123 Doc EDUCATION

FOUNDATION YEARS JOURNAL

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EDITOR IN CHIEF, MICHAEL VASSALLO

Volume 3, Issue 2: Cardiology, General Practice

Contents

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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 2

Foundation Years Journal

Foundation Years Journal is an international peer-viewed journal which seeks to be the pre-eminent journal in the field of patient safety and clinical practice for Foundation Years' doctors and educators. The journal welcomes papers on any aspect of health care and medical education which will be of benefit to doctors in the foundation training grade in the UK or international equivalents. The predominant emphasis in **Foundation Years Journal** is on work related to patient safety and in health care education.

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FOUNDATION YEARS JOURNAL 2009

Volume 3, Issue 2

Volume 3, Issue 2: Cardiology, General Practice

Foundation Years Journal is the ONLY journal for Foundation Years doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month, each issue delivers practical and informative articles tailored to the needs of junior doctors. The journal closely follows the Foundation Yearssyllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning.The answers will be published in the next issue, but 123Doc will advance answers to clinical tutor subscribers so they can engage their students in the learning process. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

- **MMC CURRICULAR-BASED CONTENT** to enhance understanding of the core competencies required from future leading doctors.
- **FOCUS ON SPECIALTY-SPECIFIC CLINICAL CASES** each month to form broad subject coverage.
- **ADDITIONAL IN-DEPTH** good clinical and acute care articles aligned with the case-based discussion assessments.
- **TRAINING GUIDE FOR FOUNDATION YEAR (FY)** educators with proposed clinical cases for teaching sessions.
- **PRACTICAL AND INFORMATIVE** articles written by senior doctors and consultants.
- **EXTRA REVISION** with comprehensive assessment. Questions and Picture Quiz.

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Vol 3, Issue 3: Gastroenterology
Vol 3, Issue 4: Gynaecology, Obstetrics
Vol 3, Issue 5: Urology
Vol 3, Issue 6: Rheumatology, Orthopaedics

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FOUNDATION YEARS JOURNAL 2009

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Aim & Scope

The Foundation Years Journal is published by 123doc and is aimed at doctors in Foundation training programmes, their educational and clinical supervisors, as well as medical students and other doctors (particularly international medical graduates) who intend to start foundation training in the United Kingdom.

Journal Sections

The journal has been redesigned and various sections have been introduced to map the journal more closely to the foundation programme curriculum. You can view the curriculum from **http://www.foundationprogramme.nhs.uk/pages/home/training-and-assessment.**

The sections are the following:

1. Good Clinical Care (syllabus section 1).
This section deals with various aspects of patient management including history, examination, diagnosis, record keeping, safe prescribing and reflective practice. Articles could also refer to other aspects of care including time management, decision-making, patient safety, infection control, clinical governance, nutrition, health promotion, patient education, public health and ethical and legal issues.

2. Good Medical Practice (syllabus section 2).
Articles could be on learning, research, evidence based guidelines and audit.

3. Training and Teaching (syllabus section 3).

4. Professionalism in Practice (syllabus sections 4,5 & 6).
This section includes relationship with patients, communication skills, working with colleagues, probity, professional behavior and personal health.

5. Patient Management (syllabus section 7).
Articles should be focused on the recognition and management of the acutely ill patients, core skills in relation to acute illness, resuscitation, management of the 'take', discharge planning, selection and interpretation of investigations.

6. Practical Procedures (syllabus section 8).

7. Test Yourself
The intention is to provide a vehicle whereby trainees and educational supervisors can present original and review articles mapped against the foundation curriculum.

Submission Of Manuscript

All articles submitted to the Journal must comply with these instructions. Failure to do so will result in return of the manuscript and possible delay in publication.

Manuscripts must be submitted exclusively by email (see detailed instructions below). Manuscripts should be written in English of a sufficiently high standard that is intelligible to the professional reader who is not a specialist in the particular field. Where contributions are judged as acceptable for publication, the Editor or the Publisher reserve the right to modify the manuscripts to improve communication between author and reader. Authors whose native language is not English are strongly recommended to have their submissions checked by a person knowledgeable of the language. If extensive alterations are required, the manuscript will be returned to the author for revision.

Covering Letter

The manuscript must be accompanied by a covering letter bearing the corresponding author's signature. Papers are accepted for publication in the Journal on the understanding that the content has not been published or is being considered for publication elsewhere. This must be stated in the covering letter. If authors submit manuscripts relating to original research in the field of education, the corresponding author must state that the protocol for the research project has been approved by a suitably constituted Ethics Committee and that it conforms to the provisions of the Declaration of Helsinki (as revised in Edinburgh 2000), available at **http://www.wma.net/e/policy/b3.htm.** All investigations involving human subjects must include a statement that the subject gave informed consent and patient anonymity should be preserved.

The covering letter must contain an acknowledgement that all authors have contributed significantly and that all authors are in agreement with the content of the manuscript.

Authors should declare any financial support or relationships that may give rise to a conflict of interest.

Submitting A Manuscript

Manuscripts should be submitted by email to **(agnes@123doc.com).** We do not accept manuscripts submitted by post. Corresponding authors must supply an email address as all correspondence will be by email. Authors should use double spacing when submitting their manuscript. Two files or documents should be supplied: the covering letter and manuscript. The covering letter should mention the title, authors, their contribution, provenance, journal section where their work is to be considered (see above) and any conflict of interests. Please supply the files in Word 2003 format.

Figures should be supplied as a separate file, with the figure number incorporated in the file name. High-resolution figures (at least 300 d.p.i.) saved as jpeg files should be submitted.

Manuscript Style

Unless otherwise stated manuscripts should follow the style of the Vancouver agreement detailed in the International Committee of Medical Journal Editors' revised 'Uniform Requirements for Manuscripts Submitted to Biomedical Journals: Writing and Editing for Biomedical Publication', as presented at **http://www.ICMJE.org/.**

Abbreviations

Abbreviations should be used sparingly to facilitate reading the article by reducing repetition of long, technical terms. Initially you must use the word in full, followed by the abbreviation in parentheses. Thereafter use the abbreviation only.

Units

All measurements must be given in SI or SI-derived units.

Trade Names

Drugs should be referred to by their generic names, rather than brand names.

References

All articles must be referenced appropriately. To reference the journal please use the following abbreviation FYJ-123Doc. (The Vancouver system of referencing should be used and some examples are given below).

References should be cited using superscript Arabic numerals in the order in which they appear. If cited in tables or figure legends, number according to the first identification of the table or figure in the text.

In the reference list, the references should be numbered and listed in order of appearance in the text. Cite the names of all authors when seven or more list the first three followed by et al. Names of journals should be abbreviated in the style used in Index Medicus. Reference to unpublished data and personal communications should appear in the text only.

References should be listed in the following forms:

Journal Article

Vassallo M, Vignaraja R, Sharma JC, et al. The Impact of Changing Practice on fall Prevention in a Rehabilitative Hospital. The Hospital Injury Prevention (HIP) Study. J Am Geriatr Soc 2004; 52:335-9. Book Azeem T, Vassallo M, SamaniNJ. Rapid review of ECG interpretation. London UK: Manson Publishing 2005.

Chapter In A Book

Martin GM. Biological mechanisms of ageing. In: Grimley Evans J, Franklin Williams T eds. Oxford Textbook of Geriatric Medicine, 1st edn. New York: Oxford University Press 1992; 41-48.

Journal Article On The Internet

british Geriatrics Society position paper. Dementia ethical issues **http://www.bgs.org.uk/Publications/Position%20Papers/psn_dementia_ethics.html.**

Tables

Tables should be self-contained and complement, but not duplicate, information contained in the text. Number tables consecutively in the text in Arabic numerals. Table should be double -spaced and vertical lines should not be used to separate columns. Column headings should be brief, with units of measurement in parentheses; all abbreviations must be defined in footnotes. Footnote symbols: †, ‡, §, should be used (in that order) and *, **, *** should be reserved for P-values. The table and its legend/footnotes should be understandable without reference to the text.

Line Figures

Line figures should be sharp, black and white graphs or diagrams, drawn professionally or with a computer graphics package. Lettering must be included and should be sized to be no larger than the journal text.

Colour Figures

We encourage authors to submit colour figures and graphics that facilitate the comprehenion of the article.

Figure Legends

Type figure legends on a separate page. Legends should be concise but comprehensive - the figure and its legend must be understandable without reference to the text. Include definitions of any symbols used and define/explain all abbreviations and units of measurement. The journal accepts the following types of articles (as title please):

Case based Discussion
(CBD - Same Format As Practical Procedure & Audit Please)

These are mainly intended for inclusion in sections 1 and 5 as highlighted above and should be about 1000-1500 words long. The CBD can focus on various aspect of patient care such as presentation, treatment or prescribing. The articles should include areas that are evaluated in the case based discussion assessment tool of the foundation programme .

The manuscript should be set out in the following sections:

- Abstract: this should refer to salient points from the case being presented together with a mention of what aspects are being discussed
- Case History: this relates to theinitial presentation and should include the clinical setting, clinical problem, investigations and treatment. The history section should also include an ongoing update (eg 2 days later, a week later etc) of patient progress and management
- Discussion. This section should include a critical analysis of patient management in relation to clinical assessment, investigations, differential diagnosis, treatment, follow-up, professionalism and clinical judgement. The discussion should also include a discussion about the ongoing management issues and decisions. It is important to note that the case based discussion is not a review of a particular condition.
- Two best of 5 MCQs to be included in the Test yourself section, with answers and detailed teaching notes explaining the answers. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

Authors writing a case based discussion should not write a short history and then write an article about the condition that the patient presented with. Such information can easily be obtained from a text book and is not the scope of journal. Case based discusions written in this style will be returned to the author without being published.

Practical Procedures

Manuscripts on practical procedures should be about 1000 – 1500 words long. They should be set out in the following sections:

- History. This should describe the presentation of the patient and mention why or how the patient ended up needing the procedure.
- The procedure itself. This should include
- indications and contraindications
- explaining the procedure to the patient (including possible complications) and gaining informed consent for procedures
- preparing the required equipment, including a sterile field
- position the patient and give premed/sedation or local anaesthesia as required and involving the anaesthetist where appropriate
- safely disposing of equipment, including sharps
- documenting the procedure, including labelling samples and giving instructions for monitoring and after care
- recording complications and the emergency management of such complications when appropriate.

Adequate pictures and diagrams need to be supplied in order to make the procedure as clear as possible.

Two best of 5 MCQs for inclusion in the test yourself section, including answers and detailed teaching notes. The answers only are NOT sufficient and it should be kept in mind when writing the teaching notes that the reader may take the test questions independently from reading the article.

Audit

Manuscripts, 1500 – 2000 words long, on audit are encouraged. The journal will only publish high quality audit i.e. completed audit cycles or audits that have led to guideline development. Part 1 audits or surveys will not be accepted for publication.

Review Articles

We are interested in review articles on any aspect of the curriculum that is of relevance to our readership. They should be a maximum 3000 words long, 30 references, 250 word structured abstract, 4 tables OR figures.

We would consider reviews on any of the following:

- Good Medical Practice
- Teaching and Training
- Professionalism
- Medical reviews subject to prior discussion with the editorial team as to the appropriateness of the article

Shorter Reflective Practice Articles

We are always pleased to receive short pieces of a thoughtful nature that describes the personal or professional experiences of colleagues working with patients or their relatives. They should have a maximum of 1000 words. As suggested in the Foundation Programme Portfolio (Reflective Practice) these articles should describe:

- What made the experience memorable?
- How did it affect you?
- How did it affect the patient?
- How did it affect the team?
- What did you learn from the experience and what if anything would you do differently next time?

Some aspects to be considered in these articles are:

Communication with the patient, ethical issues, aspect of your works with colleagues, probity and honesty, personal health

Research Papers

The Foundation Years Journal would welcome research articles on Medical Education. Other research papers would be considered if thought to be of interest to the readership of the journal. Articles should be written using the following headings (title page, abstract, introduction, methods, results, discussion acknowledgements, references, tables, illustrations legends.). They should be of a maximum of 2500 words of text, plus abstract, 30 references, 3 tables or figures. Manuscripts should include a structured abstracts should have a maximum of 250 words using the headings introduction, methods, results, conclusion. The title page should contain (i) the title of the paper, (ii) the full names of the authors and (iii) the addresses of the institutions at which the work was carried out together with (iv) the full postal and email address, plus facsimile and telephone numbers, of the author to whom correspondence about the manuscript should be sent.

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THE PATIENT WITH SYNCOPE

Simon W Dubrey

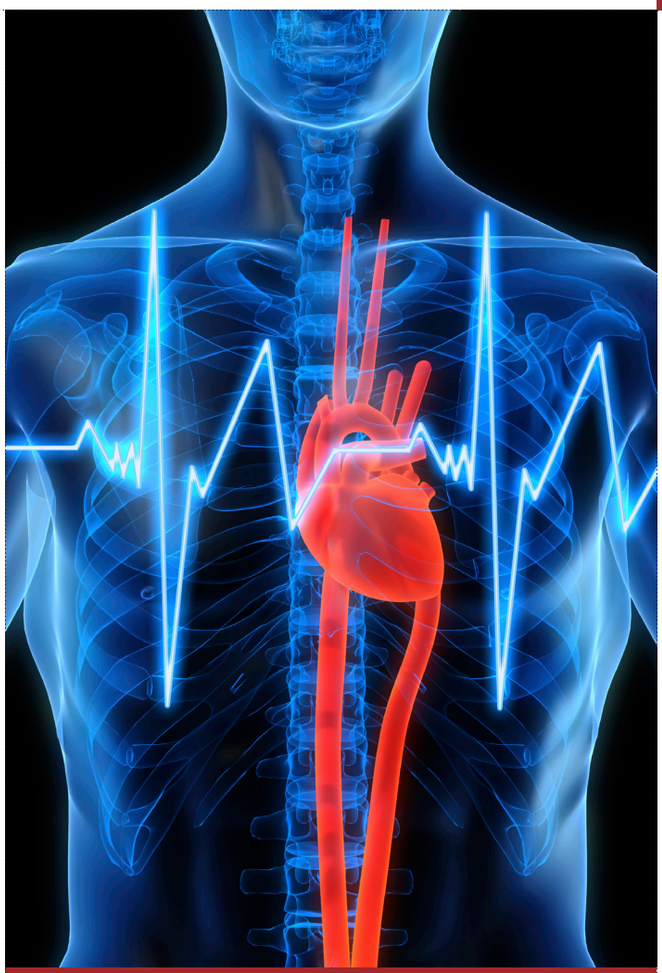
The Case

A 55-year-old black male experienced an episode of loss of consciousness while attending a funeral in Trinidad. He had flown from the UK the day before and although a little "jet-lagged" was feeling his normal self. Shortly after the ceremony he found himself in a collapsed state in the toilets of the hotel at which he was staying. He had no recollection of the events surrounding his circumstances and was not aware of any warning or aura. He assumed he had possibly had too much to drink or had simply fainted. He attended a local hospital, to check that nothing more serious was responsible. There was no shortness of breath, ankle swelling or chest discomfort.

While waiting to be seen in the A&E department he felt "giddy" for a few seconds. An electrocardiogram showed sinus rhythm at 78 beats/min with a normal axis and morphology. On a cardiac monitor there was a brief run of a broad complex tachycardia at around 190 beats per minute. Blood tests, including a full blood count, electrolytes, magnesium and calcium were normal, as was his d-dimer level. A chest X-ray and transthoracic echocardiogram were reported as normal. He settled without therapy and was discharged the next day with advice to return to the UK to seek follow-up care.

He was referred by his GP to a cardiology outpatient clinic. Analysis of a 24-hour Holter ECG, performed just prior to this appointment, revealed self-terminating runs of polymorphic ventricular tachycardia at rates of up to 230 beats/min (Figure 1). Some of these episodes corresponded to diary recordings of feeling non-specifically unwell. He had had no further episodes of collapse but felt periodically unsteady on walking. On direct questioning both his parents were hypertensive and his father had suffered with angina since the age of 49. The remaining family history included one uncle (paternal side) who had died suddenly at the age of 35 of a suspected cardiac cause.

Our patient was a non-smoker with a moderate social alcohol intake of around 20 units per week and had a physically inactive occupation as an IT consultant. Examination revealed a man of normal to muscular build, a regular pulse at 82 beats/min and a blood pressure of 140/85mm Hg. The heart sounds were unremarkable and there were no signs to support any evidence of cardiac failure. The 12 lead ECG was unremarkable, showing sinus rhythm, a normal axis and morphology. Specifically the cardiac intervals were all within normal limits and there was no evidence of an accessory pathway (i.e. delta wave or short PR interval).



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Patient Management.

THE PATIENT WITH SYNCOPE

Simon W Dubrey

What Would You Do Next?

A rhythm disturbance causing a disturbance of consciousness requires urgent investigation. As such, he was admitted from the outpatient clinic to the coronary care unit for continuous cardiac monitoring. The blood pressure was adequate to tolerate initiating a first line pharmacological therapy to try and prevent these paroxysms of ventricular tachycardia. The short acting beta blocker metoprolol was started at 12.5mg tds. In addition we commenced aspirin at 75mg daily and a prophylactic dose of subcutaneous low molecular weight heparin (LMWH).

Blood tests were again normal and this time included thyroid function tests, a fasting glucose and troponin I. A repeat transthoracic echocardiogram was unremarkable, showing no evidence of a regional wall motion abnormality or criteria for left ventricular hypertrophy. A repeat chest X-ray was reported as normal, with clear lung fields, normal cardiac silhouette and hilar structures. After 24 hours, the beta blocker was changed to a once daily preparation of bisoprolol at 5mg daily. He remained in sinus rhythm and the LMWH was stopped after 3 days.

What Are The Possible Causes Of Syncope Of This Nature?

The first presentation in Trinidad with an episode of collapse after a long airline flight would raise the likelihood of a pulmonary embolus. However, he was not breathless and the ECG and echocardiographic findings did not support this. Furthermore, the d-dimer test was normal and has its strength in being a useful “rule out” test for any significant thrombotic event.

In a male patient of this age with a family history of ischaemic heart disease and a significant rhythm disturbance the first aetiology to exclude would be ischaemic heart disease.

The normal echocardiogram has ruled out any obvious structural heart disease that might have included aortic stenosis or significant left ventricular outflow tract obstruction. It would also seem unlikely that this is a rhythm disturbance due to some form of hypertrophic or infiltrative cardiomyopathy. Additionally, all the above diagnoses are unlikely in the context of an entirely normal 12 lead ECG.

Another possible explanation could be some form of intrinsic heart disease initiating the ventricular tachycardia. One, not uncommon cause of such a presentation is arrhythmogenic right, or indeed also left, ventricular dysplasia (AVD). AVD is a structural abnormality, usually of the right ventricular free wall, in which the myocytes are replaced by a fibro–fatty material. This renders the patient liable to significant ventricular rhythm disturbances.

Conduction system disease, for example, intermittent Wolff-Parkinson-White syndrome or an ion channel disease, including the Brugada syndrome or a long QT syndrome are also possible. Although the resting ECG’s did not have features to suggest this, all these conditions can be occult and may only appear sporadically or in response to chemical challenges.

What Other Investigation Might Prove Useful?

He was referred for an urgent inpatient coronary angiogram which showed entirely normal coronary arteries and left ventricular function.

Following this, a cardiac MRI scan was ordered to obtain more detailed images of cardiac structure and the myocardium per se. The MRI scan confirmed that both ventricles were of normal size and function. The scan used the contrast agent gadolinium to detect any areas of the myocardium that might be abnormal due, for example, to fibrosis, infiltration or infarction. On these delayed contrast enhanced images, there was hyperenhancement of the anteroseptal segments of the basal portion of the left ventricle (Figure 2). This was located in an epicardial distribution which is the opposite to that seen in ischaemia, where the enhancement with gadolinium is usually endocardial. An additional feature to be detected was the presence of enlarged lymph nodes, in a pre-aortic position that had not been visible on the previous chest X-rays.

What Would You Say To The Patient?

Prior to making the diagnosis an explanation needs to be provided to the patient as to why he requires inpatient assessment. In the knowledge that the condition will undoubtedly require significant intervention either in the form of invasive studies, pharmacological or device therapy it is useful to gently introduce these ideas to the patient. “Sowing the seeds” of future management can reduce later anxieties when these treatment options are formally proposed. The patient should also be given an opportunity to ask questions.

Discussion

Based on the features to emerge from the cardiac MRI scan, a provisional diagnosis of sarcoidosis was made. Subsequent ultrasound examination of the neck identified the presence of a submandibular lymph node which could be biopsied. This confirmed the diagnosis of sarcoidosis. Histologically, sarcoid granulomas are described as non-caseating (non-necrotising) and contain epithelioid cells and large multinucleated giant cells. Interestingly, there was no evidence of further pulmonary involvement on a computed tomographic (CAT) scan of the thorax.

In order to prevent further rhythm disturbance the medication was changed to the antiarrhythmic beta blocker, sotalol at 40mg bd. An automated implantable cardioverter defibrillator (AICD) was implanted as a “backup”. This is a NICE recommendation in case of haemodynamically significant ventricular tachycardia and will ameliorate the potential to cause sudden arrhythmic cardiac death1. The disease state of sarcoidosis with heart involvement, was treated with oral prednisolone at 20mg/day.

THE PATIENT WITH SYNCOPE

Simon W Dubrey

Cardiac sarcoidosis is a relatively unusual presentation for this disease. The quoted clinical incidence of heart involvement is around 4–5%2, this figure is higher, at between 20–25%, in post-mortem studies[3]. Involvement can affect individuals of any age, gender or race. As in the case described, there is a predilection for the conduction system of the heart[4]. Heart involvement can also cause a dilated cardiomyopathy with consequent progressive heart failure. Studies have shown diastolic dysfunction, even in asymptomatic individuals with no signs of heart involvement. A restrictive picture can be the presenting feature although this is less common than an electrical or dilated-type of presentation. Significant or massive cardiac “infiltration” is very unusual. However, the most common presentation remains pulmonary infiltration. Many cases will be asymptomatic and are detected when a routine chest X-ray reveals thoracic lymphadenopathy.

Frequently, sarcoid granulomas resolve spontaneously. Cases presenting in an asymptomatic way with isolated bilateral hilar lymphadenopathy are likely to resolve spontaneously within 2 years. Symptoms at presentation mean the condition is less likely to resolve.

Unlike isolated pulmonary disease the prognosis with cardiac involvement is much worse. It is estimated that between 5–8% of patients will eventually die of their disease; respiratory failure being the principle cause[5]. In Japan, nearly 80% of patients die from cardiac involvement[6]. However, owing to improved rhythm management with pacemakers and implanted defibrillators, the primary cause of death has moved from sudden death to heart failure[7].

Sarcoidosis should be suspected in any patient, younger than expected, presenting with complete heart block or indeed heart failure[4]. Sarcoid is an infiltrative disease in character and has a predilection to involve the cardiac conduction system. Patients can develop various degrees of heart block and tachyarrhythmias. Patients are also liable to sudden death[2]. A dilated or restrictive cardiomyopathy can occur, with the left ventricle and the interventricular septum primarily involved. Sarcoid has a predilection for the base of the interventricular septum, which by virtue of the location can cause heart block or an arrhythmia. Mitral valve abnormalities, papillary muscle dysfunction, left ventricular aneurysm formation and pericardial effusions are also seen. Cor pulmonale can develop due to chronic pulmonary fibrotic disease.

