investigations are complete, whether by the primary or specialist cardiac pathologist. However, one has to be mindful of family, religious and cultural feelings on this matter and the benefit of having digital images of progressive dissection of cardiac tissues and then localised or targeted sampling is important. This can often obviate the need for whole organ retention.

It is also important, in potential cases of inherited cardiac disease, to advise the family to retain tissue blocks and slides. Whilst slide scanning may come to be a more useful medium in the near future, the value of retaining some tissue in paraffin-based format cannot be underestimated with evolving antibodies and other molecular tests.

In cases with medicolegal implications (usually those with forensic potential), cardiac tissue should ideally be retained until the police, court or Coroner/Fiscal completes their investigations.

[Level of evidence: GPP.]

12 Imaging and the cardiac autopsy

Post-mortem imaging is increasingly used in cases of sudden death. It currently has limited centre application in cardiovascular pathology, although this is likely to increase. At present, multidetector-computed tomography (MDCT), CT angiography (PMCTA) and cardiac magnetic resonance imaging (MRI) may be used.

[Level of evidence: GPP.]
13 Specific categorisation of sudden cardiac deaths

All causes of sudden cardiac death have to be correlated with the available clinical data and autopsy pathology findings. Simply finding some atheroma or microscopic small foci of scarring does not make this instantly the cause of death.

For cases of ischaemic/atheroma-related deaths, the coronary artery tissues should only be regarded as being of significance to the cause of death where there is 70% or more stenosis (e.g. lumen less than 1 mm), where one can show adherent/occluding thrombus or where there is ante-mortem evidence of high-grade occlusion by means of imaging criteria.

The presence of high-grade stenosis (greater than 90% occlusion in one or more vessels) is usually sufficient to assign the cause of death as ischaemic heart disease. This causes fatal cardiac dysrhythmia and complete occlusion by thrombus of such vessels can be the trigger factor which may have disappeared prior to postmortem.

Lesser degrees of atheroma (less than 70% stenosis) can also have the same reality, but this must be tested against absence of other pathology before deciding that this was the cause of death (on the balance of probability). It should be remembered that lower-grade atheroma may be complicated by acute plaque rupture/thrombosis which raises the degree of luminal obstruction dramatically and suddenly. This is often the cause of acute or zonal myocardial infarction. The finding of an acute myocardial infarct should point to the risk of cardiac dysrhythmia – often by re-entrant tachycardia, culminating in ventricular fibrillation and cardiac arrest.

Ischaemic heart disease of both acute and chronic patterns of myocardial scarring/damage is associated with cardiac dysrhythmias and death.

Acute and chronic myocardial infarction is also associated with ‘pump-failure’. The heart fails to maintain adequate output with secondary consequences upon the lungs with pulmonary oedema, pleural effusions, kidneys tubular necrosis and liver centrilobular necrosis.

Valvular heart disease is less common, and often under-appreciated, as a cause of sudden cardiac death. It is important assess the severity of any valvular disease, and measure fully the chamber diameters and wall thickness.

It may be necessary to take representative histology of the lungs, especially in congenital heart disease and when there is right ventricular hypertrophy pointing to pulmonary hypertension.

Inflammatory infiltrates and abnormal infiltrates such as amyloid should be sought.

Cardiac tumours are rarely encountered and their significance should be balanced against the ante-mortem history and other autopsy findings.

Cardiomyopathies deserve special mention. These include dilated cardiomyopathy (DCM; multiple causes being recognised), hypertrophic cardiomyopathy (HCM), arrhythmogenic (right ventricular) cardiomyopathy (ARVC) and other degenerative cardiomyopathies (mitochondrial, muscular dystrophy, etc.). These cardiomyopathies are usually associated with progressive cardiac failure as well as a risk of cardiac dysrhythmic death.

Cardiac hypertrophy, without overt genetic/structural aetiology, may be recognised as a cause of sudden death but requires exclusion of hypertension, genetic cardiomyopathic disease, infiltrates and valvular disease. However, to invoke cardiac hypertrophy (not otherwise specified) requires a cardiac mass in excess of 30% expected and no other drivers for the hypertrophy.
Finding a sudden death where there is no cardiac anomaly could point towards sudden arrhythmic death syndrome (SADS). Criteria to explore this potential require negative general autopsy findings both macroscopic and histological, along with negative toxicology and a morphologically normal heart. In such cases, it is strongly recommended that the heart is retained in its entirety and either sent for expert cardiac opinion or examined histologically in detail locally in an attempt to elucidate any underlying cause.