1. A Reasonably Common Cause Of An Emergency Admission To Hospital Due To Syncope Occurring In A Bathroom Or Toilet Might Be:

a. A pulmonary embolic event?
b. A myocardial infarction?
c. A patient combing their hair?
d. A male patient who has gone to the bathroom to urinate?
e. A patient who looks round, over their shoulder, to talk to someone who has called to them from the adjoining room.

Syncope in bathrooms usually involves older men who have experienced micturition syncope. Frequently in the night, a patient will have a lower blood pressure due to vasodilatation. The blood pressure will fall further with the vagal effects on the heart associated with micturition. Syncope can result.

Patients are also “recovered” from bathrooms in hospitals, hotels and airports after experiencing a pulmonary embolus. In these cases, patients who are unwell, post-surgical or have undertaken a long period of travel are likely to develop venous thrombosis and experience embolic events. Emboli often arrive in the lungs but can also enter the systemic system including the coronary and cerebral circulations via intracardiac shunts (ASD or VSD). The reason for the bathroom location is that an obstructive pulmonary vascular event leads to elevated right heart pressures with a resultant congestion of the liver and bowel. In turn, patients will experience a sensation that they need to open their bowels and will seek out a bathroom facility.

Patients do not have any particular tendency to experience myocardial infarction in bathrooms. Looking over your shoulder or turning your head can cause vertebrobasilar insufficiency and disturbed consciousness but this has no particular association with bathrooms. It is sometimes reported in the elderly patient who has looked over their shoulder when manoeuvring a car out of a parked position to join the flow of traffic. Patients with the subclavian steel syndrome can have disturbances of consciousness when their arms are used in positions above their head. This might include combing their hair, painting a ceiling or even shaving. The mechanism is through a proximal subclavian artery stenosis which has increased resistance to blood flow. Blood flow can be “stolen” (occasionally blood flow is reversed) from the more distal ipsilateral vertebral artery when the arms are active. While a recognised phenomena, it is not that common.

2. The Aetiology Of Sarcoidosis Is Due To:

a. An atypical mycobacterial agent?
b. Exposure to pesticides and some chemicals used in the photographic industry?
c. No known cause has been established?
d. Deposition of abnormal proteins (protein deposition disease) in various organs and tissues?
e. An autoimmune disease?

THE PATIENT WITH SYNCOPE

Simon W Dubrey

The aetiology of sarcoidosis remains unknown, although numerous infective agents (e.g. atypical mycobacteria) and environmental exposures (e.g. insecticides and agricultural employment) have been proposed. Reports of community outbreaks and clustering of cases among working colleagues, including nurses, firefighters, US Navy personnel and neighbours seem to support a contagious aetiology. The likelihood is that there is a genetic predisposition as evidenced by such clustering, and that an as yet unknown, stimulus triggers an exaggerated immune response. Sarcoidosis occurs more commonly in monozygotic than dizygotic twins and familial "clusters" of the disease are described. No autoimmune entity has been identified. The protein deposition disease frequently affecting the heart is amyloidosis and not sarcoidosis. Both may affect the conduction system and the myocardium.

3. A Good Diagnostic Test For Sarcoidosis Is:

a. Measurement of the serum angiotensin converting enzyme level?
b. Biopsy of an affected organ, tissue or lymph node?
c. The Kveim test?
d. An echocardiogram?
e. A chest X-ray?

A biopsy will reveal the characteristic non-caseating granulomas that are the hallmark feature of sarcoidosis. The angiotensin converting enzyme (ACE) level is no longer considered reliable as a diagnostic test; this is largely because any local stimulation of macrophages leads to abnormal ACE secretion. This can occur in other granulomatous diseases. There may be a role for ACE measurement, once the diagnosis is established, in following any response to therapy. The Kveim skin test is no longer performed, largely due the inherent risk of transfer of an "infective" agent. In this test, a preparation from the spleen of a patient with sarcoidosis was injected into the skin of a patient suspected of having the disease and the cutaneous response examined. An echocardiogram can be normal despite significant cardiac involvement affecting the conduction system. A chest X-ray is always a useful test to perform to look for pulmonary involvement. A characteristic feature is bilateral hilar lymphadenopathy in around 50–80% of patients. Enlargement of the right paratracheal nodes is also not uncommon. Parenchymal infiltrates of the lungs are seen in around 25–50% of patients with a predilection for the central and upper regions of both lungs.

4. A Reasonable First Line Treatment For Hypotension Resulting In Syncope Would Be:

a. An alpha agonist (midodrine)?
b. A beta blocker (metoprolol)?
c. A serotonin uptake inhibitor (fluoxetine)?
d. Support stockings?
e. A mineralocorticoid steroid (fludrocortisone)?

Fludrocortisone is a mineralocorticoid which provides useful treatment for hypotension through its effects on salt and water retention. The usual starting dose would be around 50–100mcg daily, increased as required up to 400mcg daily. A search for any iatrogenic drug-induced cause must be excluded. Support stockings, increased fluid and salt intake are also appropriate measures. In severe cases, usually due to an autonomic neuropathy, the alpha agonist midodrine with its vasoconstrictive actions can prove very effective. However, this drug remains unlicensed and is not a first line choice. When used, midodrine is administered twice or three times daily and titrated against the blood pressure response. Beta blockers are used for vasovagal syncope which at first sight seems counter-intuitive with their hypotensive and bradycardic actions. Their beneficial action appears to be via blocking the adrenergic stimulation that occurs prior to collapse. Beta blockers reduce myocardial contractility and by so doing reduce stimulation of the cardiac and great vessel mechanoreceptors. As a result, the reflex (Bezold-Jarisch reflex) vagal stimulation of the heart and vasculature is reduced and vasovagal syncope abolished. Serotonin uptake inhibitors have also been used to treat hypotension but neither this class of drugs nor beta blockers would be considered first line therapy in the UK. Occasionally, patients will require cardiac pacemakers for severe vasovagal syncope (sometimes termed malignant vasovagal syncope) but this requires careful electrophysiological evaluation during tilt table testing.

5. The epidemiology of sarcoidosis shows us that:

a. The disease can resolve without any treatment?
b. Sarcoidosis is more common in black patients?
c. Males are more frequently affected by the disease?
d. It is an inherited disease?
e. It is a disease of middle and old age?

THE PATIENT WITH SYNCOPE

Simon W Dubrey

No single cause has been established as the cause of sarcoidosis. It is still widely, and incorrectly, believed that the incidence of sarcoidosis is higher in black populations. Sarcoidosis is recognised worldwide with a high prevalence in Sweden (64 per 100,000), Norway, Denmark and Iceland. However, the severity of the disease does appear to be greater in black populations, both in the United States and in those who have migrated to Europe. In the United States, the incidence for the white population is estimated to be 11 per 100,000. It predominantly affects a young age group, in the region of 20–30 year olds. There is a female predominance which is unexplained and while there are reports of clustering in families it is not a genetically inherited disease.

Figure Legends

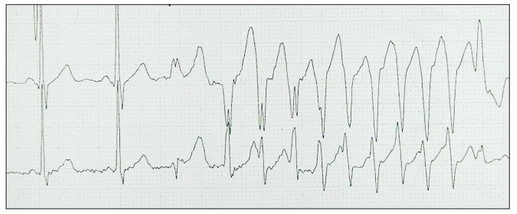

Figure 1: Rhythm strip showing sinus rhythm with a self-terminating run of polymorphic ventricular tachycardi

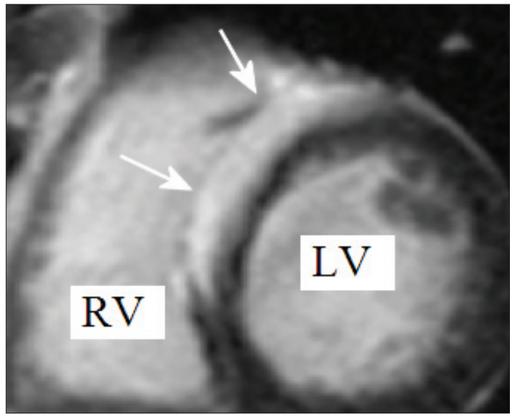

Figure 2. MRI scan showing a short axis view of both ventricles. The white arrows illustrate the area of late gadolinium enhancement due to sarcoid involvement. LV, left ventricle, RV, right ventricle.

Answers

Answer To Question 1: The best answer is d.

Answer To Question 2: The best answer is c.

Answer To Question 3: The best answer is b.

Answer To Question 4: The best answer is e.

Answer To Question 5: The best answer is a.

References

1. National Institute for Health and Clinical Excellence (NICE). Implantable cardioverter defibrillators (ICDs) for arrhythmias. January 2006. Available at: **(www.nice.org.uk/TA095).**

2. Sharma O, Maheshwari A, Thaler K. Myocardial sarcoidosis. Chest, 1993, 103:253–258.

3. Ratner SJ, Fenoglio JJ Jr, Ursell PC. Utility of endomyocardial biopsy in the diagnosis of cardiac sarcoidosis. Chest, 1986, 90:528–533.

4. Fleming HA. Sarcoid heart disease. Br Med J, 1986, 292:1095–1096.

5. Chesnutt AN. Enigmas in sarcoidosis. West J Med, 1995, 162:519–526.

6. Iwai K, Sekiguti M, Hosoda Y, et al. Racial difference in cardiac sarcoidosis incidence observed at autopsy. Sarcoidosis, 1994, 11:26–31.

7. Sekiguchi M, Yazaki Y, Isobe M, Hiroe M. Cardiac sarcoidosis: diagnostic, prognostic, and therapeutic considerations. Cardiovasc Drugs Ther, 1996, 10(5):495–510.

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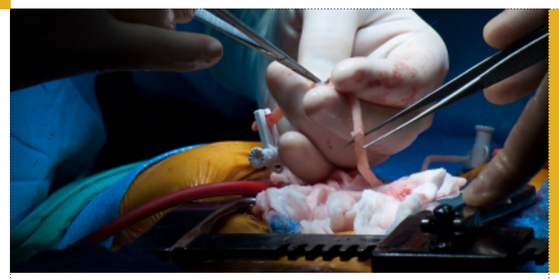
PRESCRIBING: SECONDARY PREVENTION AFTER MI

Jessica Webb, Laura Cochrane and Pitt O Lim

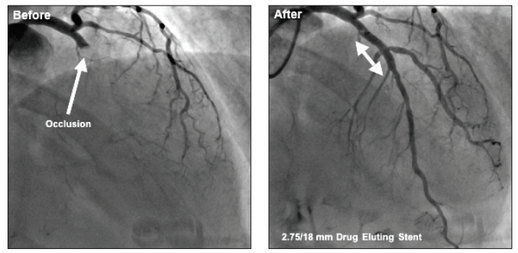


A 40-year-old lorry driver presented to the Emergency Department an hour after onset of central chest pain. He was a smoker with a family history of coronary artery disease. Good Clinical Care.

Case History

A 40-year-old lorry driver presented to the Emergency Department an hour after onset of central chest pain. He was a smoker with a family history of coronary artery disease. His 12 lead electrocardiogram confirmed an acute anterolateral ST elevation myocardial infarction (STEMI). Following intravenous morphine for analgesia and anti-emetics, he underwent immediate primary percutaneous coronary intervention (PCI) with preoperative loading doses of aspirin and clopidogrel, as well as an intravenous bolus dose of heparin.

Coronary angiogram revealed occlusion of the left anterior descending (LAD) artery with clots. All remaining coronary arteries were patent and without significant disease. The LAD was successfully wired and recanalised with balloon angioplasty. The lesion was then stented with a single drug eluting stent. Following the procedure the patient was transferred to the Coronary Care Unit where he was started on a beta blocker, a statin and an angiotensin converting enzyme inhibitor (ACE inhibitor). His peak creatine kinase was under 1000IU and an echocardiogram revealed preservation of left ventricular systolic function with mild apical and anteroseptal hypokinesia. He was discharged home after 3 days and referred for outpatient cardiac rehabilitation. Follow-up echocardiogram at 6 months revealed a normal heart without any wall motion abnormalities.



What Is Secondary Prevention?

Myocardial Infarction (MI) is an acute thrombotic event resulting in cardiac muscle cell death, and hence possible left ventricular systolic dysfunction, on a background of unstable atherosclerotic coronary artery disease. Current secondary prevention measures in the UK target different aspects of MI pathophysiology using a combination of pharmacological intervention (antiplatelet therapy, beta blockade, ACE inhibition and statin treatment), cardiac rehabilitation and lifestyle changes. Lifestyle measures post MI consist of smoking cessation, exercise and dietary changes. The introduction of the National Service Framework (NSF) and the National Institute Clinical Excellence (NICE) guidelines assist in achieving consistency in post MI management across the UK.

Why Is Secondary Prevention Important?

In the UK over 250,000 individuals have an MI annually. Further MIs among these high-risk individuals is not uncommon with approximately 25% of all men and 33% of women experiencing a second MI within 6 years. Although the mortality rate from coronary heart disease has fallen since the 1970s, it is still in excess of 100,000 deaths per annum in the UK, the third highest in Western Europe after Finland and Ireland[1].

Coronary heart disease has major economic implications. Lost days at work are estimated to cost over 5 billion pounds to the economy and nearly 2 billion pounds for the NHS per annum[2]. The need for secondary prevention following MI has been highlighted by the ASPIRE study (Action on Secondary Prevention through Intervention to Reduce Events)[3]. This study reports potential to reduce secondary ischaemic events in those with coronary heart disease by effective lifestyle intervention, rigorous risk factor management and appropriate use of preventative drugs. Secondary prevention in coronary heart disease is likely to result in major health and socio-economic benefits.

PRESCRIBING: SECONDARY PREVENTION AFTER MI

Jessica Webb, Laura Cochrane and Pitt O Lim

Why Give Aspirin?

Low dose aspirin mediates an antiplatelet effect by irreversibly inhibiting thromboxane through cyclo-oxygenase-1 enzyme (COX-1), resulting in inhibition of platelet aggregation and therefore clot formation. The evidence for aspirin dates back to the 1980s with the seminal ISIS 2 trial[4]. In this study patients admitted within 24 hours of the onset of symptoms or suspected MI were recruited. These patients were allocated randomly to receive thrombolytic therapy in the form of streptokinase or placebo, and half of all patients were further allocated randomly to receive oral aspirin or placebo in a 2x2 factorial study design. In the first 35-days post MI, 26 fewer deaths per 1,000 were recorded for those treated with aspirin and 29 fewer deaths per 1,000 for those randomised to streptokinase. More recent meta-analyses of antiplatelet therapy in high-risk patients identified 12 trials on patients with recent MI (20,006 patients with mean duration 27 months)[5]. These demonstrated that treatment with aspirin post MI resulted in 36 fewer vascular deaths per 1,000 patients treated or a number to treat (NNT) of 28 patients to avoid one death, and also reduced the risk of re-infarction. Low dose aspirin, 75mg daily, is therefore a cheap and clinically effective drug that should be taken by all post MI patients unless contraindicated by allergy or serious gastro-intestinal side effects.

When Is Clopidogrel Needed?

Clopidogrel is a thienopyridine derivative that inhibits ADP dependant platelet activation. This disrupts the glycoprotein IIb/IIIa pathway which ensures that platelet aggregation is prevented. The loading dose is either 300mg or 600mg depending on the urgency, and the daily maintenance dose is 75mg. It is used in conjunction with aspirin in patients presenting with an acute coronary syndrome. For patients following an MI without ST elevation (NSTEMI) or coronary drug eluting stent implantation, its use is recommended for 12 months. In patients implanted with coronary bare metal stents in the setting of stable angina, those presenting with a STEMI treated with thrombolysis or primary PCI with bare metal stents, clopidogrel is recommended for at least 1 month.

In the patient described, a drug eluting stent was chosen in view of the fact that the occlusive lesion was less than 3mm diameter and longer than 15mm in length, as recommended by NICE guidelines. In these cases, dual antiplatelet therapy is recommended for at least 12 months to prevent stent thrombosis.

The evidence for clopidogrel is from the CURE6 and PCI CURE7 trials. CURE was a double-blind randomised trial comparing clopidogrel with placebo in patients with angina or NSTEMI. Patients were randomly allocated to either a loading dose of clopidogrel followed by 75mg clopidogrel daily (6,259 patients) or placebo (6,303 patients). They all received aspirin and other concurrent medications. Clopidogrel treatment significantly reduced ischaemic events (MI, cardiovascular death and stroke) by 20% (9.3% versus 11.4%) compared to standard therapy alone. The NNT to prevent one cardiovascular death or non fatal MI was 48 patients over 12 months.

Why Give Statins?

Statins reduce cholesterol by inhibiting HMG-CoA reductase which is the rate limiting enzyme of the mevalonate pathway of cholesterol synthesis. Inhibition of this enzyme in the liver upregulates the low density lipoprotein (LDL) receptors. This results in increased clearance of LDL cholesterol from the circulation with a reduction in blood cholesterol levels. The statin effect can be seen within 1 week with a maximal effect at 4 to 6 weeks.

The landmark 4S (Scandinavian Simvastatin Survival Study) study[8] looked at 4,444 patients aged between 35 and 70 years with angina, previous MI and cholesterol levels between 5.5–8.0mmol/L. The absolute cardiac mortality was reduced with simvastatin treatment from 8.5% to 5.0%, making the NNT around 30 (30 patients would need to be treated to prevent one death) with a median follow-up period of 5.4 years. Simvastatin produced a relative risk reduction of 42% in cardiac deaths, 34% in all major coronary events, 37% in non-fatal myocardial infarction and 37% in the need for coronary revascularisation. The NNT for the above composite end point was in the region of 8–59 depending on the subgroup (men NNT 22–33, women NNT 25–59, diabetics 8–32, over 65 years old 21–34). The beneficial effects of simvastatin in a more diverse group of patients with significant risk factors for coronary artery disease is further strengthened by the results of the Heart Protection study[9].

Simvastatin 40mg is the recommended first line therapy post MI and the lipid profile should be repeated 4–6 weeks after drug initiation. If necessary more potent statins, such as atorvastatin or rosuvastatin, prescribed if cholesterol targets are not achieved. Combination therapy with ezetimibe and fibrates is not uncommon in patients with familial hypercholestrolaemia. In the above patient, a statin was started irrespective of the baseline cholesterol level as there is current opinion that following a cardiac event, even a low baseline cholesterol is too high. Furthermore, statins are thought to have beneficial pleiotropic effects beyond cholesterol lowering, especially that which improves vascular endothelial function. Statin treatment is generally well tolerated but common side effects are muscle pain and liver dysfunction, rarely myositis could occur hence the need for routine blood tests following drug initiation.

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Should All Patients Have Beta Blockers?

There are several known subtypes of beta receptors but the ß1 receptor has been shown to be present in the heart and in the kidney. Stimulation of ß1 receptors induces positive chronotropic and inotropic effects on the heart, as well as increasing renin release from the kidney. The sum total of these effects result in cardiac ischaemia by increasing myocardial oxygen demand and impeding coronary blood flow. Therefore cardioselective ß1 beta blockers, such as bisoprolol, atenolol and metoprolol, will have favourable effects by blocking these in the setting of cardiac ischaemia. This is achieved largely by reducing the heart rate, systolic blood pressure, myocardial contraction and hence the extent of cardiac ischaemia and infarct size peri MI.

There is evidence supporting the use of beta blockers in patients with MI. Trials such as ISIS 110, 11 proved a reduction in death, infarct size, re-infarction and cardiac arrest. The current NICE guidelines recommend long-term treatment with beta blockers (6 months to 4 years) in unselected MI patients, which is based on trials reporting a 1.2% annual risk reduction with beta blocker treatment with a 23% reduced odds of death compared to placebo[12]. Cross trial comparisons have suggested that the higher the level of beta blockade, as measured by heart rate reduction relative to the control group, the greater the benefit.

There are some patients, however, who should only be prescribed beta blockers with caution or after specialist input. Accepted contraindications are bradycardia (heart rate under 50bpm), hypotension (systolic blood pressure under 90mm Hg), severe heart failure requiring intravenous diuretics or inotropes, cardiogenic shock, asthma or reactive airways disease necessitating bronchodilator therapy and second or third degree atrioventicular block.

Which Patients Benefit From Ace Inhibitors?

The rationale for inhibition of the renin-angiontensin-aldosterone axis includes experimental and clinical evidence of a favourable impact on ventricular remodelling, improvement in haemodynamics and reductions in heart failure. ACE inhibitors lower arteriolar resistance hence afterload reduction, and increase venous capacity therefore a reduction in central venous pressures reducing diastolic ventricular interaction. This in turn promotes left ventricular diastolic filling with subsequent increase in cardiac output, stroke work and volume, and increased natriuresis via neurohormonal modulation. The use of maximal tolerable dose of ACE inhibitors in post MI patients is justified because it improves clinical outcomes, independent of the blood pressure lowering effect of ACE inhibitors. Additional benefits include prevention of diabetic nephropathy, congestive heart failure and prophylaxis of cardiovascular events. Such therapy requires careful and gradual titration of dose to prevent hypotension and there is an additional need for monitoring plasma electrolytes within the first 2 weeks of therapy to detect potential renal dysfunction and hyperkalaemia.

The key ISIS 4 trial13 included over 58,000 patients with suspected MI randomised to receive 1 month of captopril treatment or placebo. This study also assessed the efficacy of oral nitrates and intravenous magnesium. Captopril was associated with a significant reduction in mortality with an absolute difference of 2.2 fewer deaths per 1,000, with a survival benefit maintained for 1 year despite the fact that the trial medication was only given for 1 month. The efficacy of captopril was even greater in those presenting with a history of MI or with heart failure.

The current NICE guidelines recommend the use of ACE inhibitors in the acute setting of MI, in patients with left ventricular dysfunction, those with signs of heart failure and in patients with stable coronary disease and normal left ventricular function. The American Heart Association (AHA) similarly supports long-term ACE inhibition in STEMI patients with reduced left ventricular ejection fraction, for those with hypertension, diabetes or chronic kidney disease. Aldosterone antagonists can be added where there is clinical evidence of heart failure during or post MI.

What Is Omacor & When Should It Be Prescribed?

The observation of low incidence of coronary artery disease among Inuit Indians, whose diet is rich in n-3 polyunsaturated fatty acids (PUFA), spurred further research between dietary fat intake and cardiovascular disease. There are now countless over-the-counter n-3 PUFA preparations but omacor is the only prescribed formulation available that contains 1g of highly purified n-3 PUFA per capsule with 46% of eicosapentaenoic acid (EPA) and 38% of decosahexaenoic acid (DHA). It has a limited cholesterol lowering effect but it is thought to have plaque stabilising and antiarrhythmic effects.

The GISSI-P14 trial included 11,324 post MI patients with 89% patients with their first MI. Patients were randomly assigned to receive supplements of omacor, vitamin E, omacor and vitamin E or no supplement. Omacor treatment over 3.5 years significantly reduced the rate of the cumulative endpoint of all-cause death, non-fatal MI and non-fatal stroke. There was a 20% (8.4% versus 10.6%, NNT 45) reduction in cardiac mortality. More recent studies using other preparations, however, failed to replicate the GISSI-P results. Currently NICE recommend considering starting Omacor 1g od within 3 months of an MI for a duration of up to 3 years when dietary intervention is insufficient.

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How Long Should These Medications Continue?

Duration of these treatments following MI is currently unclear. Polypharmacy is associated with poor drug compliance, particularly as most of these patients are asymptomatic, and many of these drugs have significant side effects. It would be unrealistic to expect a 40-year-old man to be on five or more drugs lifelong following an uncomplicated MI. For aspirin this should be taken indefinitely unless not tolerated. Clopidogrel is given for a defined period of time. In the patient described, clopidogrel was advised for 1 year, as dual antiplatelet therapy with aspirin is essential when a drug eluting coronary stent has been implanted. Statin therapy was suggested to continue long term as the atherosclerotic process is a dynamic one. However, it is important to reassess this with elderly patients on an individual basis as there is a lack of evidence supporting their general use in this age group. There is also a need to interrupt treatment in young women planning for a family.

Patients who tolerate their medications should probably continue them beyond 1 year. This is based on the assumption that the benefits from the trials persist long term. However most trials only follow-up their patients for a limited length of time and rarely for more than 5 years post event. Also, most trials supporting the use of these medications were performed in the pre-routine coronary angioplasty era. Managing acute MI and secondary prevention have come a long way from the prolonged bed rest in the pre-thrombolysis era, just over 2 decades ago.

The advent of coronary angioplasty has certainly changed the acute management of ischaemic events as the priority is getting the patient to he cardiac catheter laboratory as quickly as possible. In a patient who has all patent coronary arteries following coronary angioplasty and preserved left ventricular systolic function, a less dogmatic approach is desirable balancing drug tolerance and quality of life free from drug side effects. Nevertheless, certain groups of patients would benefit more than others from continuing medical therapy. These include patients with impaired left ventricular systolic function, and multiple unresolved risk factors for coronary artery disease.

What Role Does Cardiac Rehabilitation Play?

Cardiac rehabilitation can improve survival in coronary heart disease patients. It is the coordinated sum of interventions required to ensure the best physical, psychological and social conditions to enable the patient to resume or preserve optimal functioning in society. It has now evolved to include overall risk factor and behavioural modification. Practically it facilitates opportunities to review patients' diets, exercise, lose weight if needed, stop smoking and helps ensure compliance with medications. The current NICE guidelines are that the Mediterranean-style diet should be recommended with more fish, bread, vegetables and fruit and less meat, butter and cheese. Exercise should be recommended to increase the patients' exercise capacity, which is broadly physical activities for 20 to 30 minutes a day to the point of mild breathlessness.

Patients who smoke should be advised to stop and offered assistance to do so if required. Current NICE guidelines set out a structured approach including local support, behavioural therapy, nicotine replacement and buproprion for smoking cessation. Good Clinical Care.

What Is Lifestyle Modifications?

Patients who smoke should be advised to stop and offered assistance to do so if required. Current NICE guidelines set out a structured approach including local support, behavioural therapy, nicotine replacement and buproprion for smoking cessation.

Weight loss should be encouraged particularly in the obese patient with an offer of support and guidance to achieve this when needed. Often a combination of rehabilitation and the introduction of an exercise programme is productive in the initial stages. The preferred approach for patients with BMI over 30kg/m^2 emphasises a three part strategy including calorie restrictions, structured physical activities and behavioural therapy. Pharmacotherapy may be appropriate for some individuals and bariatric surgery can be considered in the morbidly obese. The NICE guidelines recommend pharmacological treatment only when behavioural and dietary modifications have been started and evaluated, or if weight loss has begun to plateau.

Patients should have their blood pressure checked regularly and ensure that they are not hypertensive. The initiation of a structured exercise programme, healthy diet and reduced alcohol consumption helps to achieve acceptable blood pressure targets. In diabetic patients, good glycaemic control is thought to reduce the risk of ischaemic events.

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How Great Is The Drug Therapy Effect For The Individual Patient?

Randomised control trials provide proof of efficacy for drug treatment relevant to their specific study population. The patients recruited are usually highly selected from a large pool of patients, these study patients are well motivated and are followed-up closely to ensure study drug compliance. The benefit of a drug treatment, therefore, is highly specific to the study population in question (i.e. the "average" patient in the study). Extrapolating this information to the "real world" individual patient is problematic especially with polypharmacy, and many trials becoming rapidly outdated with advances in health care technology, such as coronary angioplasty and stenting.

Patient education and counselling is key if drug compliance is to be maintained. The question of what are the added benefits of taking 3 instead of 4 or 5 drugs to the individual patient is without precise answers. The oft quoted relative risk reduction by 20% to 30% of an outcome, such as death, for each drug for which many trials are designed to accomplish, appears impressive but has very little relevance to individual patients. NNT (100/absolute risk reduction) which takes into account the actual benefits does offer some understanding of the size of drug treatment effects. However, if for example, taking simvastatin over 5 years gives an NNT of 30, this would appear to mean that 29 patients could potentially be taking the drug without benefits over that time period[8]. If the statin effect is linear then over 150 patients per year will be taking the drug just to benefit a single patient, not a particularly good investment for the individual patient. This, however, only applies to the "hard" end point of death, not taking into account the "softer" end points of MI and revascularisation.

Another way of presenting study information in a more meaningful way to the individual patient is illustrated by the CONSENSUS I study which assessed the efficacy of enalapril in patients with severe heart failure with a follow-up period of 10 years to near total mortality in both treatment arms. Enalapril was found to extend survival by 8.7 months on average over 10 years or 26 days per year. Simvastatin treatment as reported in the 4S study offers over 2 months of "life extension (absolute risk reduction x follow-up/100)" or 13 days for each patient per year. In comparison, omacor offers 8 days of life extension per year. Captopril treatment in the ISIS 4 study in contrast only extends life by 2 extra days in the first year and by 1 month with longer follow-up[15]. The COURAGE trial demonstrated that in coronary heart disease large ischaemic myocardial burden is associated with higher mortality with medical therapy[16]. There is some evidence that revascularisation with PCI in such patients improves survival. The small SWISS II study[17] followed up 200 patients with 2 or less coronary vessel disease over 10 years comparing medical therapy against PCI. Cardiac death in the PCI group was 3:96 (3%) versus 22:105 (21%) in the medical group. Hence PCI offers an average life extension of 1.8 years over 10 years or about 2 months of life extension per year.

It is important not to underestimate favourable effects of cardiac rehabilitation, in particular smoking cessation. A meta-analysis of 20 studies[18] conducted in the 1960s/1970s involving 12,603 subjects with a follow-up of 5 years, documented a mortality rate of 18.5% among 5,659 subjects who stopped smoking against 27.1% of 6,944 in those who continued smoking. This means that stopping smoking can extend life by 1 extra month every year, an effect that is far greater than many drugs combined.

When Can Patients Go Back To Work?

Most patients are advised to take 1 month off work after an MI. In the case of the above patient who worked as a lorry driver it is important to review the DVLA guidelines. After an acute coronary syndrome, NSTEMI or STEMI treated with successful coronary angioplasty patients are permitted to drive if they are a group one license holder (car and motorcycle) 1 week after the procedure and there is no need to contact the DVLA. This is only if no other urgent revascularisation is planned, left ventricular ejection fraction is over 40% and there is no other disqualifying condition. For group two license holders (LGV, PCV – lorry drivers and bus drivers) all acute coronary syndromes disqualify the licensed driver from driving for 6 weeks. The DVLA should to be contacted and relicensing can only be permitted after further evaluation. This means that the above patient will need to be assessed either with an exercise test or another functional test before driving can be resumed.

Questions

1. What medications are prescribed for post MI secondary prevention? (True/False)

a. Clopidogrel
b. Eplerenone if there is clinical LV dysfunction
c. Propanolol
d. Ezetimibe
e. Long acting oral nitrates

2. What are the contraindications to beta blockers? (True/False)

a. Gastro-oesophageal reflux disease
b. Severe asthma
c. Acute heart failure
d. Diabetes
e. Atrial fibrillation

3. What are the current NICE guidelines for secondary prevention post MI? (True/false)

a. Omacor to be prescribed 3 months after MI if dietary intake is insufficient
b. Clopidogrel for a minimum of 4 weeks after a STEMI
c. Beta blockers should be given to all patients
d. ACE inhibitors for patients with or without abnormal LV function
e. Aspirin to be avoided in patients with previous peptic ulcer disease

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References

1. WHO, Death Rates from CHD, Men and Women Aged 35-74, Selected Countries. **(http://www.who.int/cardiovascular_diseases/en/)** 2007.

2. BHF, Incidence of Myocardial Infarction. **(http://www.heartstats.org/homepage.asp)** 2008.

3. Bowker TJ, et al. A British Cardiac Society survey of the potential for the secondary prevention of coronary disease: ASPIRE (Action on Secondary Prevention through Intervention to Reduce Events). Heart, 1996, 75(4):334–342.

4. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Lancet, 1988, 2(8607):349–360.

5. Baigent C, Collins R, Peto R. Article makes simple errors and could cause unnecessary deaths. BMJ, 2002, 324(7330):167.

6. Yusuf S, et al. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. N Engl J Med, 2001, 345(7):494–502.

7. Mehta SR, et al. Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: the PCI-CURE study. Lancet, 2001, 358(9281):527–533.

8. Randomised trial of cholesterol lowering in 4,444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). Lancet, 1994, 344(8934):1383–1389.

9. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet, 2002, 360(9326):7–22.

10. Randomised trial of intravenous atenolol among 16,027 cases of suspected acute myocardial infarction: ISIS-1. First International Study of Infarct Survival Collaborative Group. Lancet, 1986, 2(8498):57–66.

11. Yusuf S, et al. Beta blockade during and after myocardial infarction: an overview of the randomised trials. Prog Cardiovasc Dis, 1985, 27(5):335–371.

12. NICE, Secondary prevention Post MI 2007.

13. ISIS-4: a randomised factorial trial assessing early oral captopril, oral mononitrate, and intravenous magnesium sulphate in 58,050 patients with suspected acute myocardial infarction. ISIS-4 (Fourth International Study of Infarct Survival) Collaborative Group. Lancet, 1995, 345(8951):669–685.

14. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico. Lancet, 1999, 354(9177):447–455.

15. Tan LB, Murphy R. Shifts in mortality curves: saving or extending lives? Lancet, 1999, 354(9187):1378–1381.16. Prasad A, Rihal C, Holmes DR Jr. The COURAGE trial in perspective. Catheter Cardiovasc Interv, 2008, 72(1):54–59.

17. Erne P, et al. Effects of percutaneous coronary interventions in silent ischemia after myocardial infarction: the SWISSI II randomised controlled trial. JAMA, 2007, 297(18):1985–1991.

18. Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. Jama, 2003, 290(1):86–97.

Answers

1. What medications are prescribed for post MI secondary prevention?

a. True **b.** True **c.** False **d.** False **e.** False

2. What are the contraindications to beta blockers?

a. False **b.** True **c.** True **d.** False **e.** False

3. What are the current NICE guidelines for secondary prevention post MI?

a. False **b.** True **c.** True **d.** True **e.** False

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SUPERVISING AN EXERCISE TREADMILL TEST

James SG Signy, Stephen F Copeland, Janet A Scott and Mark Signy

An abnormal treadmill test will make the diagnosis easier.
Patient Management.

The Test

The exercise treadmill test, usually performed to a graduated routine known as the Bruce protocol, has been a standard test for investigating chest pain and ischaemic heart disease (IHD) for many years and is still very widely used. As a Foundation Year colleague you may be asked as part of your programme in cardiology or acute medicine to assist with the supervision of these tests. When used properly they are easy to do, informative and a very useful contribution to the diagnosis and subsequent treatment of patients with ischaemic heart disease. In this short paper, we have tried to summarise the indications for treadmill testing, the pretest contraindications to look out for, the test itself, what constitutes a positive or negative test and the possible complications. It is by necessity a brief run-through and further reading is recommended below. The section about the test itself has been written by a senior cardiac physiologist and a consultant nurse. When supervising a test, you should always be accompanied by a well-qualified cardiac physiologist or nurse and do not feel any qualms about asking them for help and advice. Many of them have supervised literally thousands of tests.

Four Case Histories: Is A Treadmill Exercise ECG An Appropriate Next Step?

Case History 1

A 55-year-old man, mildly overweight with a past history of moderate smoking and alcohol intake, and a family history of an 80-year-old uncle with ischaemic heart disease attends the rapid access chest pain clinic with a 2-month history of slightly atypical chest pain, variable in onset, but sometimes exertional. No abnormalities on clinical examination and resting ECG is normal.

Case History 2

A 76-year-old man with a long history of smoking and a past medical history of diabetes and hypertension is admitted as an emergency through A&E with a 3-day history of deteriorating chest pain both on exertion and at rest. Examination shows him to be hypertensive, resting ECG demonstrates dynamic ST segment depression in the anterior leads, and a 12-hour troponin measurement is raised into the significant range. His pain persists after admission.

Case History 3

A 19-year-old female is seen in the clinic with a 2-year history of lower chest/upper abdominal discomfort which is not exertional, but also not clearly related to food. She has occasional symptoms of "missed beats", but no other relevant history. Examination and resting ECG are normal.

Case History 4

A 45-year-old man is admitted via A&E with a 3-day history of chest pain without obvious precipitating factors. Examination is normal, serial resting ECGs show no significant abnormalities and 12-hour troponin is within the normal range. However, he remains very anxious and gets intermittent recurrent pain.

Case History 1 Review

Yes, this is a standard type of case presenting to a rapid access chest pain clinic. The story is quite good for ischaemic heart disease, although his risk factors are rather general. An abnormal treadmill test will make the diagnosis easier, while a normal test to high workload, while not entirely ruling out ischaemia, would be prognostically relatively reassuring.

Case History 2 Review

No, this man has a high-risk acute coronary syndrome (risk factors of age, continuing/deteriorating symptoms, dynamic abnormal ECG changes and a raised troponin). An exercise ECG will add very little to the diagnosis (which is unstable angina/ischaemic heart disease until proved otherwise), and would be significantly high-risk investigation. This man needs an urgent inpatient coronary angiogram (ideally within 48–72 hours) and may well need percutaneous coronary intervention or bypass grafting as an emergency.

Case History 3 Review

Probably not, the difficulty here is that the pretest probability of underlying ischaemia is pretty low, which makes the likelihood of a positive result being a false positive increasingly high. The story, normal examination and resting ECG make a diagnosis of IHD unlikely and her symptoms should be investigated by other means in the first instance. If her symptoms persist and no explanation is found cardiac investigation could be reconsidered, but alternative investigations may be more discriminatory than a treadmill test.

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Case History 4 Review

This man fits into the lower-risk category of acute chest pain admissions, but in view of the persistent pain an exercise treadmill before discharge may add to the confidence of the diagnosis. While a normal exercise test even in these circumstances does not entirely rule out IHD, it is prognostically reassuring if completed to a good workload and along with the other low-risk factors would allow him to be discharged with some degree of confidence. Outpatient follow-up is warranted and investigation is indicated if his symptoms do not settle.

The exercise ECG is a far from perfect test even for the indications where it is widely used. The problem is that it is neither sensitive enough nor specific enough, leading to both false positives and false negatives (2). This is a particular problem when the pretest likelihood of underlying abnormality is low, as this leads to situations where an abnormal result may be as likely to be a false-positive as a true-positive. In the absence of complete reliability of a negative result, the test's usefulness in these circumstances is very limited, it has not added to the diagnosis at all. Recent work has suggested that in many cases the additional information obtained is not significantly more than from a good history, examination and resting ECG (1). Unfortunately it is usually the best (or only) non-invasive test that we have available and it should be used with care to avoid wasteful use of time and resources (including Foundation Year doctors).

Indications[2, 4]

- Aid to diagnosis in ambulant patients with chest pain and low/ intermediate probability of coronary disease (e.g. rapid access chest pain clinic).
- Aid to diagnosis in some patients with other exertional symptoms.
- Risk stratification after myocardial infarction (or acute cardiac event).
- Estimate of success (or risk stratification after) of PCI, bypass grafting or drug treatment.
- Estimate of exercise tolerance/risk stratification for occupational reasons, etc. (e.g. DVLA/pilots).
- Demonstration of ischaemia (or its absence) in patients with known coronary disease (e.g. to aid decision on revascularisation by PCI or surgery or with residual disease in a second artery after single vessel PCI).

May also be used in patients with some cardiomyopathies, arrhythmias, heart failure and other exertional symptoms, although these indications are outside the scope of this paper.

Before The Test

There is no point in offering a treadmill test if the patient is unable physically (or occasionally mentally) to do the test. "We have lost count of the number of wheelchair/crutch-bound patients who arrive in our department having been referred for treadmill testing."

All patients should be given a full description of the test and the risk/benefits of having it. As a minimum, verbal consent should be obtained. Unless there is a specific trust policy about this, the default position should be always to obtain written consent.

There should always be two trained professionals supervising the test, and both should be fully trained in resuscitation.

Except in urgent situations, digoxin should be stopped if possible, ideally for a week or more, as it makes interpretation of the ECG results very difficult.

There is some argument about stopping beta blockers for the test. This is usually required for 48 hours before a DVLA test, for example, but the ACC/AHA guidelines[4] suggest that "for routine exercise testing it appears unnecessary for physicians to accept the risk of stopping beta blockers before testing when a patient exhibits possible symptoms of ischaemia or has hypertension" (this of course applies to most of the patients we exercise).

Contraindications[2, 4]

Treadmill testing is contraindicated either absolutely or relatively:

- In the first few days after acute myocardial infarction (although is sometimes used towards the end of an inpatient stay for early prognostic testing.
- In patients with high-risk acute coronary syndromes (assessed by age, ECG changes, troponin rise), severe uncontrolled hypertension (BP >220 systolic), severe heart failure, severe aortic stenosis, aortic dissection and uncontrolled arrhythmias.
- In known severe underlying coronary disease (especially left main stem disease. Any extra information is unlikely to be justified by the risk in disease where intervention is essentially mandated by the underlying anatomy).
- Severe outflow tract obstruction (e.g. severe hypertrophic cardiomyopathy, although sometimes used in assessment of this).
- Acute myocarditis, pericarditis or systemic infection.

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Possible Complications/Adverse Effects:

- Myocardial infarction/mortality (various estimates – overall risk about 1:10,000 for all comers)
- Cardiac arrest
- Arrhythmias including sustained VT and VF
- Dizziness/collapse
- Hypotension
- Hypertension
- Falls/injury

All these are fortunately extremely rare if patients are appropriately screened pretest. However, they must be properly managed and it is essential that all attendants are fully up to date with their formal resuscitation training.

When To Stop A Test:

- At the patient's request; due to pain, breathlessness or inability to cope
- Onset of severe chest pain
- Limiting breathlessness
- Significant dizziness, lightheadedness or syncopal symptoms
- Cardiac arrest/collapse
- Development of neurological symptoms or signs
- Significant fall in BP or excessive rise in BP
- ST segment depression > than 2–3mm (positive test – no need to continue) or new bundle branch or high-grade AV block
- Significant ST elevation
- Ventricular fibrillation, ventricular tachycardia or salvos, or frequent ventricular ectopic/couplets (particularly increasing frequency)
- Significant supraventricular arrhythmias

What Is A "Positive" Test?

- Horizontal or downward sloping ST depression (by convention >2mm). Even upsloping ST depression can sometimes be abnormal. Widespread ST changes are an additional significant finding.
- Rapid onset (stage 1–2) of ST changes, and persistence into recovery stage, is highly significant.
- Early onset of symptoms of chest pain (and breathlessness).
- Exertional drop in systolic BP.
- Completing stage 3 of the Bruce protocol without symptoms or ECG changes is a relatively reassuring sign.
- Various algorithms are used to increase sensitivity and specificity. The best of these is probably the Duke score (3) which combines exercise time, ECG changes and ST depression to give a score which is easily obtained from a nomogram and gives useful prognostic information.

Supervising/Running The Test

In accordance with SCST/BCS Guidelines[5] the exercise tolerance test should always be done in a well-lit and ventilated room (air conditioning is a bonus). The most common method of exercise used is the treadmill (the bicycle may induce early leg fatigue). Treadmill systems may offer a number of varying exercise protocols but the main ones used are the Bruce and Modified Bruce protocols. (The gentler modified Bruce protocol is particularly useful in patients with poor mobility and in the relatively elderly with reduced exercise capacity). The room should be large enough to accommodate the patient, the treadmill, the supervising staff and any equipment to perform resuscitation procedures or for any other emergencies, with easy access for an emergency trolley. Ideally the exercise room should be located close to the cardiac care or cardiac wards. Patient information leaflets (usually sent out before the test) should provide advice on the tests, procedures and information about remaining on medication or stopping medication before the test.

All resuscitation equipment should be checked before the patient's arrival. UK resuscitation guidelines recommend that defibrillator, suction and oxygen equipment should be present in the exercise room. Cardiac arrest alarms/phones should be checked and all necessary documentation should be available and checked before the test, this includes a detailed signed request form and the patient's notes. There needs to be two qualified supervisors present and immediately available for the test.

All patients should have been seen and examined by a physician before the test to ensure there are no contraindications to testing. Suitability for exercise testing should be formally indicated by the completed request form[5]. On the day of the exercise test the supervisor should check with the patient for any changes in symptoms and clinical findings and also confirm medication instructions have been followed. Skin preparation prior to exercise tolerance testing is extremely important to ensure good signal quality during the test. Chest hair should be removed (usually shaved) and ECG electrode sites wiped with an alcohol-based substance and skin abraded in order to lower skin resistance. The patient's history can be checked and further explanation of the test can be undertaken during skin preparation.

Good communication is essential before, during and after the test in order to continually assess the patient's symptoms, condition and ability to perform/continue the test. This information is vital as it will need to be submitted in the final report for subsequent review and for decisions on the patient's future care pathway. Patients should always be encouraged to undertake the maximum exercise they possibly can in order to achieve their target heart rate. Most ECG recording systems are computer-based and should allow continuous monitoring. Usually there are several lead configurations to choose from when analysing ECG data but all systems should, as a minimum, contain the facility to display and record a 12 lead ECG. Modern systems provide a computerised analysis of the pretest exercise and recovery ST segments, and a hard copy printout must be included in the final investigation report[5].

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Computerised averaged ST analysis is often useful in eliminating movement artefacts but observation/comparison of this data along with raw data is very important to ensure accuracy. It is important to note any differences in the final report; relying on the averaged ST analysis alone may lead to incorrect interpretation. Automated blood pressure machines are usually linked to the exercise computers but may be inaccurate at high levels of exercise due to increased noise, and in these circumstances manual BP should be measured.Electrode positions need to be modified from standard resting ECGs as it is impractical to place limb leads on the arms and legs during vigorous exercise.

The electrodes are placed in the following sites:

- Chest electrodes – as per standard 12 lead ECG
- Right arm electrode – upper right side of torso, 2cm below clavicle
- Left arm electrode –upper left side of torso, 2cm below clavicle
- Right leg electrode – lower right side of torso, halfway between costal margin and iliac crest
- Left leg electrode – lower left side of torso, halfway between costal margin and iliac crest

A standard resting ECG should ideally be compared with the modified resting 12 lead ECG before testing commences and any difference due to the modified positions noted. The patient should be informed about methods of mastering the treadmill (and stopping it/getting off). A demonstration of the technique is sometimes useful to reassure the patient before starting. A resting ECG and BP should be recorded before starting the exercise test. The patient's ability to perform each stage should be assessed; encouragement is reassuring to the patient and helpful in obtaining maximum heart rate. Heart rate and blood pressure are continuously monitored and the supervisor should constantly monitor the ECG signals, looking for any ST segment changes, significant arrhythmias or other abnormalities. Observation of the patient is, of course, necessary throughout all phases of testing.Indications for stopping the test have already been referred to above, but all tests should ideally be symptom limited or maximal (to target heart rate), whichever is the sooner. On termination of the test the patient will require a recovery period, usually a minimum of 6 minutes or until the heart rate and blood pressure have returned to the baseline values and any ECG changes have returned to normal.