Note: It is essential that a 2 cm cube of fresh spleen should be retained, as described above.

[Level of evidence: GPP.]

14 Clinico-pathological correlation

Where possible, it may be valuable to debate the case with clinical colleagues. Clinical liaison with the general practitioner and or specialists in the hospital setting may serve to assist case review as well as benefit the understanding by relatives.

The pathologist should be prepared to speak to relatives and explain the findings, whether it be in the court setting or in a more neutral environment. Pathologists should be encouraged to promote awareness of this condition by means of close liaison with HM Coroner or other medicolegal authorities.

Referral to inherited heart disease clinics will be of benefit where a genetic cause is suspected, particularly with SADs and cardiomyopathies. The need for this should be highlighted in the post-mortem report so that those in receipt of this report can arrange such family follow up. This may be through the general practitioner. The College believe that while it is the responsibility of the pathologist to highlight the fact that such follow up may be beneficial, it is not up to the pathologist or pathology department to arrange such follow up. Rather it is the responsibility of the person in receipt of the post-mortem report, be that the lead clinician or the Coroner.

It should be noted that the words ‘cardiac failure’ ideally should not appear as part of the cause of death formulation. Whilst it is often of benefit to individual families to have it explained thus, it is better to define the cardiac pathology in technical format. ‘Acute cardiac failure due to myocardial infarction, due to coronary artery thrombosis’ may be easier to follow for relatives. However, it is debatably less pathologically rigorous than the alternative, ‘myocardial infarction, due to coronary thrombosis’, which serves in exactly in the same way.

[Level of evidence: Stage D.]

15 Examples of cause of death opinions/statements

1a. Acute myocardial infarction due to
1b. Coronary artery thrombosis with atheroma

1a. High grade (i.e. 90%+) occlusive coronary atheroma (i.e. no infarction)
1b. Established myocardial infarction
1b. Occlusive coronary atheroma

1a. Cardiomyopathy (note: specify the subtype)
1a. Aortic and mitral valve disease
1b. Rheumatic fever
1a. Rheumatic mitral and aortic valve stenosis (operated)
1b. Acute myocarditis
1c. Echovirus infection

1a. Sudden arrhythmic death syndrome (note: this will usually require a detailed discussion of the relevant possibilities and suggested investigations)

1a. Left ventricular hypertrophy (note: this will require usually a detailed discussion of the relevant possibilities and suggested investigations. It is likely that correlation against body mass and height data will be relevant, but one must be aware that a variety of social, racial and gender factors need to be considered).

Note: Modes of death, such as cardiac failure, dysrhythmia should not feature, as one should aim to delineate solely the pathology.

[Level of evidence: GPP.]

16 Criteria for audit

The following standards are suggested criteria that might be used in periodic reviews to ensure a post-mortem report for coronial autopsies conducted at an institution comply with the national recommendations provided by the 2006 NCEPOD study.6

• Supporting documentations:
  - standards: 95% of supporting documentation was available at the time of the autopsy
  - standards: 95% of autopsy reports documented are satisfactory, good or excellent.

• Reporting internal examination:
  - standards: 100% of the autopsy report must explain the description of internal appearance
  - standards: 100% of autopsy reports documented are satisfactory, good or excellent.

• Reporting external examination:
  - standards: 100% of the autopsy report must explain the description of external appearance
  - standards: 100% of autopsy reports documented are satisfactory, good or excellent.

A template for coronial autopsy audit can be found on The Royal College of Pathologists’ website (www.rcpath.org/profession/clinical-effectiveness/quality-improvement clinical-audit-templates.html).
17 References


Appendix A  AGREE compliance monitoring sheet

The accredited guidelines of The Royal College of Pathologists comply with the AGREE standards for good quality clinical guidelines. The sections of this guideline that indicate compliance with each of the AGREE standards are indicated in the table below.