The supervisor should always assess the patient's condition before discharge ensuring the patient is stable and that all parameters have returned to their baseline values. A comprehensive report needs to be generated to aid subsequent consultant decisions for the patient's further investigation and treatment. This is usually generated by the system but will require a hand written or typed free text report in addition.

The table below (reproduced from the BCS/SCST 2008 guidelines[5]) indicates the minimum requirements for inclusion in a full exercise tolerance test report.

- Indication for the investigation
- The protocol used
- The resting ECG appearances
- Any deviation from the last clinical assessment by a physician
- Any deviation from the pretest instructions
- Whether medication was terminated for the purpose of the investigation
- Total exercise times and recovery times
- % of predicted maximum heart rate achieved
- Whether the test was maximal or sub-maximal relating to the maximum heart rate
- Reason for termination
- When a significant event developed, reached a peak and resolved
- If appropriate, the maximum ST changes in which lead and when
- Patient's cardiac symptoms – what, when, association with other abnormalities (e.g. ECG changes)
- Details regarding any arrhythmias
- Patient's other symptoms, for example, claudication, dizziness, fatigue
- A comment on the patient's appearance before, during and after the exercise test
- Whether computerised ST analysis was reliable or otherwise

If in any doubt about a patient's condition or any findings, the responsible clinician/consultant should be informed prior to discharging the patient from the test department.

Multiple Choice Questions

1. The following are recognised indications for a treadmill exercise test:

a. To help confirm suspected is chaemic heart disease.
b. Assessment of cardiac function and exercise tolerance.
c. Prognosis following myocardial infarction.
d. Assessment of high-risk unstable angina.
e. Assessment of exercise induced arrhythmias .

2. In exercise testing:

a. Upsloping ST depression is more significant than downsloping.
b. Early and prolonged ST depression is a strongly positive sign.
c. Mortality is less than 1:1,000,000.
d. A positive treadmill picks up 75% of patients with significant CAD.
e. False positives are least common in middle-aged women.

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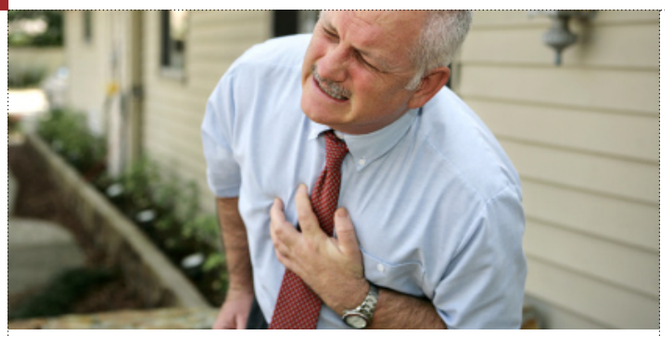

3. A 62-year-old man is admitted with crushing central chest pain. ECG shows widespread ST depression and the troponin is positive. Which of the following are appropriate?

a. Pain relief.
b. Aspirin and clopidogrel.
c. Treadmill exercise testing for prognosis.
d. 24-hour tape.
e. Coronary angiogram.

4. The following commonly indicate coronary disease on exercise testing:

a. Early ST/T segment depression.
b. inability to complete stage 6 of the Bruce protocol due to exhaustion.
c. Hyperventilation.
d. Chest pain in stage 2.
e. Systolic hypertension.

References

1. Sekhri N, Feder GS, Junghans C, Eldridge S, Umaipalan A, Madhu R, Hemingway H, Timmis AD. Incremental prognostic value of the exercise electrocardiogram in the initial assessment of patients with suspected angina: cohort study. BMJ, November 2008, 13(337):A2240.

2. Hill J, Timmis A. Exercise tolerance testing (review). BMJ, 4 May 2002, 324(7345):1084–1087.

3. Mark DB, Hlatky MAS, Harrell FE, Lee KL, Califf RM, Pryor DB. Exercise treadmill score for predicting prognosis in coronary artery disease. Ann Intern Med, 1987, 106:793–800.

4. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines. J Am Coll Cardiol, 16 October 2002, 40(8):1531–1540 (full guideline on ACC website).

5. Clinical guidance by consensus: recommendations for clinical exercise tolerance testing. Society for Cardiological Science and Technology/British Cardiovascular Society 2008. (Guideline document.)

Abramson B. Electrocardiography in suspected angina. BMJ, November 2008, 13(337):A2340.

Gershlick AH, de Belder M, Chambers J, Hackett D, Keal R, Kelion A, Neubauer S, Pennell DJ, Rothman M, Signy M, Wilde P. Role of non-invasive imaging in the management of coronary artery disease: an assessment of likely change over the next 10 years. A report from the British Cardiovascular Society Working Group (review). Heart, April 2007, 93(4):423–431.

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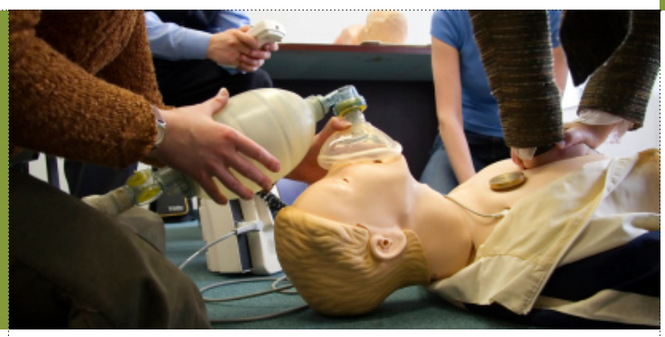
ASSESSMENT OF RESUSCITATION SKILLS OF FOUNDATION YEAR 1 HOSPITAL DOCTORS

D McCluskey, N Sayee, M Connolly, P Toner and M Stevenson

All FY1 doctors in a major UK teaching hospital were asked to perform two, 3-minute sessions of CPR on a skills reporter manikin separated by a 5-minute rest period, one session using a compression-to-ventilation ratio of 15:2 and the other using a ratio of 30:2. Good Medical Practice.



Abstract

Aims Of Study

To assess the ability of Foundation Year 1 (FY1) doctors to perform effective cardiopulmonary resuscitation (CPR) and determine what factors might influence their performance.

Methods

All FY1 doctors working in a major UK teaching hospital were asked to participate in this study. These individuals were regularly on-call for acute medical emergencies including cardiac arrest. They were asked to perform two, 3-minute sessions of CPR on a skills reporter manikin separated by a 5-minute rest period, one session using a compression-to-ventilation ratio of 15:2 and the other using a ratio of 30:2. Their performance was assessed both objectively, by measuring the depth of chest compressions and subjectively by a panel of five advanced life support (ALS) instructors who reviewed tracings of each CPR session.

Results

A total of 34 out of 40 (85%) FY1 doctors working in the hospital participated in the study. The objective results show that males performed significantly better than females using both the 15:2 and 30:2 ratios. The male FY1 doctors performed equally well using both 15:2 and 30:2 ratios while the female doctors appeared to be better when using 15:2.

When using the 30:2 ratio, doctors with a BMI greater than the mean for the group, performed significantly better than their colleagues who had a lower BMI.

Statistical analysis of the subjective assessment of CPR performance by five experienced ALS instructors showed that males performed equally well using either ratio, male performance was better than that of their female colleagues and females were better when using a ratio of 15:2. Body mass index seemed to be an important factor and correlated with chest compression depth. Females with a low BMI performed less well when using a ratio of 30:2. Overall, expert opinion significantly favoured the 15:2 ratio for the FY1 doctor group.

Conclusions

CPR performance of FY1 doctors showed that males and those females with a BMI of >24 were satisfactory when using the recommended Resuscitation Council Guidelines. Females with a BMI <24 appeared to be better at CPR when using a ratio of 15:2. FY1 doctors should be fully assessed before they perform CPR at in-hospital cardiac arrests. Those found to be ineffective should be given remedial instruction until they are shown to be competent. Consideration should be given to factors (such as gender and BMI) when deciding what ratio of compression to ventilation an individual rescuer should perform to optimise patient outcome.

Introduction

Foundation Year 1 (FY1) doctors are usually the first medical staff responders at in-hospital cardiac arrests. Their ability to perform effective cardiopulmonary resuscitation (CPR) is essential for a successful outcome. Not only will inadequately performed CPR compromise patient survival1 but also, of the patients that do survive, the majority will have poor neurological recovery, which results in significant impairment in quality of life[2].

In an effort to improve survival and post resuscitation quality of life, the European Resuscitation Council Guidelines 2005 placed greater emphasis on the rescuer's performance of chest compressions[3,4] and recommended using 30 rather than 15 chest compressions per CPR cycle. However, a number of studies have highlighted the problem of rescuer fatigue during the performance of 30 chest compressions[5,6,7] which may adversely affect overall CPR performance.

As a group, FY1 doctors are expected to be highly proficient at CPR. These individuals are usually young, healthy adults who have received CPR instruction on a number of occasions during their undergraduate training. In addition, most hospitals insist that all junior medical staff undergo a refresher training course in CPR before taking up an FY1 post.

It is known that survival figures after in-hospital cardiac arrest are poor[8]. This may be due to a variety of factors including severity of pre-existing cardiac disease, comorbidity and multiple drug therapy. However, it is vitally important that FY1 doctors are truly proficient in CPR if cardiac arrest patients in hospital are to have the optimal chance of survival.

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Aims & Objectives

1. To objectively and subjectively assess FY1 doctors' ability to perform CPR.

2. To determine whether CPR performance is affected by the ratio of chest compressions: ventilations used, gender or BMI of the FY1 doctor.

Materials & Methods

All 40 FY1 doctors were employed and actively working in a large UK teaching hospital, and were asked to volunteer for the study. All had received CPR training on at least two occasions as undergraduate medical students and had undertaken a CPR training course, which was a compulsory requirement, prior to starting employment in the hospital trust, less than 3 months before the study.

As FY1 doctors they were expected to act as first medical responders at in-hospital acute medical emergencies including cardiac arrest.

Subjects were asked to perform CPR using a "Laerdal Resusci-Anne Skills Reporter Manikin" (Laerdal Medical, Norway). Subjects were randomly allocated to perform 3 minutes of CPR using a compression to ventilation ratio of either 15:2 or 30:2. Following a 5-minute rest, a further 3 minutes of CPR was performed using the other compression to ventilation ratio. Group A (18 FY1 doctors) used a ratio of 15:2 while Group B (16 FY1 doctors) used 30:2 for their initial 3-minute period of resuscitation.

The complete tracing of each individual performance was saved on to a laptop computer by a single investigator. Chest compression depths (mm) obtained from the tracings were measured and recorded by a second investigator who was blinded to the age, gender, BMI and study group to which the subjects belonged.

Statistical analysis of the crossover trial was performed using the Hills and Armitage method[9]. The Mann-Whitney U Test[10] was used to analyse the data on chest compression depths when comparing subpopulations of different gender, BMI and ratios used. McNemara's Test[11] was used for analysis of the subjective data recorded by the 5 ALS instructors who viewed the individual session tracings. For all statistical analysis, a p value of <0.05 was taken as significant.

For the purpose of this study we assessed adequacy of CPR performance by the percentage of chest compressions of depth ≥38mm. We judged a 3-minute period of resuscitation to be effective when >80% of all compressions given were of a depth ≥38mm.

Statistical analysis was carried out comparing:

1. male versus female using ratio of 15:2
2. male versus female using ratio of 30:2
3. 15:2 versus 30:2 ratio with male and female FY1 doctors combined
4. 15:2 versus 30:2 for male FY1 doctors
5. 15:2 versus 30:2 for female FY1 doctors
6. BMI<24 versus BMI>24 when using ratio of 15:2
7. BMI<24 versus BMI>24 using ratio of 30:2
8. BMI<24 versus BMI>24 using both compression ratios combined

In addition to the objective study, all CPR tracings were inspected independently by five experienced advanced life support (ALS) instructors who were asked to classify each tracing as either:

1. Effective (i.e. likely to result in neurologically intact patient survival)
or
2. Ineffective (i.e. unlikely to result in neurologically intact patient survival)

The ALS instructors had no knowledge of the occupation, age, gender or BMI of those performing the CPR, nor the study group to which subjects belonged.

Since it was possible that tracings of borderline performance could result in some difference of opinion, it was decided that, for a period of resuscitation to be deemed either effective or ineffective, a minimum of four ALS instructors had to be in agreement.

Results

Of the 40 FY1 doctors working in the hospital, 34 (85%) agreed to participate in the study. Of these 18 were male and 16 female, the age range was 23–35 years (mean 24years) and the mean BMI was 24 (see Table 1).

Seven of the 34 subjects (21%) reported that they had performed CPR as part of an in-hospital acute medical emergency since appointment as an FY1 doctor, less than 3 months earlier.

Objective Results

As stated above, we assessed CPR performance as adequate when >80% of all chest compressions given during a 3-minute period of resuscitation, were of depth ≥38mm.

Gender

Using the 15:2 ratio, 88.9% of males and 44% of females (p = 0.008) performed effective resuscitation. This gender difference was more marked when subjects used the 30:2 ratio with 83.3% of males and only 25% of females (p = 0.005) being deemed adequate (see Table 2). Males performed equally well using 15:2 or 30:2 (88.9% and 83.3% respectively p = 0.5). While the results for the female FY1 doctors did not reach statistical significance (p = 0.12) they tended to be better when using a ratio of 15:2 rather than 30:2 (44% compared with 25%).

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At the 15:2 ratio, all males except 3 (when using 15:2) and all except 4 (when using 30:2) achieved 100% of compressions ≥38mm. Females performed less well at both ratios with much fewer achieving 100% of compressions at either ratio (see Figure 1).

Although the sample sizes were small, when we compared males at the two ratios we found no statistical difference (see Table 2). Four males performed better using the 15:2 ratio, 5 were better when using 30:2 and 9 showed no difference in performance.

Of the 16 female doctors, 8 performed better using the 15:2 ratio, 5 were better using 30:2 and 3 were equally good at both ratios. Because of the small sample size (16 subjects) the difference did not quite reach statistical significance (p = 0.12).

BMI

Eighty-two per cent of the FY1 doctors with BMI>24 compared to 53% of doctors with BMI<24 performed effective CPR using the 15:2 ratio. However, this did not reach statistical significance (p = 0.126).

Using the 30:2 ratio, subjects with a BMI of >24 showed significantly better CPR performance than those with a lower BMI (76% compared with 35% respectively p = 0.045) see Table 3. When we compared CPR performance by all doctors (gender and ratio combined) we found that BMI made a significant difference with 74% of doctors with BMI>24 and only 44% of doctors with BMI<24 performing >80% of compressions ≥38mm (p = 0.03).

Subjective Results

As stated earlier, we only considered those CPR session tracings when four or more expert ALS trainers were in agreement and deemed a CPR session either "effective" or "ineffective".

Gender

Because the sample size of the subpopulations was small, levels of significance could not be achieved. However, there were clearly differences between the male and female performance when using different compression-to-ventilation ratios (see Table 4). More females were rated "effective" when using 15:2 rather than 30:2. Taking the group as a whole, and using McNemara's test[11] on the mismatches (i.e. those subjects who were deemed to succeed under one and only one regime) the results showed 7 mismatched pairs. In every single case the 15:2 regime was considered effective and the 30:2 ratio ineffective. McNemara's test recorded a two-sided p value of 0.016 which is highly statistically significant with expert opinion favouring the 15:2 regime.

BMI

As stated above with respect to gender, the sample sizes of males and females of differing BMI were too small to reveal statistically significant results. The subjective results, however, would indicate that BMI has little influence on CPR performance of the male doctors but may influence the performance of the group as a whole and female doctors, especially when using the 30:2 ratio (see Table 5).

Discussion

Adequate chest compression depth is required to ensure optimal cardiac output and therefore coronary and cerebral perfusion pressures, and is arguably the most useful indicator to the quality of CPR. This study demonstrates that there are large variations in the quality of CPR, in terms of chest compression depth, as preformed by FY1 doctors. Multiple factors can affect CPR performance. In this study we investigated the impact that gender, BMI and the ratio of chest compressions to ventilations used.

The objective results of this study clearly show that male FY1 doctors perform better than their female counterparts when using both the 15:2 and 30:2 ratios. There was no significant difference in CPR performance of males using 15:2 and 30:2 although female FY1 doctors tended to be better using the 15:2 regime.

When using the 15:2 ratio, there was no statistical difference in performance of doctors with regards to BMI. However, using the 30:2 ratio, doctors with BMI>24 performed better than their colleagues who had BMI<24. A possible explanation for these results is that males are more likely to be stronger than females, usually have a greater BMI than females and because of their increased muscle mass are less likely to fatigue as quickly. Although the sample sizes were small, the subjective results fully support our objective findings and indicate that males perform more effective CPR than females when using the current European Resuscitation Guidelines (30:2), females perform deeper chest compressions when using a ratio of 15:2 and females with a BMI<24 deliver suboptimal compressions when using a ratio of 30:2.

Rescuer fatigue is more likely to occur when using 30:2 due to the increased number of compressions and absence of muscle recovery time available during the ventilation cycles. A previous study found that although the 30:2 ratio was more exhausting, the 30:2 technique delivered more chest compressions and, as in the combined doctors results of this study, the overall quality of compressions remained unchanged7. However, the investigators did not specifically assess the impact of gender on CPR performance. Sanders et al. has suggested that an increased rate of chest compression is associated with a better outcome. They compared the neurological outcome in pigs following induced cardiac arrest and found better neurological recovery at higher rates of compression and lower rates of ventilation[12].

In our study, when FY1 doctors were assessed as a group, overall there was no statistical difference in the percentage of doctors performing chest compressions ≥38mm between 15:2 and 30:2. This confirms a findings of Conrad et al.[13] who found no statistical decline in chest compression depth and rate when comparing 15:2, 30:2 or 50:2 ratios using male and female subjects.

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While we have demonstrated that male gender and an increased BMI are associated with an increased percentage of chest compressions ≥38mm, previous studies have investigated the impact other factors have on chest compressions, such as the position of the manikin[14], whether or not a backboard for the patient is being used[15] and the affect different mattresses have on chest compression depth[16]. With so many factors affecting the depth of chest compressions and therefore the quality of CPR, it is essential to optimise these variables to provide the best quality compressions.

Conclusions

This study shows that FY1 doctors perform effective CPR as judged by the depth of chest compressions ≥38mm. However, both the gender and body mass index of the FY1 doctor influence the effectiveness of performance, especially when a chest compression-to-ventilation ratio of 30:2 is being used.

Male FY1 doctors perform equally well using both 15:2 or 30:2 ratio while females are more effective when using a ratio of 15:2.

FY1 doctors with a BMI greater than 24 are capable of more effective CPR, as judged by the depth of chest compressions, when using a ratio of 30:2.

We would recommend that FY1 doctors CPR performance should be carefully assessed before they are on-call for in-hospital cardiac arrests. Those found to be unable to perform adequately deep chest compressions should be given remedial instruction until they are judged fully competent. Our findings indicate that individual rescuers resuscitation performance is likely to be different to their colleagues and therefore a standard recommendation of the ratio of compressions to ventilations does not necessarily equate to optimal performance. If doctors, and other groups in the population, are being trained in resuscitation, consideration should be given to factors such as gender and BMI when deciding what ratio of compression to ventilation the individual rescuer should perform to optimise patient outcomes.

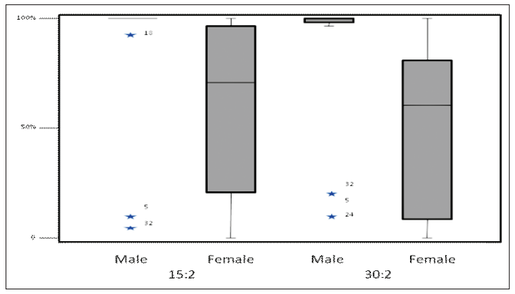


Figure 1: Plots of individual male and female FY1 doctors performance of CPR using two chest compression to ventilation ratios.

Sex	Number	Mean Age (Years)	Mean Height (CM)	Mean Weight (KG)	Mean BMI	Previous Active CPR
Male	18	24.9	180.9	77.8	25.3	5
Female	16	23.9	167.0	63.3	22.6	2
Total	34	24.4	174.0	70.6	24.0	7

Table 1: Summary of study population characteristics.

Ratio	Male	Female	P Value
	N = 18	N = 16	
15:2	88.9%	44%	0.008*
30:2	83.3%	25%	0.005*
P value	0.51	0.12	

Table 2: Male and female FY1 doctors using two different CPR ratios. Percentage achieving >80% of compressions of depths of ≥38mm.

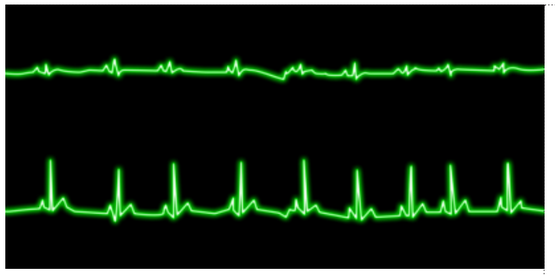
Ratio	BMI>24	BMI<24	P Value
	N = 17	N = 17	
15:2	82%	53%	0.126
30:2	76%	35%	0.045*
P value	Ns	Ns	

Table 3: All FY1 doctors. Comparison of those of BMI >24 with those of BMI<24. Percentage achieving >80% of compressions of depth ≥38mm.

	Ratio 15:2			Ratio 30:2		
	Male N = 18	Female N = 16	Both N = 34	Male N = 18	Female N = 16	Both N = 34
Effective	15 (83%)	7 (43%)	21 (62%)	13 (72%)	2 (13%)	15 (44%)
Ineffective	2 (11%)	8 (50%)	10 (29%)	3 (17%)	9 (56%)	13 (38%)

Table 4: Number and percentage of CPR sessions rated either "effective" or "ineffective" by ≥4 of the five ASL instructors for male and female doctors.

D McCluskey, N Sayee, M Connolly, P Toner and M Stevenson



	Ratio 15:2						Ratio 30:2					
	BMI>24			BMI<24			BMI>24			BMI<24		
	M	F	M F	M	F	M F	M	F	M F	M	F	M F
Effective % Age	11 92	2 40	13 77	4 66	5 46	9 53	10 83	1 20	11 65	3 50	1 9	4 24
Ineffective % Age	0 0	3 60	3 18	2 33	5 46	7 41	0 0	3 60	3 18	3 50	7 64	10 59

Table 5: Number and percentage of CPR sessions rated "effective" or "ineffective" by ≥4 of the five ASL instructors for male and female doctors with a BMI above or below 24 (the mean BMI for the study population).

References

1. Van Hoeywenghen RJ, Bossaert LL, Mullie A, Calle P, Martens P, Buylaert WA, Delooz H. Quality and Efficiency of Bystander CPR. Resuscitation, 1993, 26(1):47–52.

2. Yi HJ, Kim YS, KO Y, Oh SJ, Kim KM. Factors Associated with Survival and Neurological Outcome After CPR of Neurosurgical ICU Patients. Neurosurgery, 2006, 59(4):838–845.

3. Handley AJ, Koster R, Monsieur K, Perkins GD, Davis S, Bossaert L. European Council Guidelines for Resuscitation 2005 Section 2. Adult Basic Life Support and the use of Automated External Defibrillators. Resuscitation, 2005, 67(1):17–23.

4. Nolan JP, Deakin CD, Soar J, Bottiger BW, Smith G. European Council Guidelines for Resuscitation 2005 Section 4. Adult Advanced Life Support. Resuscitation, 2005, 67(1):539–586.

5. Ochoa FJ, Ramalle-Gormara E, Lisa V, Saralegui I. The Effect of Rescuer Fatigue on the Quality of Chest Compressions. Resuscitation, 1998, 37:148–152.

6. Ashton A, Mc Cluskey A, Gwinnutt CL, Keenan AM. Effect of Rescuer Fatigue on Performance of Continuous External Chest Compressions Over Three Minutes. Resuscitation, 2007, 74:113–118.

7. Deschilder K, de Vos R, Stockman W. The Effect on Quality of Chest Compressions and Exhaustion-Ventilation Ratio of 30:2 Verses 15:2 During CPR – A Randomised Trial. Resuscitation, 2007, 74:113–118.

8. Peberdy MA, Kaye W, Ornato JP. Cardiopulmonary Resuscitation of Adults in the Hospital: A Report of 14, 720 Cardiac Arrests From the National Registry of CPR. Resuscitation, 2003, 58:297–308.

9. Hills M, Armitage P. The Two Period Cross Over Clinical Trial. British Journal of Clinical Pharmacology, 1979, 8:7–20.

10. Siegel C, Castellan J. Non Parametric Statistics for the Behavioural Sciences, 2nd edn. McGraw-Hill International Editions, section 6.4, pp. 128–137.

11. Siegel C, Castellan J. Non Parametric Statistics for the Behavioural Sciences, 2nd edn. Mc Graw-Hill International Editions, p 79.

12. Sanders AB, Kern KB, Berg RA, Hilwig RW, Heindenrich J, Ewy GA. Neurological Outcome With Four Different Types of Ventilation to Chest Compressions Ratios. Ann Emerg Med, 2002, 40(6):553–562.

13. Conrad AB, Soreide E, Torsteinbo TH, Lexow K, Nilsen OB, Sunde K. Quality of Chest Compressions During 10min of Single – Rescuer Basic Life Support With Different Compression: Ventilation Ratios in a Manikin Model. Resuscitation, 2008, 77(1):95–100.

14. Chi CH, Tsou JY, Su FC. Effects of Rescuer Position on the Kinematics of CPR and the Force of Delivered Compressions. Resuscitation, 2008, 76(1):69–75.

15. Anderson LO, Isbye DL, Rasmussen LS. Increasing Comprssion Depth During Manikin CPR Using a Simple Backboard. Acta Anaesthesiol Scand, 2007, 51(6):747–750.

16. Perkins GD, Benny R, Giles S, Gao F, Tweed MJ. Do Different Mattresses Affect Quality of CPR? Intensive Care Med, 2003, 29(12):2330–2335.

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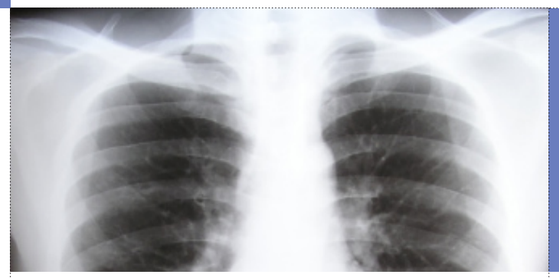
CARDIOVERSION

Roger Hayward



A 69-year-old practicing dentist was referred by his GP with a 6-month history of mild exertional dyspnoea and a newly detected systolic murmur.
Practical Procedures.

Case History

A 69-year-old practicing dentist was referred by his GP with a 6-month history of mild exertional dyspnoea and a newly detected systolic murmur. He was still able to play 18 holes of golf using a motorised trolley and had no symptoms at rest.

He had a past history of ex-smoking, hyperlipidaemia on simvastatin, troublesome psoriasis on topical steroids and mild prostatism on alfuzosin (an alpha blocker).

He was mildly overweight but looked well despite extensive guttate psoriasis, in sinus rhythm at 80/minute with a BP of 138/76, a normal venous pressure and a grade 2 crescendo pansystolic murmur at the apex. There was a grade 1 basal ejection systolic murmur but a normal carotid upstroke. There were no other murmurs, no third sound and the lungs were clear with no peripheral oedema.

A 12 lead ECG revealed sinus rhythm with a normal PR interval, a biphasic P wave in lead V1 and no significant abnormalities.

Blood analysis revealed a total cholesterol of 3.56mmol/L, LDL cholesterol of 1.88mmol/L and HDL cholesterol of 1.02mmol/L. Haematology was normal with normal hepatorenal and thyroid function and a raised level of NT-proBNP of 271pg/mL (normal <125).

Echocardiography revealed good left ventricular function with mild hypertrophy and grade 2 mitral regurgitation due to posterior mitral leaflet prolapse into a slightly enlarged left atrium. The aortic valve appeared normal. Right ventricular function was good with mild tricuspid regurgitation and a calculated right ventricular pressure of 45mm Hg.

MCQs

1. What is the diagnosis?
2. Is this condition related to psoriasis?
3. What is the significance of the right ventricular pressure?
4. Does this patient need antibiotic cover for dentistry, etc?
5. What is the likely outcome for this patient?

The patient was informed of the diagnosis and given an outline of the likely course of events. He was advised that he should avoid heavy lifting, rest when dyspnoea became apparent and report any change in symptoms. He was commenced on gentle diuretic treatment with indapamide, but vasodilators were not started at this time.

Nine months later he reported increased shortness of breath. He was then in sinus rhythm with a BP of 140/72, a raised venous pressure, pansystolic apical murmur and scanty basal lung crackles.

Echocardiography revealed still good left ventricular function, grade 3 mitral regurgitation with posterior mitral leaflet prolapse, grade 3 tricuspid regurgitation and a calculated right ventricular peak pressure of 65mm Hg. The left ventricular end-diastolic dimension was 62mm, the end-systolic dimension was 38mm.

MCQs

6. How is mitral regurgitation classified on echocardiographic criteria?
7. What is the significance of these left ventricular measurements?
8. What is the significance of the increased tricuspid regurgitation and peak right ventricular pressure?
9. What medical options are available for this patient?
10. What surgical options are available for this patient?

The patient was commenced on a loop diuretic combination (co-amilofruse 40/5) once daily, and arrangements were made for cardiac catheterisation and coronary angiography. While awaiting this procedure, he developed sudden palpitation and shortness of breath.

He was comfortable despite a ventricular rate of 145/minute, a BP of 125/80 with signs of mitral regurgitation and pulmonary congestion. An ECG revealed atrial flutter with a superior flutter wave vector and 2:1 A-V block. Following treatment he reverted to normal sinus rhythm.

MCQs

11. How should this arrhythmia be treated?
12. Is immediate cardioversion required?
13. What anti-embolic anticoagulation is necessary?
14. Why did this arrhythmia occur?
15. Does this episode have implications for the planning of cardiac surgery?

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Cardiac catheterisation confirmed good left ventricular function, severe mitral regurgitation into a slightly dilated left atrium, normal coronary arteries and a peak pulmonary artery pressure of 65mm Hg.

Within 2 days, he underwent open heart surgery involving repair of the prolapsing posterior mitral leaflet plus mitral and tricuspid valve annuloplasty. He also had a Maze procedure to the left atrial insertion of all four pulmonary veins.

Post-operative progress was good and he was discharged in sinus rhythm on warfarin, co-amilofruse 20/2.5 and omeprazole.

After 3 months he was still in sinus rhythm and was switched from warfarin to aspirin but atrial flutter recurred 10 months after the heart operation, accompanied by shortness of breath and evidence of mild mitral regurgitation.

MCQs

16. Why has this further arrhythmia occurred?
17. Should a further attempt at rhythm control be made by cardioversion?
18. Should atrial flutter in future be managed by rate control only?
19. What anticoagulation regime should be used?
20. What are the long-term implications?

He was successfully cardioverted to sinus rhythm using 200 joules energy delivered via a right anterior to left posterior electrode position, and remains well on warfarin plus low dose sotalol 6 months later.

Cardioversion: History

Since the discovery of electricity in the 18th century, for example, using the Leyden Jar in 1745, the effects of electricity on the body have fascinated both scientists and the general public.

In the late 19th century, Swiss scientists, possibly prompted by a wish to understand the effects of lightening injury to livestock, found that they could convert ventricular fibrillation into normal rhythm in animals by direct cardiac stimulation.

The first successful direct current cardioversion procedure on a man was carried out in 1947 by Claude Beck, a surgeon in Cleveland, Ohio, who administered an electrical shock directly to the fibrillating heart exposed by open thoracotomy.

In 1956 Zoll and colleagues1 reported successful external cardioversion from ventricular fibrillation in man, and in 1962 Lown described cardioversion of atrial fibrillation using an external synchronised capacitor2.

Physiology Of Cardioversion

All tachyarrhythmias are treatable by cardioversion, including atrial fibrillation and ventricular fibrillation. The majority are based on a re-entry mechanism, involving circus movements of electrical impulses within the cardiac chamber.

There are three theories about the way cardioversion terminates arrhythmias and restores sinus rhythm. These are the "critical mass" theory, the "prolonged refractoriness" theory and the "upper limit of vulnerability" theory. They may all play a part.

In the critical mass theory, cardioversion simply depolarises a substantial mass of myocardium, so blocking the fibrillation or re-entry wavefront and terminating the arrhythmia.

The prolonged refractoriness theory suggests that the defibrillating impulse lengthens the refractory period of a large mass of myocardial cells, so they are unable to conduct the arrhythmia wavefront.

The upper limit of vulnerability theory suggests that the defibrillation energy blocks the arrhythmia wavefront, and suppresses re-emergence of the arrhythmia that might occur due to myocardial stimulation during the vulnerable period.

Types Of External Cardioverter Device

Cardioversion by alternating current (AC) shock causes myocardial damage; all cardioversion devices are now based on the delivery of direct current (DC).

A DC cardioversion device is frequently referred to as a DC defibrillator or a "defib". The device has two outputs which are connected to hand-held defibrillator paddles or alternatively to specially designed adhesive electrode pads.

The DC impulse may be monophasic or biphasic. Most older DC defibrillators deliver a monophasic voltage across the two defibrillator electrodes. Newer defibrillators deliver a biphasic stimulus.

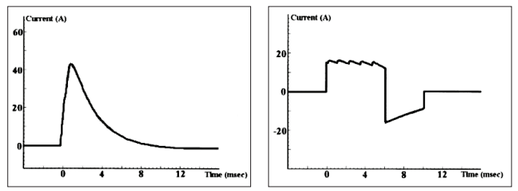

Figure 1: The upper diagrams illustrate a traditional monophasic shock waveform (i.e. a damped sine-wave). The right diagram depicts a more modern biphasic shock waveform.

In general biphasic shocks tend to be successful at lower energy levels, and provide higher success rates, hence their increasingly widespread use.

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Shock Vector

The shock vector is a theoretical line joining the two electrodes positioned on the outside of the chest.

Despite delivering a large amount of electrical energy across the thorax, only about 10% of this energy actually reaches the heart.

Dissipation of energy is due partly to transthoracic impedance, and partly to the complex route taken by the stimulus along pathways of least resistance within the chest.

Electrode Positions

The success rates and required energy settings depend on the external electrode positions and shock vectors that are chosen.

For cardioversion of atrial fibrillation, the standard electrode placement is left anterior (left parasternal) to left posterior (i.e. below the left scapula), if the arrhythmia is likely to be focused in the left atrium, as in mitral valve conditions. If both atria are equally involved, a right anterior chest (right parasternal) and left posterior vector is most likely to succeed.

For emergency cardioversion of ventricular arrhythmias, an apex to right anterior shock vector is advised.

Synchronisation

It is important to synchronise the 8–10msec defibrillation shock to arrive during ventricular depolarisation and particularly to avoid intruding into the vulnerable period of the ventricular repolarisation process, which could provoke ventricular fibrillation by an R on T effect.

Delivery of unsynchronised shocks to any arrhythmia other than ventricular fibrillation is therefore to be avoided.

External defibrillators are equipped with a synchronisation switch or button; when activated the machine will read the patient's ECG and deliver the shock only during the QRS complex. It is important to be aware that the machine will default back to unsynchronised mode after delivery of a shock.

Indications For Cardioversion

These can be subdivided into emergency and elective indications.

Emergency indications for cardioversion.

The role of defibrillation under cardiac arrest conditions has been summarised by the UK Resuscitation Council Guidelines of 2005.

Under cardiac arrest conditions the arrhythmias or "shockable rhythms" which require DC shock are:

- Ventricular fibrillation
- Pulseless ventricular tachycardia

The non-shockable arrhythmias, in which cardioversion should not be used are:

- Asystole
- Pulseless electrical activity (PEA), formerly called electromechanical dissociation (EMD)
- Fine VF which cannot be distinguished from asystole

The modern generation of Automatic External Defibrillators (AEDs) can interpret the patient's ECG; they perform well in distinguishing between shockable and non-shockable rhythms, and can either take action automatically or issue voice prompts to the rescuers.

Guidelines indicate that the first shock applied for VF and pulseless VT should be 150–200 joules if using a biphasic machine and 360 joules if using a monophasic device.

Outside of a cardiac arrest scenario, urgent cardioversion is appropriate for any poorly tolerated arrhythmia, for example, ventricular tachycardia or ventricular flutter with a cardiac output, torsades des pointes, poorly tolerated supraventricular tachycardias, etc.

Elective indications for cardioversion:

- Atrial fibrillation (synchronised shock, anticoagulation usually necessary)
- Atrial flutter (synchronised shock, anticoagulation usually necessary)
- Re-entry junctional tachycardias, if refractory to adenosine, etc. and particularly those associated with pre-excitation syndromes, notably the Wolff-Parkinson-White syndrome (synchronised shock)

Guidelines suggest that for effective and reliable cardioversion of atrial fibrillation, an initial energy of 200 joules should be selected, whether using a monophasic or biphasic defibrillator.

Success Rates With Cardioversion

Success rates for cardioversion under cardiac arrest conditions decrease rapidly with time, and the first few minutes are vital. Even where facilities exist for public access defibrillation (PAD), survival to leaving hospital after a cardiac arrest in the community is between 3% and 20%.

In the context of elective cardioversion for atrial fibrillation, sinus rhythm can be restored by drugs (pharmacological cardioversion) or by DC cardioversion. Drugs of proven value in converting atrial fibrillation to sinus rhythm when given orally are amiodarone, flecainide, ibutilide and propafenone.

The prospects for success in elective cardioversion of atrial fibrillation depend substantially on the duration of atrial fibrillation before the cardioversion attempt. In some trials of DC cardioversion, 25% of patients either would not cardiovert or relapsed back into AF within a few minutes but, with good case selection, success rates of around 90% are achievable. Pretreatment with either amiodarone, flecainide, ibutilide, propafenone or sotalol can enhance success rates and the preservation of sinus rhythm afterwards.

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However, there is a gradual attrition rate over the ensuing years. For example, in the AFFIRM trial comparing rhythm control against rate control in atrial fibrillation[3], only 63% were still in sinus rhythm after 5 years, despite resorting to DC cardioversion two or more times in half the patients.

Hazards Of Cardioversion

The procedure is generally very safe. DC cardioversion can be carried out either with full anaesthesia or increasingly using benzodiazepine-induced sedation only, either using diazemuls or midazolam, in which case the entire procedure can be nurse-led with physician backup only.

Complications include arrhythmias and embolic events, particularly embolic stroke. In early reports embolic stroke occurred in 5% of patients; this unacceptable figure has been reduced to less than 1% by adequate anticoagulation.

All guidelines agree that anticoagulation with warfarin or coumadin should be provided before cardioversion of atrial fibrillation, if AF has been present for more than 48 hours, and that a 4-week period of adequate anticoagulation is necessary to reduce the risk of embolism.

Guidelines For Cardioversion In Atrial Fibrillation & Atrial Flutter

Helpful guidelines were issued in 2006 concerning management of patients with these supraventricular arrhythmias, both internationally by the Joint ACC/AHA/ESC Task Force[4], and in the UK by the National Institute of Clinical Excellence[5].

Within the UK, NICE recommends adoption of a rhythm control strategy involving cardioversion in:

- patients who are symptomatic due to their AF
- younger patients
- patients presenting for the first time with lone AF
- patients with AF secondary to a treated/corrected precipitant
- patients with congestive heart failure

The Joint Task Force recommends urgent cardioversion of AF when accompanied by heart failure, hypotension or angina and that under these circumstances, low molecular weight heparin should be given beforehand.

The Joint Task Force also recommends cardioversion for patients with hemodynamically stable atrial fibrillation, if AF causes symptoms like palpitation and dyspnoea which are unacceptable to the patient.

When atrial fibrillation recurs soon after direct current cardioversion, a further cardioversion can be attempted after administration of antiarrhythmic medication.

To restore and maintain normal sinus rhythm (rhythm control) in patients with atrial fibrillation, the Joint Task Force acknowledges that cardioversion is a part of the long-term management strategy.

Similarly it is accepted that patient preference is a reasonable consideration in the selection of infrequently repeated cardioversion for the management of symptomatic or recurrent AF.

However, the role of cardioversion is played down in the recent Joint Task Force guidelines, reflecting the fact that other measures, particularly atrial fibrillation ablation, are becoming preferable.

Thus the Task Force recommends drug therapy as first line and fibrillation ablation as the second line option to achieve rhythm control.

Drug Treatment After Cardioversion

Embolic events, including stroke, can occur at any time up to 4 weeks after successful cardioversion from atrial fibrillation or flutter. This is because of atrial stunning, in which the left atrium takes time to regain its contractile efficiency after cardioversion.

Therefore patients pretreated with warfarin to minimise the risk of embolism and embolic stroke during cardioversion, should be maintained on warfarin for a minimum period of 4 weeks.

Unfortunately patients who have been successfully cardioverted from AF remain at increase risk of embolism.

This is partly due to the significant risk of recurrent AF after cardioversion, and partly because factors that frequently coexist in patients with AF (advanced age, HF, hypertension, LA enlargement, and LV dysfunction) are themselves risk factors for thromboembolism. Therefore, guidelines indicate that it is best to continue warfarin anticoagulation after successful cardioversion.

Internal Cardioversion

Low energy cardioversion from atrial fibrillation can be achieved using catheters sited in the right atrium and in either the coronary sinus or the pulmonary artery. This technique can succeed where external cardioversion has failed (e.g. in patients with COPD or obesity). Energy levels are usually between 5 and 10 joules, and the procedure can be done under light sedation only.

Implantable Cardioverters

Implantable atrial defibrillators (IADs) have been used in patients with highly symptomatic recurrent atrial fibrillation. Results are mixed[6], many patients developed increasing defibrillation thresholds, pain during internal cardioversion and a range of additional arrhythmias include bradycardia.

For major ventricular arrhythmias, implantable cardioverter defibrillators (ICDs) have proved highly successful and life saving. This topic has been reviewed in-depth in the recent joint ACC/AHA/ESC guidelines[7].

CARDIOVERSION

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Conclusion

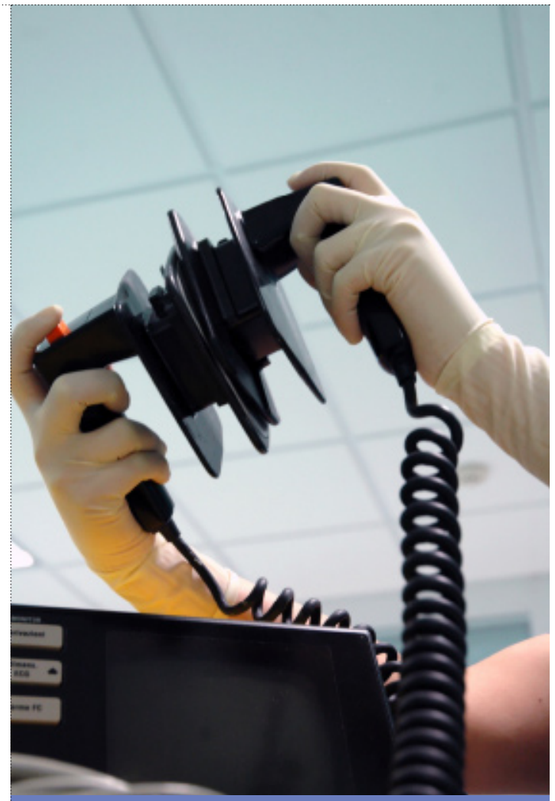
Because of advances in catheter ablation of arrhythmias, and arrival of new drugs such as dronedarone, the role of DC cardioversion is shrinking but it remains an important tool in arrhythmia management.

MCQs

1. What is the diagnosis?
2. Is this condition related to psoriasis?
3. What is the significance of the right ventricular pressure?
4. Does this patient need antibiotic cover for dentistry, etc?
5. What is the likely outcome for this patient?
6. How is mitral regurgitation classified on echocardiographic criteria?
7. What is the significance of these left ventricular measurements?
8. What is the significance of the increased tricuspid regurgitation and peak right ventricular pressure?
9. What medical options are available for this patient?
10. What surgical options are available for this patient?
11. How should this arrhythmia be treated?
12. Is immediate cardioversion required?
13. What anti-embolic anticoagulation is necessary?
14. Why did this arrhythmia occur?
15. Does this episode have implications for planning of cardiac surgery?
16. Why has this further arrhythmia occurred?
17. Should a further attempt at rhythm control be made by cardioversion?
18. Should atrial fibrillation be managed by rate control only?
19. What anticoagulation regime should be used?
20. What are the long-term implications?

Answers To MCQs

1. He has developed mitral regurgitation, this may be ischaemic, degenerative, due to mitral valve prolapse, even possibly rheumatic.
2. Some reports have suggested a link between mitral valve prolapse and psoriasis.
3. The normal peak right ventricular pressure is 30mm Hg, but due to the methodology, a reading of 45 is just compatible with a pressure at the upper limit of normal.
4. On the basis of recently revised criteria he does not, though he would do so under previous criteria.
5. His valve lesion may remain static, but is apparently new and therefore may progress over time.
6. Mitral regurgitation is classified according to the amount of blood that flows back into the left atrium per heart beat, which can be measured in terms of the area of the regurgitant jet. Less than 20% of the area of the left atrium suggests mild regurgitation, 20–59% is moderate and more than 60% is severe.
7. The left ventricular end-diastolic dimension was 6.2cm, the end-systolic dimension was 3.8cm. Thus the end-systolic dimension is not more than 4.0cm. Systolic function is deemed to be deteriorating if this dimension is exceeded. The normal end-diastolic dimension is <6.0cm, this is exceeded in keeping with volume overload.



Because of advances in catheter ablation of arrhythmias, and arrival of new drugs such as dronedarone, the role of DC cardioversion is shrinking but it remains an important tool in arrhythmia management.
Practical Procedures.