<table>
<thead>
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<th>AGREE standard</th>
<th>Section of guideline</th>
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<tr>
<td><strong>SCOPE AND PURPOSE</strong></td>
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<tr>
<td>1. The overall objective(s) of the guideline is (are) specifically described.</td>
<td>Foreword</td>
</tr>
<tr>
<td>2. The clinical question(s) covered by the guidelines is (are) specifically described.</td>
<td>Foreword</td>
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<tr>
<td>3. The patients to whom the guideline is meant to apply are specifically described.</td>
<td>Foreword</td>
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<tr>
<td><strong>STAKEHOLDER INVOLVEMENT</strong></td>
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<tr>
<td>4. The guideline development group includes individuals from all the relevant professional groups.</td>
<td>Foreword</td>
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<tr>
<td>5. The patients’ views and preferences have been sought.</td>
<td>n/a</td>
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<tr>
<td>6. The target users of the guideline are clearly defined.</td>
<td>1</td>
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<tr>
<td>7. The guideline has been piloted among target users.</td>
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<tr>
<td><strong>RIGOUR OF DEVELOPMENT</strong></td>
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<tr>
<td>8. Systematic methods were used to search for evidence.</td>
<td>Foreword</td>
</tr>
<tr>
<td>9. The criteria for selecting the evidence are clearly described.</td>
<td>Foreword</td>
</tr>
<tr>
<td>10. The methods used for formulating the recommendations are clearly described.</td>
<td>Foreword</td>
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<tr>
<td>11. The health benefits, side effects and risks have been considered in formulating the recommendations.</td>
<td>Foreword</td>
</tr>
<tr>
<td>12. There is an explicit link between the recommendations and the supporting evidence.</td>
<td>Throughout</td>
</tr>
<tr>
<td>13. The guideline has been externally reviewed by experts prior to its publication.</td>
<td>Foreword</td>
</tr>
<tr>
<td>14. A procedure for updating the guideline is provided.</td>
<td>Foreword</td>
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<tr>
<td><strong>CLARITY OF PRESENTATION</strong></td>
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<tr>
<td>15. The recommendations are specific and unambiguous.</td>
<td>2–15</td>
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<td>16. The different options for management of the condition are clearly presented.</td>
<td>Foreword</td>
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<tr>
<td>17. Key recommendations are easily identifiable.</td>
<td>2–15</td>
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<tr>
<td>18. The guideline is supported with tools for application.</td>
<td>Throughout</td>
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<tr>
<td><strong>APPLICABILITY</strong></td>
<td></td>
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<tr>
<td>19. The potential organisational barriers in applying the recommendations have been discussed.</td>
<td>Foreword</td>
</tr>
<tr>
<td>20. The potential cost implications of applying the recommendations have been considered.</td>
<td>Foreword</td>
</tr>
<tr>
<td>21. The guideline presents key review criteria for monitoring and/audit purposes.</td>
<td>16</td>
</tr>
<tr>
<td><strong>EDITORIAL INDEPENDENCE</strong></td>
<td></td>
</tr>
<tr>
<td>22. The guideline is editorially independent from the funding body.</td>
<td>Foreword</td>
</tr>
<tr>
<td>23. Conflicts of interest of guideline development members have been recorded.</td>
<td>Foreword</td>
</tr>
</tbody>
</table>
Appendix B  Summary table – Explanation of levels of evidence
(Modified from Palmer K et al. BMJ 2008;337:1832.)

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Nature of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level A</td>
<td>At least one high-quality meta-analysis, systematic review of randomised controlled trials or a randomised controlled trial with a very low risk of bias and directly attributable to the target cancer type, or A body of evidence demonstrating consistency of results and comprising mainly well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias, directly applicable to the target cancer type.</td>
</tr>
<tr>
<td>Level B</td>
<td>A body of evidence demonstrating consistency of results and comprising mainly high-quality systematic reviews of case-control or cohort studies and high-quality case-control or cohort studies with a very low risk of confounding or bias and a high probability that the relation is causal and which are directly applicable to the target cancer type or, Extrapolation evidence from studies described in A.</td>
</tr>
<tr>
<td>Level C</td>
<td>A body of evidence demonstrating consistency of results and including well-conducted case-control or cohort studies and high-quality case-control or cohort studies with a low risk of confounding or bias and a moderate probability that the relation is causal and which are directly applicable to the target cancer type or, Extrapolation evidence from studies described in B.</td>
</tr>
<tr>
<td>Level D</td>
<td>Non-analytic studies such as case reports, case series or expert opinion or, Extrapolation evidence from studies described in C.</td>
</tr>
<tr>
<td>Good practice point (GPP)</td>
<td>Recommended best practice based on the clinical experience of the authors of the writing group</td>
</tr>
</tbody>
</table>