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8. The right ventricular peak RV pressure has risen to 65, the patient is becoming significantly pulmonary hypertensive. These data indicate the need for surgical treatment, even though ventricular function remains good.
9. Medical options are now limited to deployment of vasodilators and diuretics with little prospect for long-term control using medication alone.
10. Surgical options involve mitral valve repair or mitral valve replacement. As set out in the recent ACC/AHA guidelines8, the ideal option would be to progress to mitral valve repair by an experienced surgeon in a high-volume centre.
11-13. The episode of paroxysmal atrial flutter with 2:1 A-V block should be converted to sinus rhythm as soon as possible either by drugs (such as amiodarone or possibly by DC cardioversion) but prompt institution of anticoagulation is necessary using low molecular weight heparin, although the arrhythmia had developed within the recent 48 hours.
14. The arrhythmia probably developed as a result of increased atrial volume and pressure due to the combination of mitral and tricuspid regurgitation.
15. It implies that it would be best to consider a surgical procedure which deals with both the mitral and tricuspid regurgitation and the tendency to atrial arrhythmias (i.e. valve repair combined with a Maze operation) in which sources of electrical instability in the left atrium around the pulmonary veins are isolated.
16. Atrial flutter has recurred after successful valve repair plus a Maze operation probably because the right atrium is electrically unstable as well as the left atrium.
17. Guidelines suggest that an attempt to restore sinus rhythm at this age should be made principally on the basis of symptoms, which are present in this case. Modern guidelines were issued in 2006 concerning management of patients with atrial fibrillation and atrial flutter, both internationally by the Joint ACC/AHA/ESC Task Force[4], and in the UK by the National Institute of Clinical Excellence5.
18. If the attempt to cardiovert him fails, then there would be no long-term disadvantage if a rate control strategy is adopted, including long-term warfarinisation.
19. He requires a minimum of 4 weeks effective anticoagulation before being cardioverted, and he should continue on warfarin whether or not the procedure is successful.
20. He is quite likely to revert to atrial flutter or fibrillation after successful cardioversion, but his valve repair has a 90% chance of remaining good at 10 years.

References

[1] Zoll, et al. Reported successful external cardioversion from ventricular fibrillation. NEJM, 1956, 254:727.

[2] Lown. Cardioversion of atrial fibrillation using an external synchronised capacitor. JAMA, 1962, 182:548.

[3] AFFIRM trial comparing rhythm control against rate controlin atrial fibrillation. NEJM, 2002, 347:1825.

[4] Joint ACC/AHA/ESC Task Force. Guidelines for patients with supraventricular arrhythmia. Eur Heart J, 2006, 27:1979.

[5] National Institute of Clinical Excellence. Giudelines 36. NICE, June 2006.

[6] Eur Heart J, 2003, 24:2083.

[7] Joint ACC/AHA/ESC Task Force. Guidelines. JACC, 2006, 48:E247–E346.

[8] Joint ACC/AHA/ESC Task Force. Guidelines. JACC, 2006, 48:1–148.

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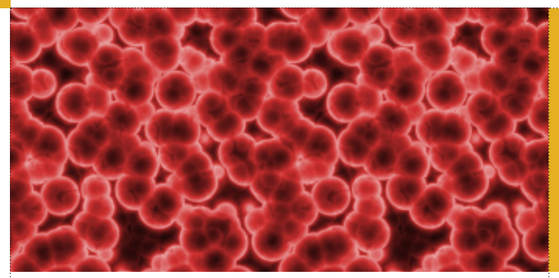
NON-SPECIFIC SYMPTOMS IN A PATIENT WITH VALVULAR HEART DISEASE

Richard Bond and Mark Dayer



Admission blood tests revealed a neutrophilia, a raised CRP of 124, a normal haemoglobin and U&Es. 6/6 blood culture bottles grew Streptococcus sanguinis which was very sensitive to penicillin (MIC = 0.06mg/L). The urine dip was negative for blood. Good Clinical Care.

Abbreviations

MIC	Minimum inhibitory concentration
LVEDD	Left ventricular end diastolic diameter
LVESD	Left ventricular end systolic diameter
LVEDP	Left ventricular end diastolic pressure
LV	Left ventricle
LVH	Left ventricular hypertrophy
IE	Infective endocarditis
TTE	Transthoracic echocardiogram
TOE	Transoesophageal echocardiogram

A 46-year-old man presented to our medical assessment unit with a 3-week history of general malaise, muscle aches, night sweats, decreased appetite and a sore throat. He had been lost to follow-up at his local cardiology department with a diagnosis of aortic regurgitation (severity unknown). He had no other past medical history and was on no regular medication. Of note, his son had been diagnosed with a bicuspid aortic valve.

On examination he was apyrexial, blood pressure was 146/69 with a regular pulse of 90 and oxygen saturations were 97% on air. Peripherally he had 7 splinter haemorrhages. There were prominent carotid pulsations. He had a 2/6 ejection systolic murmur and a 3/4 early diastolic murmur. His chest was clear and there was no organomegaly on abdominal examination.

What First Line Investigations Would You Request?

- FBC, U&Es and CRP/ESR
- 3 sets of blood cultures
- Urine dip
- ECG
- Transthoracic echocardiogram

Results Of The Above Investigations

Admission blood tests revealed a neutrophilia, a raised CRP of 124, a normal haemoglobin and U&Es. 6/6 blood culture bottles grew Streptococcus sanguinis which was very sensitive to penicillin (MIC = 0.06mg/L). The urine dip was negative for blood. His ECG (see Figure 1) is shown below. What does it show and why does this concern you?

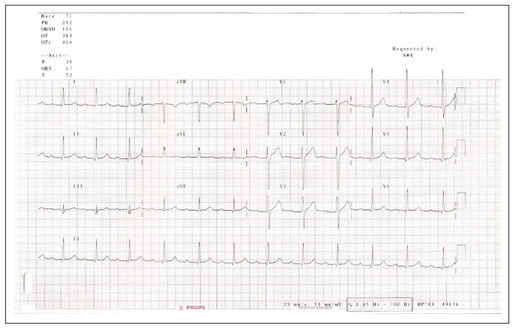


Figure 1: ECG.

The ECG shows first degree atrioventricular block and left ventricular hypertrophy (LVH). AV block in the context of infective endocarditis should raise suspicions of a possible aortic root abscess.

His transthoracic echocardiogram (TTE) revealed a bicuspid aortic valve with severe aortic regurgitation. The left ventricle was dilated (LVEDD 6.1cm, LVESD 3.7cm) with normal systolic function. There were no obvious vegetations or root abscess detected.

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What Would You Do Now?

He was started on intravenous benzylpenicillin and gentamicin as per microbiology advice. As there was a strong suspicion of infective endocarditis with aortic root abscess development he underwent a transoesophageal echocardiogram (TOE). This confirmed a bicuspid aortic valve, severe aortic regurgitation (see Figure 2) and demonstrated an aortic root abscess involving the aorto-mitral continuity (see Figure 3). He was referred to the local cardiothoracic centre and underwent successful aortic valve replacement.

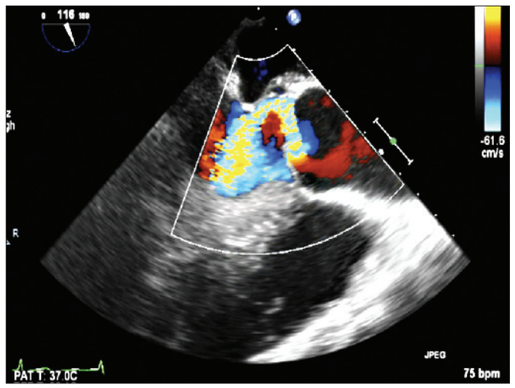

Figure 2: Severe aortic regurgitation with eccentric jet hitting aorto-mitral continuity.

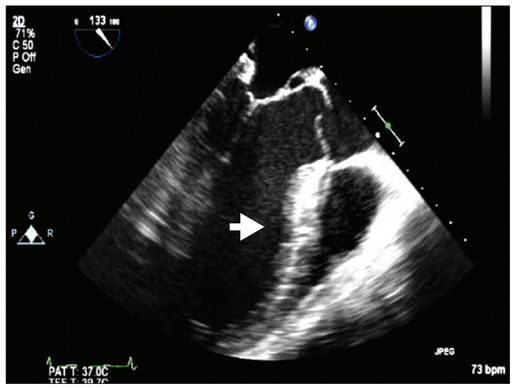

Figure 3: Abscess cavity (White arrow). The aortic valve leaflets are clearly failing to coapt.

Discussion

Bicuspid Aortic Valve

A bicuspid aortic valve is the most common congenital cardiac abnormality occurring in 1–2% of the population. It has been found to be an inheritable condition with an incidence of 10–17% in affected families[1]. The male to female ratio is 2:1.

The normal aortic valve is composed of 3 cusps – the right, left and non-coronary cusp (see Figure 4). The congenitally bicuspid aortic valve has two functional cusps (see Figure 5) usually of unequal size. The larger cusp is called the conjoined cusp and is usually a fusion of the right and left cusps. There is usually a central ridge (raphe) in the centre of this cusp.

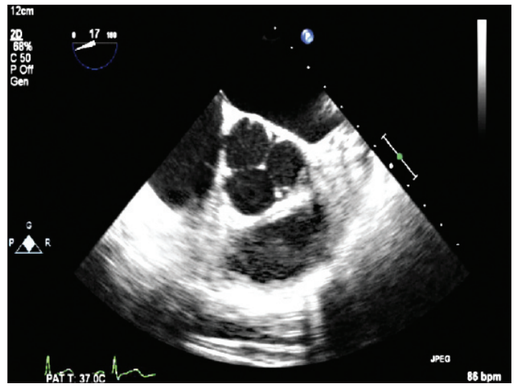

Figure 4: Closed tricuspid aortic valve (TOE).

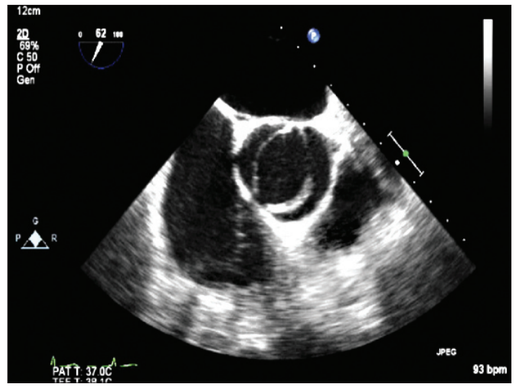

Figure 5: Open bicuspid aortic valve (TOE).

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There are two common abnormalities associated with the bicuspid aortic valve:

- Coarctation of the aorta
- Left coronary artery dominance (posterior-descending coronary artery arising from the left coronary artery) is more common and the left main stem is less than 5mm (normal approx 10mm) in 90% of cases[2,3]. This is important information to know when planning for surgery.

About 30% of individuals with a bicuspid aortic valve develop complications[4]:

- Aortic stenosis: this is due to fibrosis and calcification of the valve. One study has shown that sclerosis of the valve begins in the second decade. In the fourth decade valve calcification is noted. The average gradient across the valve increases by 18mmHg per decade5. The average age of valve replacement is 59; this compares with "senile" aortic stenosis, where the average age of replacement is 646.
- Aortic regurgitation: this occurs as a result of prolapse of the larger cusp, associated aortic root dilatation, coarctation of the aorta or infective endocarditis.
- Infective endocarditis: the risk of developing infective endocarditis on a bicuspid aortic valve is 10–30% over a lifetime. Surgical studies have shown that approximately 50% of all infected aortic valves are bicuspid at the time of operation[7,8]. In one study the mean age of presentation was 39. Complications were common including heart failure (72%) and periannular abscesses (30%). Overall mortality was 14% and surgical mortality was 9%[9]. With the disappearance of rheumatic fever in developed countries, bicuspid aortic valves are likely to become the most common cardiac predisposing factor for infective endocarditis.
- Aortic dissection: the risk of aortic dissection is nine times higher in bicuspid aortic valves compared to tricuspid valves[10]. Hypertension predisposes to aortic dissection which explains some of this increased risk in the presence of coexistent coarctation. There is also evidence that the aortas of bicuspid aortic valve patients contain less elastic tissue leading to aortic fragility and possible dissection[11].

Patients with a bicuspid aortic valve should be followed-up at regular intervals.

Aortic Regurgitation

Aortic regurgitation (AR) is the diastolic flow of blood from the aorta into the left ventricle. Regurgitation can result from disease of the cusps, disease/dilatation of the aorta or trauma. More than half of cases are caused by aortic root dilatation which prevents leaflet coaptation. The causes of AR are listed in Table 1.

Chronic Aortic Regurgitation	
1. Valvular:	Endocarditis
	Rheumatic heart disease
	Congenital – bicuspid valve, subaortic and supraaortic stenosis
	Connective tissue disease – rheumatoid arthritis, SLE, ankylosing spondylitis
2. Aortic root diseases:	Dilatation – Marfan, hypertension, Ehlers-Danlos, pseudoxanthoma elasticum, aortitis
	Distortion – dissection (types I and II), syphilis, Reiters syndrome, rupture of sinus of Valsalva aneurysm

Table 1: Causes of aortic regurgitation.

Acute Aortic Regurgitation

This is usually a surgical emergency. A large volume of blood regurgitates back into a non-adapted left ventricle (LV). This produces an increase in the LV end-diastolic pressure (LVEDP) which leads to pulmonary congestion and oedema. There is reduced cardiac output which together with the raised LVEDP results in reduced coronary blood flow and ischaemia. This progresses to refractory heart failure and cardiogenic shock.

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Chronic Aortic Regurgitation

There is combined volume and pressure overload on the LV. Initially the patient remains asymptomatic as there is compensatory LV dilatation, hypertrophy and an increased preload. The increase in diastolic filling increases stroke volume (Starling's mechanism) so that cardiac output is maintained. As the valve continues to fail and an increased blood volume regurgitates into the LV there is continued hypertrophy and remodelling of the LV (increased dilatation and its shape becomes more spherical). The size of the ventricle and ejection fraction are important determinants for prognosis and are used as markers to aid in decision making for possible surgery.

Symptoms

Patients usually present with dyspnoea, orthopnoea and paroxysmal nocturnal dypsnoea. They may also get angina symptoms.

Signs

The pulse is collapsing in nature. There is a wide pulse pressure. The apex beat is displaced and hyperdynamic. The murmur of AR is a high pitched, early diastolic murmur best heard with the patient sitting forward in held expiration. It is best heard at the left lower sternal edge. There may be an associated ejection systolic murmur due to either mixed aortic valve disease or due to increased forward flow volume across the valve. An Austin Flint murmur can sometimes be heard. This is a mid diastolic murmur heard over the mitral valve due to partial closure of the anterior mitral valve leaflet as it hit by the regurgitant jet of AR.

There are many eponymous signs associated with AR some of which are listed below:

- Corrigan's sign – visible carotid pulsation
- De Musset's sign – head nodding with each pulse
- Muller's sign – visible pulsation of the uvula
- Quinke's sign – visible capillary pulsation of the nail bed
- Traube's sign (pistol shot femorals) – booming systolic and diastolic sounds heard over the femoral artery
- Duroziez's sign – systolic murmur heard over femoral artery on proximal compression and a diastolic murmur on distal compression

Investigations

- Chest radiography – to assess cardiac size, ascending aortic root and for signs of pulmonary oedema
- ECG – usually normal in early disease but in severe cases may show LVH with a strain pattern and left axis deviation.
- Echocardiography (transthoracic/transoesophageal) – helps to confirm the diagnosis and severity. It can also give clues to the aetiology. The valve anatomy can be assessed, aortic root dimensions can be measured and an intimal flap (indicating aortic dissection) or vegetations on the valve may be seen. Follow-up with serial echocardiography is recommended in patients with AR.
- Cardiac catheterisation – is used to assess for coronary artery disease prior to aortic valve replacement and can assess severity of AR. If coronary artery disease is present then coronary artery bypass grafting can occur at the time of valve replacement. An aortogram is used to look at the aortic root.

Natural History Of Ar[12]	
Asymptomatic patients with normal LV systolic function	Progression to symptoms and/or LV dysfunction <6%/yr
	Progression to asymptomatic LV dysfunction <3.5%/yr
	Sudden death <0.2%/yr
Asymptomatic patients with LV systolic dysfunction	Progression to cardiac symptoms >25%/yr
Symptomatic patients	Mortality rate >10%/yr

Table 2: Natural history of AR[12].

Treatment Of AR

1. Medical – symptoms of fluid overload respond to diuretics. Vasodilators (ACE inhibitors, calcium channel blockers) are used to reduce afterload. This reduces the amount of regurgitation and improves LV systolic function.

2. Surgery is indicated in patients with severe AR, surgery is indicated if:

- Patients with NYHA class III/IV dyspnoea
- Asyptomatic patients when LV function declines or dilatation is seen (ejection fraction <50%, LVESD > 55mm)
- Coexisting aortic root dilatation >50mm

Infective Endocarditis

Infective endocarditis (IE) – is a microbial infection of the endocardium. It usually involves the heart valves (native or prosthetic) but can affect any part of the endocardium (e.g. at the site of a VSD) and intracardiac devices (e.g. pacemakers). The incidence of infective endocarditis is approximately 1.7–6.2 cases per 100,000 patient years[13]. Prosthetic valve endocarditis accounts for 10–15% of cases.

Traditionally IE is classified as acute or subacute. Acute IE is usually caused by Staphylococcus aureus and progresses rapidly over days to weeks leading to valve destruction and metastatic infection. Subacute IE is often caused by a streptococcus of low virulence. The disease progresses over weeks to months.

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Mechanism Of Infection

Damage to the endothelium results in direct contact of subendothelial components with blood and activation of the coagulation cascade. The coagulum that is produced contains large quantities of fibrinogen fibrin, fibronectin, plasma proteins and platelet proteins. Bacteria bind to these structures and colonises them during a bacteraemia. The infected coagulum is known as a vegetation (see Figures 6 and 7). The organisms responsible for IE are those that have the greatest ability to adhere to damaged valves[14].

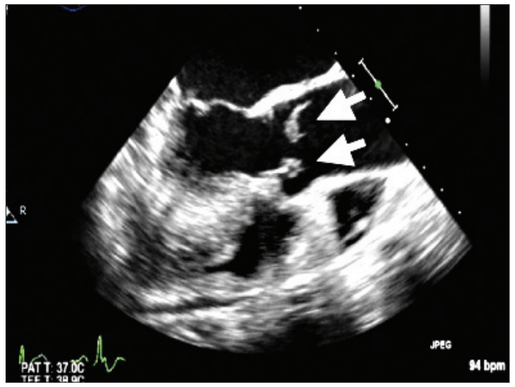

Figure 6: Vegetations on a mitral valve.

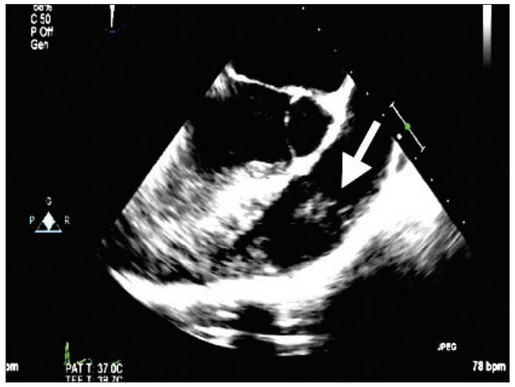

Figure 7: Vegetation on a pulmonary valve.

Microorganisms Responsible	
Staphylococcus aureus	33%
Streptococcus viridians	13%
Other streptococci	15%
Enterococci	14%
Other microorganisms	10%
No microorganisms	14%

Table 3: Microorganisms responsible (native and prosthetic valve endocarditis)[15].

Symptoms & Signs

Symptoms are often non-specific and include fever, rigors, weight loss, malaise and night sweats. Long standing infection produces anaemia, clubbing and splenomegaly. A new or changing murmur is common. Patients may present with congestive cardiac failure, AV block or pericarditis. Peripheral stigmata of endocarditis such as splinter haemorrhages, Roth's spots and glomerulonephtitis are common, however, the classic textbook signs (such as Oslers nodes and Janeway lesions) are rare, except for in the developing world. Systemic embolisation occurs in 22–50% of cases. This is more common with Staphylococcus aureus and HACEK organisms[16].

Diagnosis

There should be a high index of suspicion in patients presenting with an unexplained fever, predisposing cardiac lesion or embolic phenomena. Blood tests should include FBC, U&Es, inflammatory markers and blood cultures. A urine dip should be performed for microscopic haematuria and proteinuria which may indicate glomerulonephritis. An ECG may show AV block (aortic root abscess) or an acute MI secondary to emboli. Initial imaging should be with transthoracic echocardiography.

IE, if untreated or treated with inappropriate antibiotics is often fatal. It is therefore important to identify the causative organism and establish its sensitivities to antibiotics. Three sets of blood cultures should be taken at least 1 hour apart from three different sites. As there is a constant bacteraemia there is no need to wait for spikes in temperature. It is advisable if possible to wait for positive blood cultures before starting antibiotic treatment.

Blood cultures are negative in approximately 12–14% of cases; the most common cause is the previous administration of antibiotics[17,18]. Often it is due to difficult to culture organisms, such as Coxiella, Bartonella, the HACEK group (Haemophilus species, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens and Kingella kingae) and fungal species. In the case of blood culture negative endocarditis, serum should be analysed for Coxiella burnetti, Bartonella and Chlamydia species antibodies and the excised valve or rarely embolic material should be analysed by microscopy, culture, histology and PCR.

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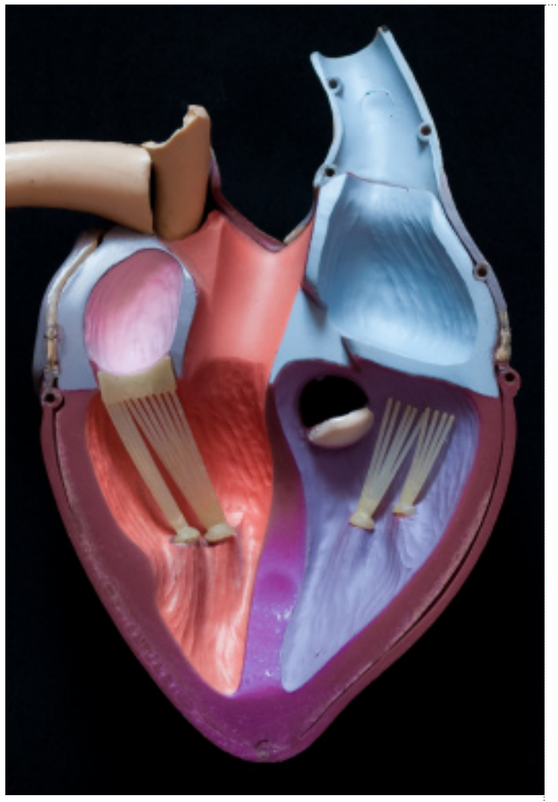

Echocardiography should be performed in all cases of suspected infective endocarditis. TTE is usually first line as this is more readily available. In low-risk groups a normal TTE suggests that infective endocarditis is unlikely. In high-risk groups with a normal TTE, a TOE should be performed as it is more sensitive than TTE for the detection of vegetations and abscesses. TOE is almost always required for investigation of prosthetic valve endocarditis as vegetations are often small and there is artefact from previous surgery. Acoustic shadows from the metallic valve itself make imaging difficult transthoracically.

The Duke classification for diagnosing endocarditis was developed in 1994 and is highly sensitive and specific. It has been modified since then to take into account the widespread use of transoesophageal echocardiography, serological testing for Coxiella burnetti and the increasing prevalence of staphylococcal infection[19].

Modified Duke Criteria[19]	
Pathological criteria	Positive histology or microbiology of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content)
Major criteria	Two positive blood cultures showing typical organisms consistent with infective endocarditis, such as Streptococcus viridans and the HACEK group
	Persistent bacteraemia from two blood cultures taken >12 hours apart or three or more positive blood cultures where the pathogen is less specific (e.g. Staphylococcus aureus and S. epidermidis)
	Positive serology for Coxiella burnetti, Bartonella species or Chlamydia psittaci
	Positive echocardiogram showing oscillating intracardiac mass, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves
Minor criteria	Predisposing heart disease or intravenous drug use
	Fever >38°C
	Immunological phenomena, such as glomerulonephritis, Osler's nodes, Roth's spots or positive rheumatoid factor
	Microbiological evidence not fitting major criteria
	Elevated C-reactive protein or erythrocyte sedimentation rate
	Vascular phenomena, such as major emboli, splenomegaly, splinter haemorrhages, petechiae or purpura
Definite infective endocarditis	Pathological criteria positive; or
	Two major criteria; or
	One major and three minor criteria; or
	Five minor criteria

Table 4: Modified Duke criteria[19].

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Antibiotic Treatment

Treatment of endocarditis requires close liaison with a microbiologist. Antibiotic choice depends on the pathogen cultured and its sensitivities. Generally prolonged intravenous antibiotic therapy (4–6 weeks) is required; most patients will require a tunnelled central line for this. If it is deemed necessary to treat empirically then guidelines have been produced by the British Society for Antimicrobial Chemotherapy; see Table 5[20].

Antimicrobial Chemotherapy	
Acute presentation	Flucloxacillin plus gentamicin
Indolent presentation	Penicillin or ampicillin/amoxicillin plus gentamicin
Penicillin allergy/MRSA	Vancomycin plus rifampicin plus gentamicin
Intracardiac prosthesis	Vancomycin plus rifampicin plus gentamicin

5: British Society for Antimicrobial Chemotherapy guidelines for empirical endocarditis therapy[20].

Once the pathogen has been identified and its sensitivities are known then the patient should be switched to the appropriate antibiotics.

Surgery

Early discussion with the local cardiothoracic centre is advised if surgery is thought necessary. Surgical intervention is required in 25–30% of patients with endocarditis[21].

Surgery is indicated in the following circumstances:

- Severe valvular incompetence and heart failure
- Annular or aortic abscess
- Infections resistant to antibiotics
- Fungal infective endocarditis
- Anterior mitral valve leaflet vegetation (particularly >10mm) and an increase in vegetation size despite appropriate antibiotic therapy
- Recurrent emboli after antibiotic therapy
- Prosthetic valve endocarditis (especially in the early post-operative period)

Timing of surgery is important. Haemodynamically unstable patients have a poor prognosis and transfer to the local cardiothoracic centre should not be delayed. Stable patients can finish their course of antibiotics prior to surgery. The surgical mortality during the acute phase of endocarditis is approximately 7.4% with a 10-year survival of approximately 61%. Recurrent endocarditis is seen in 20% of patients over a 10-year period post surgery[22].

Nice Guidance On Prophylaxis For Endocarditis[23]

In March 2008, NICE published new guidance on prophylaxis against infective endocarditis. Previously antibiotics had been offered routinely to those patients at risk of infective endocarditis undergoing interventional procedures. However, there is little evidence to support this. NICE recommends that patients at risk of infective endocarditis are not offered antibiotic prophylaxis when undergoing:

- dental procedures
- upper and lower gastrointestinal procedures
- genitourinary tract procedures
- upper and lower respiratory tract, including ear, nose and throat procedures

There has been some controversy over this policy and there may be local variations on its implementation.

Key Learning Points

1. A bicuspid aortic valve is the most common congenital cardiac abnormality occurring in 1–2% of the population.
2. A bicuspid aortic valve is commonly associated with coarctation of the aorta.
3. Thirty per cent of people with a bicuspid aortic valve develop complications including aortic stenosis, aortic regurgitation, infective endocarditis and aortic dissection.
4. Initial management of chronic aortic regurgitation is medical. Surgery is indicated in patients with NYHA class III/IV dyspnoea or asyptomatic patients when LV function declines or dilatation is seen.
5. Symptoms of IE are often non-specific and include fever, rigors, weight loss, malaise and night sweats. A new or changing murmur is common.
6. It is important to identify the causative organism of IE as treatment with inappropriate antibiotics is often fatal. Three sets of blood cultures should be taken at least 1 hour apart from three different sites.
7. Blood cultures are negative in approximately 12–14% of cases of IE; the most common cause is the previous administration of antibiotics.
8. Echocardiography should be performed in all cases of suspected infective endocarditis. TTE is usually first line as this is more readily available.
9. Prolonged intravenous antibiotic therapy (4–6 weeks) is required for the treatment of IE. Surgical intervention is required in 25–30% of patients with endocarditis.
10. NICE guidance (2008) recommends that patients at risk of infective endocarditis are not offered antibiotic prophylaxis when undergoing dental procedures.

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Questions (True/False Answers)

1. With regards to the Bicuspid aortic valve:

a. Is the most common congenital cardiac abnormality.
b. Is not inheritable.
c. Is associated with coarctation of the aorta.
d. Is associated with both aortic stenosis and regurgitation.
e. The risk of aortic dissection is 9 times higher when compared to a tricuspid aortic valve.

2. With regards to aortic regurgitation:

a. Is most commonly caused by aortic root dilatation.
b. Produces an early diastolic murmur heard best at the left sternal edge.
c. Is always associated with an Austin Flint murmur.
d. Echocardiography can give clues as to the aetiology.
e. Surgery should be considered if the LVESD is more than 55mm.

3. With regards to Infective endocarditis:

a. It only affects the heart valves.
b. It is most commonly caused by the HACEK organisms.
c. Osler's nodes are commonly seen in the UK.
d. Blood cultures are always positive.
e. Treatment is with oral antibiotics.

4. With regards to Infective endocarditis:

a. Transoesophageal echocardiography is always required.
b. The Duke criteria are used to aid diagnosis.
c. Antibiotics should be started as soon as the diagnosis is suspected and before blood cultures taken.
d. Surgery is always indicated.
e. Antibiotic prophylaxis is no longer offered to patients at risk of endocarditis prior to an invasive dental procedure.

References

[1] Cripe L, Andelfinger G, Martin LJ, et al. Bicuspid aortic valve is heritable. J Am Coll Cardiol, 7 July 2004, 44(1):138–143.

[2] Hutchins GM, Nazarian IH, Bulkley BH. Association of left dominant coronary arterial system with congenital bicuspid aortic valve. Am J Cardiol, 1978, 42:57–59.

[3] Murphy ES, Rösch J, Rahimtoola SH. Frequency and significance of coronary arterial dominance in isolated aortic stenosis. Am J Cardiol, 1977, 39:505–509.

[4] Bayne E. Aortic Valve, Bicuspid. **http://emedicine.medscape.com/article/893523-overview.**

[5] Beppu S, Suzuki S, Matsuda H, et al. Rapidity of progression of aortic stenosis in patients with congenital bicuspid aortic valves. Am J Cardiol, 1993, 71(4):322–327.

[6] Mautner GC, Mautner SL, Cannon RD, et al. Clinical factors useful in predicting aortic valve structure in patients >40 years of age with isolated valvular aortic stenosis. Am J Cardiol, 1993, 73:194–198.

[7] Janatuinen MJ, Vanttinen EA, Nikoskelainen J, Inberg MV. Surgical treatment of active native valve endocarditis. Scand J Thorac Cardiovasc Surg, 1990, 24(3):181–185.

[8] Varstela E, Verkkala K, Pohjola-Sintonen S, Valtonen, V, Maamies T. Surgical treatment of infective aortic valve endocarditis. Scand J Thorac Cardiovasc Surg, 1991, 25:167–174.

[9] Lamas CC, Eykyn SJ. Bicuspid aortic valve – A silent danger: analysis of 50 cases of infective endocarditis. Clin Infect Dis, 2000, 30:336–341.

[10] Larson EW, Edwards. Risk factors for aortic dissection: a necropsy study of 161 cases. Am J Cardiol, 1984, 53:849–855.

[11] Parai JL, Masters RG, Walley VM, Stinson WA, Veinot JP. Aortic medial changes associated with bicuspid aortic valve: myth or reality? Can J Cardiol, 1999, 15:1233–1238.

[12] ACC/AHA 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 (Committee on Management of Patients With Valvular Heart Disease). JACC, 1998, 32:1486–1588.

[13] Mylonakis E, Calderwood SB. Infective endocarditis in adults. N Engl J Med, 2001, 345:1318–1330.

[14] Moreillon P, Que YA. Infective endocarditis. Lancet, 2004, 363:139–149.

[15] Tornos P, Lung B, Permanver-Miralda G, Baron G, Delahaye F, Gohlke-Bärwolf C, et al. Infective endocarditis in Europe: lessons from the Euro heart survey. Heart, 2005, 91:571–575.

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16. Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Bolger AF, Levison ME, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the committee on Rheumatic Fever, Endocarditis and Kawasaki disease, Council on Cardiovascular disease in the Young and the Councils on Clincal Cardiology, Stroke and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. Circulation, 2005, 111:E393–E434.

17. Hoen B, Selton-Suty C, Lacassin F, Etienne J, Briançon S, Leport C, Canton P. Infective endocarditis in patients with negative blood cultures: analysis of 88 cases from a 1-year nationwide survey in France. Clin Infect Dis, 1995, 20:501–506.

18. Lamas CC, Eykyn SJ. Blood culture negative endocarditis: analysis of 63 cases presenting over 25 years. Heart, 2003, 89:258–262.

19. Li JS, Sexton DJ, Mick N, Nettles R, Fowler VG Jr, Ryan T, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis, 2000, 30:633–638.

20. Elliott TSJ, Foweraker J, Gould FK, Perry JD, Sandoe JAT. Guidelines for the antibiotic treatment of endocarditis in adults: report of the Working Party of the British Society for Antimicrobial Chemotherapy. J Antimicrob Chemother, 2004, 54:971–981.

21. Olaison L, Pettersson G. Current best practices and guidelines. Indications for surgical intervention in infective endocarditis. Cardiol Clin, 2003, 21:235–251.

22. D'Udekum Y, David, TE, Feindel CM, Armstrong S, Sun Z. Long-term results of surgery for active infective endocarditis. Eur J Cardio-Thoracic Surgery, 1997, 11:46–52.

23. National Institute for Health and Clinical Excellence. Prophylaxis Against Infective Endocarditis 2008. (NICE clinical guideline no. 64.) Available at: **http://www.nice.org.uk/CG064.** Patient consent obtained.

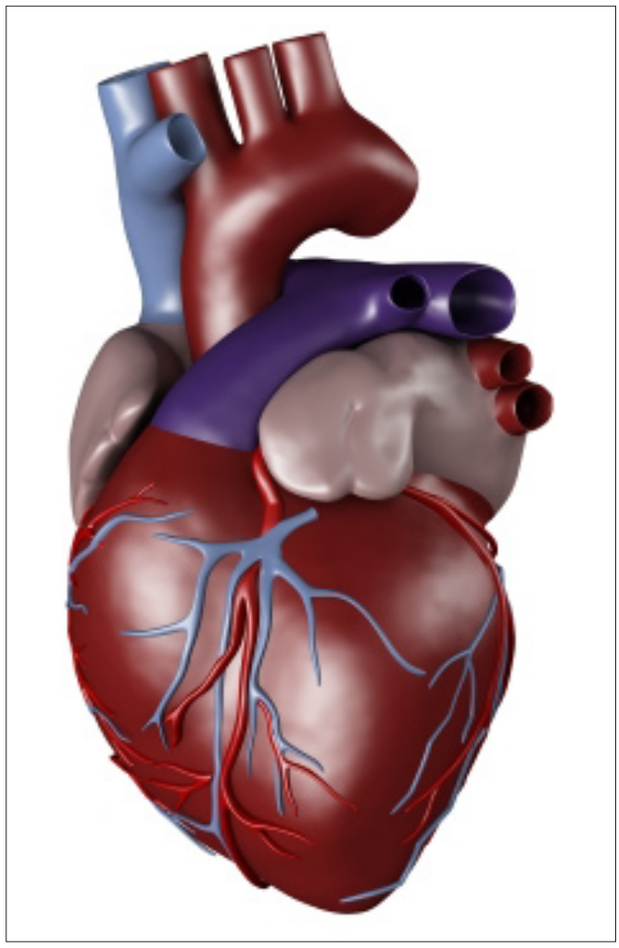
Answers

1. With regards to the Bicuspid aortic valve:
a. True **b.** False **c.** True **d.** True **e.** True

2. With regards to aortic regurgitation:
a. True **b.** True **c.** False **d.** True **e.** True

3. With regards to Infective endocarditis:
a. False **b.** False **c.** False **d.** False **e.** False

4. With regards to Infective endocarditis:
a. False **b.** True **c.** False **d.** False **e.** True



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MANAGEMENT OF NON-ST ELEVATION ACUTE CORONARY SYNDROME

Kalyan Gurazada and Jagdip Sidhu

A 62-year-old manual worker presents to the A&E department with a 2-hour history of central chest pain radiating to both arms. Patient Management.

A 62-year-old manual worker presents to the A&E department with a 2-hour history of central chest pain radiating to both arms. His pain subsided after being given sublingual glyceryl trinitrate and aspirin 300mg in the ambulance. Prior to his admission he smoked 20 cigarettes a day for over 40 years. Examination was unremarkable.

The patient's 12 lead ECG shows biphasic T waves in leads V2-V4 and T wave inversion in leads V5, V6 and aVL (see Figure 1). This suggests an ischaemic event in the anterolateral wall of the left ventricle.

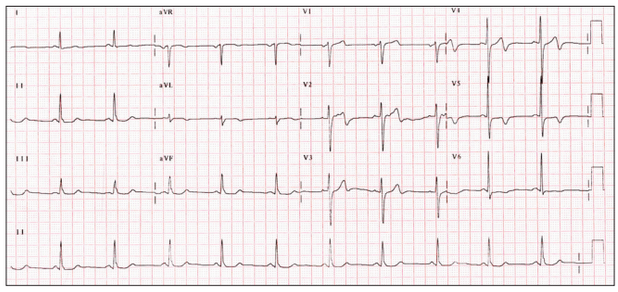

Figure 1: 12 lead ECG showing biphasic T waves in leads V2-V4 and T wave inversion in leads V5, V6 and aVL.

Patients presenting acutely with ischaemic-type chest pain without persistent ST elevation on their ECG are classified as having non-ST elevation acute coronary syndrome (ACS). The presenting ECG may show ST segment depression, T wave inversion/flattening or no detectable abnormalities. The initial priorites in such patients are to alleviate symptoms and treat ischaemia. The underlying pathology is atherosclerotic plaque rupture or erosion with superimposed thrombus leading to subtotal occlusion of the coronary artery. In contrast, patients with acute chest pain and persistent ST elevation or new left bundle branch block (LBBB) on their ECG usually have complete occlusion of the culprit coronary artery and require emergency reperfusion with thrombolysis or primary angioplasty to achieve reperfusion.

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What Would You Like To Do Next?

The initial management in non-ST elevation ACS involves[1]:

1. Pain relief with sublingual or, if necessary, IV nitrates. Opiates may be needed if pain does not resolve with nitrates. Supplemental oxygen if saturation level <90%.
2. Antiplatelet agents reduce thrombus burden and improve clinical outcomes. Aspirin 300mg bolus then 75mg od. Clopidogrel 300mg loading dose then 75mg od. Glycoprotein IIb/IIIa inhibitors are intravenous antiplatelet agents that should be considered in high-risk patients (e.g. ongoing or recurrent ST depression or diabetics).
3. Anticoagulant therapy with low molecular weight heparin are used as first line agents. Other alternative anticoagulant agents used in ACS include unfractionated heparin (in patients where anticoagulation may need to be reversed quickly), specific anti-factor Xa agent fondaparinux and the direct thrombin inhibitor, bivalirudin.
4. Anti-ischaemic therapy with beta blockers as first line agents. If beta blockers are contraindicated or not tolerated, the calcium antagonists diltiazem or verapamil may be used as an alternative. Statins and ACE Inhibitors have specific anti-atherosclerotic effects and should also be started. Statins should be started in patients with confirmed ACS regardless of their admission cholesterol levels.
5. Admission to the Coronary Care Unit. Serial ECGs and troponin I or T measurement at least 12 hours after onset of pain. Troponin indicates myocardial necrosis and is an important risk stratification tool. Patients with non-ST elevation ACS and elevated troponin are further classified as having non-ST elevation myocardial infarction (MI) while patients with non-ST elevation ACS and normal troponin are classified as having unstable angina.

The patient received aspirin, clopidogrel, enoxaparin 1mg/kg bd, bisoprolol and a statin on admission. An ACE inhibitor was added the next day. His 12-hour troponin I was elevated at 1.2ng/mL indicating non-ST elevation myocardial infarction. His full blood count, glucose and renal function were normal. His admission cholesterol level was 5.9mmol/L with an LDL level of 3.9mmol/L. He remained pain-free following his admission and serial ECGs remained unchanged.

What Would You Like To Do Next?

This patient is at increased risk of further MI/cardiac death. He has had a confirmed non-ST elevation myocardial infarction (NSTEMI). The patient should have early coronary angiography and revascularisation, either by percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG) depending on his coronary anatomy. A bedside echocardiogram is performed to ascertain left ventricular function which is an important prognostic indicator. This shows overall good left ventricular function with mild hypokinesis of the septum and anterior wall.

On day 2, coronary angiography was performed and revealed a focal stenosis of 90% in the proximal left anterior descending artery (see Figure 2). This was successfully treated with a single coronary stent (see Figure 3). The patient made an uneventful recovery from the procedure.

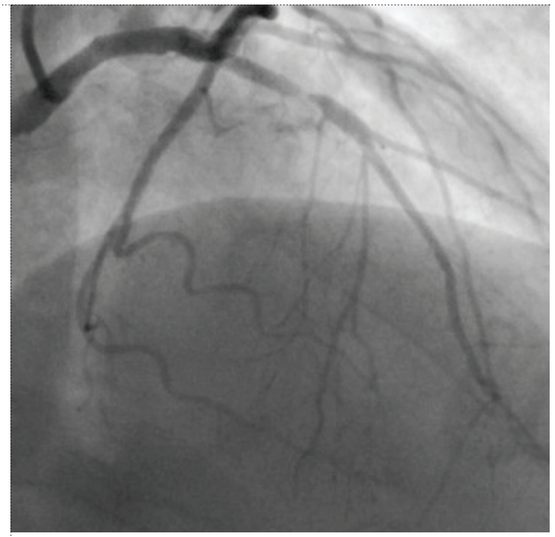

Figure 2: Coronary angiography was performed via the right femoral artery. A catheter is engaged in the left main coronary artery and contrast medium is injected to opacify the artery. There is a 90% stenosis in the proximal left anterior descending artery.

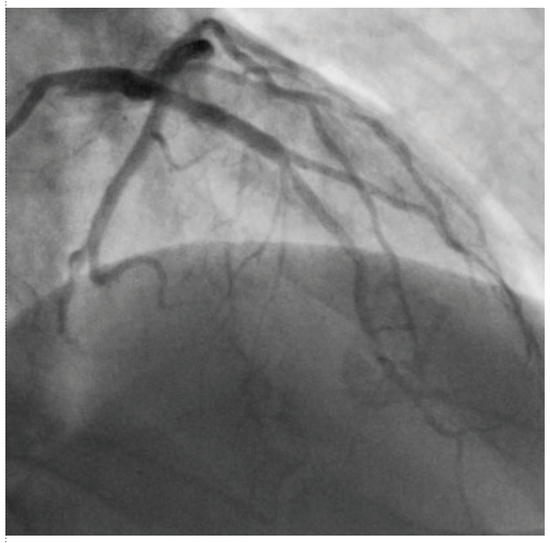

Figure 3: Result after deployment of a coronary stent. The stenosis has been treated with a good angiographic result.

MANAGEMENT OF NON-ST ELEVATION ACUTE CORONARY SYNDROME

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What Would You Like To Do Next?

A multidisciplinary approach is required for secondary prevention[2]. The patient was seen by a member of the cardiac rehabilitation team in the hospital. He received information about his condition and advice on risk factor modification with emphasis on smoking cessation. The patient was invited to attend an outpatient cardiac rehabilitation course. The patient was discharged from hospital on day 3. The patient was prescribed long-term aspirin and clopidogrel for 12 months following his confirmed NSTEMI. Long-term statin, beta blocker and ACEI were continued for prognostic benefit following his NSTEMI.

Non-ST Elevation Myocardial Infarction

Patients with NSTEMI are at increased risk of death and recurrent non-fatal MI. In hospital the mortality rate for this condition is 5% and mortality at 6 months is 13%[1].

Several risk stratification scores can be used to estimate the risk of further cardiac events. In routine clinical practice the thrombolysis in myocardial infarction (TIMI) risk score is the simplest to use and has been validated in several studies[3].

The TIMI risk score is summarised below:

	Points
1. Age ≥65	1
2. ≥3 coronary artery disease risk factors (family history, cholesterol, HT, smoking, DM)	1
3. Known coronary artery disease	1
4. Aspirin used regularly in past week	1
5. Recent severe angina in 24 hours prior to admission	1
6. Elevated cardiac enzymes (troponin)	1
7. ST segment deviation ≥0.5mm	1

Risk Score	Risk Of Death/MI At 14 Days (%)
0/1	3
2	3
3	5
4	7
5	12
6/7	19

The aims of initial treatment are to alleviate ischaemia, reduce thrombus burden, monitor the patient with serial ECGs and to measure markers of myocardial necrosis at an appropriate time interval. Early coronary angiography with a view to revascularisation has been shown to significantly reduce the rate of death and non-fatal MI4. This invasive strategy should be considered in all patients after taking into account comorbidities and assessing the risk benefit ratio for the individual patient.

Immediate, urgent angiography and revascularisation should be undertaken in patients with:

- Angina refractory to standard medical treatment
- Recurrent angina associated with ischaemic ECG changes despite having full pharmacological treatment for NSTEMI
- Heart failure or haemodynamic compromise (shock)
- Ventricular fibrillation or ventricular tachycardia

Most NSTEMI patients respond to initial medical treatment and can be stabilised before undergoing early angiography and revascularisation prior to hospital discharge (ideally within 72 hours of admission)[1].

Patients with NSTEMI still carry a significant risk of recurrent ischaemic events after their initial inpatient treatment including revascularisation. Secondary prevention including lifestyle interventions and long-term drug therapy have been shown to improve long-term morbidity and mortality. Cardiac rehabilitation including both an exercise programme and lifestyle advice/counselling should be offered to all patients post discharge. In the absence of any contraindication, patients should receive the following drug therapy after their NSTEMI[2]:

- Aspirin (long term)
- Clopidogrel (12 months)
- Statin (long term) with target LDL<2.0 mmol/L
- Beta blocker (long term)
- ACE inhibitor (long term)

MANAGEMENT OF NON-ST ELEVATION ACUTE CORONARY SYNDROME

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Good Clinical Care Assessment Questions

1. Which of the following conditions can cause an elevated troponin level?
a. Pulmonary embolism
b. Severe sepsis
c. Myocarditis
d. Acute stroke
e. Renal failure

2. In NSTEMI, which of the following factors are predictors of cardiac death and recurrent MI?
a. Severity of chest pain
b. Diabetes mellitus
c. Impaired left ventricular systolic function
d. Chronic renal impairment
e. Patient aged >75 years

3. Contrast medium used during angiography/angioplasty may lead to contrast induced nephropathy especially in patients who are:
a. Receiving intravenous fluid
b. Hypertensive
c. Diabetic
d. Taking aspirin
e. Aged >75 years

4. Which of the following conditions can often mimic the clinical features of Non-ST elevation acute coronary syndromes?
a. Aortic dissection
b. Aortic valve stenosis
c. Vasovagal syncope
d. Pneumothorax
e. Pneumonia

5. Following NSTEMI:
a. Beta blockers should be avoided in patients with impaired left ventricular function
b. Statin therapy can be stopped once lipid levels return to normal
c. Patients can resume driving after 2 weeks if they are asymptomatic
d. NSAIDs can be continued in patients who have no peptic ulcer disease
e. Cardiac rehabilitation has been found to especially beneficial in Patients <75 years of age

Answers To Questions

1. Which of the following conditions can cause an elevated troponin level?
a. True **b.** True **c.** True **d.** True **e.** True

2. In NSTEMI, which of the following factors are predictors of cardiac death and recurrent MI?
a. False **b.** True **c.** True **d.** True **e.** True

3. Contrast medium used during angiography/angioplasty may lead to contrast induced nephropathy especially in patients who are:
a. False **b.** False **c.** True **d.** False **e.** True

4. Which of the following conditions can often mimic the clinical features of Non-ST elevation acute coronary syndromes?
a. True **b.** True **c.** False **d.** True **e.** True

5. Following NSTEMI:
a. False **b.** False **c.** False **d.** False **e.** False

References

1. Guidelines for the management of non-ST segment elevation acute coronary syndromes. European Heart Journal, 2007, 28:1598–1660.

2. MI: secondary prevention. NICE clinical guideline 48. May 2007.

3. Antman EM, et al. The TIMI risk score for unstable angina/non-ST elevation MI: a method for prognostication and therapeutic decision-making. JAMA, 2000, 284:835–842.

4. Mehta SR, et al. Routine vesus Selective invasive strategies in patients with acute coronary syndromes: a collaborative metanalysis of randomised trials. JAMA, 2005, 293:2908–2917.

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SURVEY OF JUNIOR HOSPITAL DOCTORS' CONFIDENCE IN CARDIO-PULMONARY RESUSCITATION

Christopher Edward Hill and Kathryn Gay

For those that do attend cardiac arrests, often the junior doctors are the first doctors to arrive, and therefore are expected to carry out basic life support measures and initiate appropriate treatment.
Teaching & Training.

Abbreviations

CPR	Cardiopulmonary resuscitation
DNAR	Do Not Attempt Resuscitation order
FY1	Foundation Year 1 doctor
FY2	Foundation Year 2 doctor
ST1	Specialist Trainee Year 1
ST2	Specialist Trainee Year 2

Abstract

The majority of arrest teams are composed of junior doctors, yet these are those that are the most inexperienced or unconfident in cardiopulmonary resuscitation. Traditionally experience and competency at dealing with arrest situations is gained in the junior doctors years, however, with the advent of "hospital at night" and shift working, many are missing out. An anonymous questionnaire was emailed to the 75 junior doctors in a District General Hospital. Fifty replied, including 25 FY1s, 12 FY2s, 9 ST1s and 4 ST2s. Twenty-two per cent had never attended an arrest call, with the majority (38%) attending only 1 to 5. Confidence levels in performing various aspects of cardiopulmonary resuscitation were assessed, but overall 44% of junior doctors were unconfident in performing CPR. Ninety-eight per cent felt they would benefit from further training in arrest situations, but 65% thought this year would not provide them with the necessary experience. This was especially relevant to the FY1 doctors, 70% of whom were prepared to work extra to gain this experience, a view backed by higher trainees. Forty-eight per cent of those who had been to a crash call, reported that a debriefing session had never occurred, despite National Guideline recommendations, yet two-thirds felt it would be beneficial. 37.5% were called to crash calls inappropriately, where a DNAR order was in place; various reasons for this will be explored.

The majority of junior doctors at this hospital trust felt they would not receive enough exposure to acutely unwell patients and crash calls to develop the experience they need and believe patient care will suffer. Further training, being allowed to work nights and post-arrest call debriefings may all be beneficial in resolving this.

Introduction

The Royal College of Physicians and the General Medical Council both state that at least basic resuscitation training should be provided in the undergraduate medical course; because once qualified junior doctors form part of the cardiac arrest team and are therefore called upon to put these skills into practice. With the advent of hospital at night and the move towards shift working, many junior doctors feel that they are missing out on the important training opportunities that these situations traditionally provide, and are not being able to realise their training. For those that do attend cardiac arrests, often the junior doctors are the first doctors to arrive, and therefore are expected to carry out basic life support measures and initiate appropriate treatment.

As such a survey in a District General Hospital was performed to ascertain the experience level junior doctors have gained in such situations, and whether further training would be beneficial.

Methods

A questionnaire was created and emailed to the 75 junior doctors in both medical and surgical specialties in a District General Hospital, with the aim of determining:

- Whether junior doctors have opportunities to attend cardiac arrest calls
- The confidence levels of junior doctors in dealing with various aspects of cardiac arrest situations
- The levels of junior doctors training in dealing with cardiac arrest situations and whether they envisage further training would be beneficial
- Whether debriefing sessions occur following cardiac arrest calls as set out by the National Guidelines for cardiopulmonary resuscitation
- Whether a DNAR status is clearly stated at cardiac arrest calls in the experience of attending junior doctors
- Whether cardiopulmonary resuscitation is ever performed inappropriately in the experience of junior doctors attending cardiac arrest calls

The questionnaire was anonymous so the non-responders could not be recontacted. It comprised of a selection of closed and multiple choice questions and concluded with a free text box for further comments. Fifty junior doctors replied to the survey including 25 FY1s, 12 FY2s, 9 ST1s and 4 ST2s.

SURVEY OF JUNIOR HOSPITAL DOCTORS' CONFIDENCE IN CARDIO-PULMONARY RESUSCITATION

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Results

Of the 50 respondents, 25 (50%) were FY1 doctors, 12 (24%) were FY2 doctors, 9 (18%) were ST1 trainees and 4 (8%) were ST2 trainees. Eleven (22%) of the respondents had never attended a cardiac arrest call, with the majority (38%) attending between 1 and 5 "crash calls" since commencing work as a doctor.

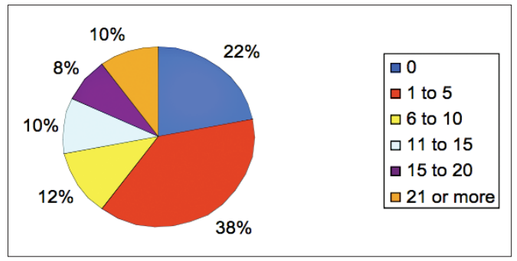

Figure 1: Number of cardiac arrest calls attended by junior doctors.

When questioned about the different aspects involved in managing arrest situations, 74% felt confident in assessing the airway, but only 56% felt confident in securing it. Seventy-eight per cent were confident in assessing breathing, but 6% felt unconfident in this field. Seventy-four per cent were confident in the assessment of circulation. The majority of respondents (44%) were not confident to secure IV access, but felt happy identifying shockable rhythms on an ECG and performing chest compressions. Junior doctors also felt unhappy administering resuscitation drugs, using a defibrillator and post-resuscitation care. Overall, only 56% felt confident in a cardiac arrest situation.

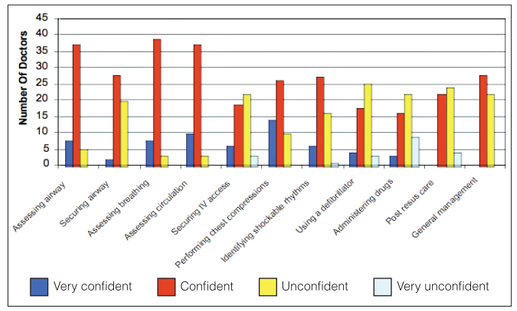

Figure 2: Confidence levels of junior doctors in various aspects of CPR.

All junior doctors had received some official training in the management of the critically ill patient, with the most common courses being "basic life support" (BLS) (74% had attended) and "intermediate life support" (ILS) (88% had attended).

Ninety-eight per cent felt they would benefit from further training in dealing with an arrest situation, but 65% felt their job this year would not provide them with the necessary experience. Seventy per cent of FY1 doctors said they would be prepared to work nights or extra hours to try and gain this experience, and this view was supported by FY2 doctors and above, the majority of whom thought FY1 doctors would not gain sufficient experience in arrest situations this year and 59% of the higher trainees thought patient care would suffer as a result.

The results highlight that post-cardiac arrest debriefing sessions happened very rarely, with 48% never having attended one and those that had only sometimes found them to be beneficial. However, two-thirds of those who had not had one felt it would be a beneficial experience.

37.5% reported attending a crash call where CPR was inappropriate due to a DNAR order already being in place. Of these 75% thought this was unacceptable. For those doctors who had attended cardiac arrest calls where there was no DNAR decision stated in the notes, 44% thought that this was due to the patient suddenly becoming unwell and therefore not previously an issue. Other potential contributory factors were highlighted as:

- Avoidance of discussion with the patient as it was thought to be too difficult (56%)
- Too difficult to discuss with the relatives (60%)
- Lack of senior member of staff to make the decision (52.1%)
- Not sufficiently informed of patient's details to make a decision (47.9%)
- Decision not even considered (50%)

However, 92% of FY2 doctors and above thought there had not been an increase in the number of inappropriate cardiac arrest calls in the last 6 months.

Discussion

In this survey of junior doctors, 44% stated they were unconfident in a cardiac arrest situation and 98% admitted they would like further training in this area. With junior doctors making up a large part of the cardiac arrest team, (which consists of 1 Spr, 3 FY2/ST1-2s and 1 FY1 at this hospital), this highlights an alarming situation. All the junior doctors have had basic training of how to perform in a crash call situation, but without the traditional exposure to acutely ill and arresting patients in the early house officer years, these skills have not been honed and the associated confidence developed. The majority of junior doctors surveyed had only attended between 1 and 5 arrest situations and two-thirds felt that their job this year would not provide them with the necessary experience they yearned for. It would appear that new initiatives, such as the hospital at night scheme (which prevents FY1's working nights at this hospital) and the European Working Time Directive, are limiting junior doctor's exposure to acutely sick patients. As a result they are missing out on vital experience, confirmed by 59% of the more experienced junior doctors who felt patient care will suffer as a result.

SURVEY OF JUNIOR HOSPITAL DOCTORS' CONFIDENCE IN CARDIO-PULMONARY RESUSCITATION

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Perhaps this is an issue that should be addressed earlier in a doctors training. If the Advanced Life Support (ALS) were incorporated into the curriculum of all medical schools and final year medical students were given more of an opportunity to shadow and be involved with the crash team, then by the time of graduation, some of that key experience may already be in place. Most of the junior doctors at this trust had already done ILS, and all had to pass an induction CPR refresher course before starting work, but to many this was not enough.

In Cardiff this year, a new initiative was launched with final year medical students becoming the "first responders" to help the ambulance service deal with cardiac arrest patients while at the same time gaining valuable experience. Perhaps this is a scheme that could be rolled out to other medical schools across the United Kingdom.

The other option is to revert back to allowing junior doctors to work nights and do more on calls, or at least to provide the opportunity to do so. After all, 70% of respondents stated they would be prepared to do this to gain more experience in acute or crash call situations. Though granted this would cause issues to arise surrounding working hours and pay banding, and so would perhaps need a voluntary aspect to it.

Just under half of the doctors who had been to a crash call, reported a debriefing session had never occurred, yet two-thirds felt it would be beneficial. The main topics doctors felt should be discussed at these debriefings were things the team did well, things the team did poorly and need to improve on and to try and identify things that could have been done to prevent a resulting negative outcome. National guidelines for cardiopulmonary resuscitation recommend that debriefing sessions should take place following every cardiac arrest call. This should ideally be lead by one of the more senior or experienced team members and can be a helpful source of non-judgemental feedback while allowing the more junior or inexperienced members the opportunity to ask questions. It also provides an opportunity for emotional support of team members and for anonymous critical event audit as part of support and clinical governance1.

37.5% of doctors were inappropriately called to a crash call where a DNAR order was already in place. One can only assume the underlying reason for this was poor communication, either verbally or in the form of unclear documentation in the notes. Many doctors arriving at crash calls found there was no DNAR decision stated in the notes, indicating therefore that the patient was for resuscitation until proven otherwise. Formal DNAR sheets placed at the front of the notes could help solve this problem and have been implemented successfully at other hospitals across the country.

Of those doctors who had attended cardiac arrest calls where there was no DNAR decision stated in the notes, 44% thought that this was due to the patient suddenly becoming unwell and therefore not previously an issue. Other factors felt to have contributed to the lack of a decision include the discussion being too difficult to have with patients and/or relatives. Doctors are trained throughout medical school about different aspects of communication and strategies to help with breaking bad news and discussing difficult topics, but its realisation can be a daunting prospect. Ideally junior doctors should try to observe their more senior and experienced team members doing this, and openly discuss the topic with them until they feel comfortable to do it themselves, although ultimately the decision over a patient's resuscitation status should come from the most senior member of the team. Interestingly however, over half of the junior doctors felt there was a lack of a senior member of staff to make this decision when needed.

SURVEY OF JUNIOR HOSPITAL DOCTORS' CONFIDENCE IN CARDIO-PULMONARY RESUSCITATION

Christopher Edward Hill and Kathryn Gay

Conclusions

The majority of junior doctors at this hospital trust feel they do not, and will not receive enough exposure to acutely unwell patients and crash calls to develop the experience they need to become better doctors. As a result just under half feel unconfident in acute arrest situations and most believe patient care will suffer. Nearly all feel they would benefit from further training.

Following CPR events, debriefings are not frequently done, despite the National Guidelines for cardiopulmonary resuscitation recommending them, and the majority of junior doctors feel they would be beneficial.

Most junior doctors believe there are a variety of reasons contributing to why patients do not have a clear DNAR status, although over a third have been called to an inappropriate crash call where a DNAR status was in place. However, 92% do not believe there has been an increase in inappropriate crash calls in the last 6 months.

Acknowledgements

We would like to thank all the junior doctors at King's Mill Hospital, part of the Sherwood Forest Hospitals NHS Foundation Trust, who took the time to complete the questionnaire. We would also like to thank Hazel Hilton for sending it out to all the appropriate doctors on our behalf.

Our grateful thanks to Mr Nicolas Watson (Surgical Specialist Registrar), Mr Krishnamurthy Badrinath (Surgical Consultant) and Mr Mukul Dube (Surgical Consultant) for their support and advice.

References

1. Morgan R, Westmoreland C. Survey of junior hospital doctors' attitudes to cardiopulmonary resuscitation. Postgraduate Medical Journal, 2002, 78:413–415.

Further Reading

1. Hulme J, McAuley DF. Cardiac arrest: addressing the training needs of medical students. StudentBMJ, March 2002, 10:45–88.

2. Philips PS, Nolan JP. Training in basic and advanced life support in UK medical schools: questionnaire survey. BMJ, 2001, 32:22–23.

3. Resuscitation Council (UK) Guidelines. Cardiopulmonary Resuscitation: Standards for Clinical Practice and Training (updated October 2004 version). A joint statement from The Royal College of Anaesthetists, The Royal College of Physicians of London and The Intensive Care Society and The Resuscitation Council (UK). Accessed on 7 January 2008 **(http://www.resus.org.uk/pages/standard.pdf).**

4. Royal College of Physicians London. Resuscitation from cardiopulmonary arrest. J R Coll Physicians Lond, 1987, 21:175–182.

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MANAGEMENT OF HYPERTENSION IN PRIMARY CARE

Amitesh Vasistha, Bhavika Vagani and Asim Zaidi

Mr Smith is a 59-year-old Caucasian gentleman, who presents to your GP clinic for the third time with a persistently raised blood pressure of 170/100.
Good Clinical Care.

Mr Smith is a 59-year-old Caucasian gentleman, who presents to your GP clinic for the third time with a persistently raised blood pressure of 170/100. Mr Smith reports no significant past medical history and denies any medication.

1. According to the current British Hypertension Society guidelines, Mr Smith would be classified as having:

a. Optimal blood pressure
b. High–normal blood pressure
c. Grade 1 hypertension
d. Grade 2 hypertension
e. Grade 3 hypertension

Answer: d

Variations in blood pressure are normal between individuals and therefore it is important to measure blood pressure accurately as this will determine long-term management. A calibrated sphygmomanometer attached to an appropriate size cuff for the patient's arm should be used in clinical practice. Blood pressure should be measured with the patient relaxed, any tight clothing removed and in the sitting position. The reading should be recorded to the nearest 2mm Hg and repeated three times.

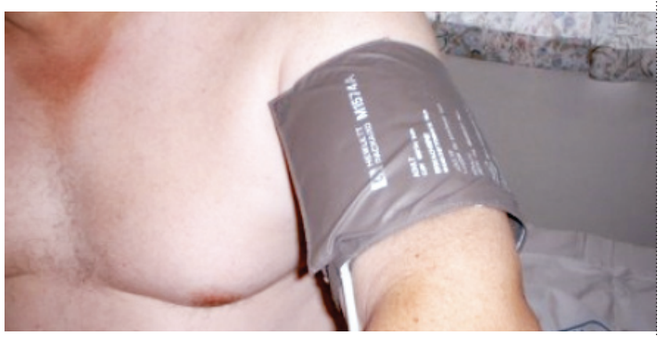

Figure 1: Inappropriate cuff size.

It is important not to treat patients on the basis of an isolated reading; a diagnosis of hypertension can only be made when blood pressure is persistently raised (140/90) on a minimum of two separate occasions.

	Systolic Blood Pressure (mm Hg)	Diastolic Blood Pressure (mm Hg)
Optimal blood pressure	<120	<80
Normal blood pressure	<130	<85
High–normal blood pressure	130–139	85–89
Grade 1 hypertension (mild)	140–159	90–99
Grade 2 hypertension (moderate)	<160–179	<100–109
Grade 3 hypertension (severe)	>180	>110
Isolated systolic hypertension (grade 1)	140–159	<90
Isolated systolic hypertension (grade 2)	>160	<90

Table 1: British Hypertension Society Classification of blood pressure levels.

2. What would be your initial management for Mr Smith?

a. No action
b. Repeat blood pressure measurement
c. Assess cardiovascular risk
d. Screen for secondary hypertension
e. Commence anti-hypertensive medication

Answer: c

Mr Smith has persistently raised blood pressure and the diagnosis of hypertension has already been established, therefore a further reading is not required. Mr Smith has no significant past medical history that obviously explains the reason for his hypertension.

A formal assessment of his cardiovascular risk prior to pharmacological intervention should be undertaken as recommended by NICE guidelines.

MANAGEMENT OF HYPERTENSION IN PRIMARY CARE

Amitesh Vasistha, Bhavika Vagani and Asim Zaidi

3. What initial investigations should you perform in order to assess cardiovascular risk?

a. FBC, U&E, urine dip, lipid profile, glucose, ECG
b. FBC, U&E, lipid profile, hormone levels
c. U&E, LFT, urine dip, glucose, ECG
d. U&E, TFT, urine dip, lipid profile, ECG
e. U&E, lipid profile, glucose, echocardiogram

Answer: a

Cardiovascular risk assessment involves monitoring for features of end-organ damage via the following parameters:

- Urea and electrolytes (U&E)
- Fasting lipid profile (total cholesterol, LDL/ HDL cholesterol, triglycerides)
- Fasting plasma glucose
- Urine for proteinuria
- 12 lead electrocardiogram

Smoking status and a positive family history of coronary heart disease should also be noted.

Patients should be offered lifestyle advice to reduce risk of ischaemic heart disease. This involves:

- Dietary modification (low calorie, low salt diet)
- Smoking cessation (advice and support)
- Low-level endurance exercise
  (aerobic exercise 30–60 minutes three to five times each week)
- Reduced alcohol intake
  (maximum 21 units/week for men and 14 units/week for women)

The majority of patients with raised blood pressure are diagnosed with essential hypertension, a multifactorial process, encompassing both genetic and environmental factors. If the patient presents with signs and symptoms suggestive of a secondary cause of hypertension (Conns syndrome, Cushings disease, renal artery stenosis, phaeochromocytoma, etc.) further investigation is warranted. Immediate referral is required if a phaeochromocytoma is suspected or if blood pressure exceeds 180/110mm Hg, suggesting malignant hypertension.

4. During the consultation, you elicit that Mr Smith has a history of asthma controlled on bronchodilators and has previously suffered from gout in the past. What pharmacotherapy should be initially commenced?

a. Amlodipine
b. Ramipril
c. Furosemide
d. Atenolol
e. Bendroflumethazide

Answer: a

Antihypertensive therapy is recommended for:

- Patients with persistent high blood pressure >160/100mm Hg.
- Patients at raised cardiovascular risk (10-year risk of cardiovascular disease >20% or existing cardiovascular disease or target organ damage) with persistent blood pressure of >140/90mm Hg.

According to NICE guidelines hypertensive patients aged 55 or older, or of Afro-Caribbean descent of any age, should receive a calcium channel antagonist or thiazide diuretic as first line therapy. Patients younger than 55 years of age should be commenced on an angiotensin converting enzyme (ACE) inhibitor.

Hypertension in subjects of Afro-Caribbean origin is associated with low renin concentrations, explaining why beta blockers and ACE inhibitors are rather ineffective in this group of patients. However, these agents may be effective when combined with drugs that raise renin levels (e.g. diuretics and calcium channel antagonists).

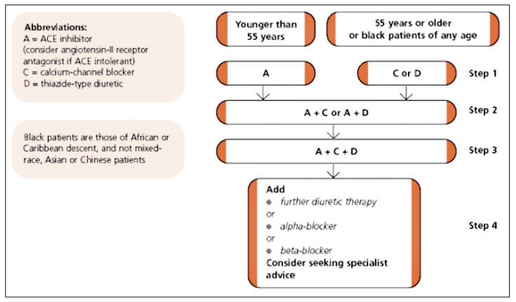

Table 2: Algorithm for the treatment of hypertension (adapted from NICE Guidelines – management of adults with hypertension in primary care).

MANAGEMENT OF HYPERTENSION IN PRIMARY CARE

Amitesh Vasistha, Bhavika Vagani and Asim Zaidi

In light of Mr Smith's history of gout, a thiazide diuretic may precipitate hyperuricaemia and therefore would not be recommended. A calcium channel antagonist would therefore be the most appropriate choice for this gentleman.

Beta adrenorecptors are no longer the main stay for the treatment of hypertension and are considered as the fourth step for hypertensive control.

In addition, beta blockers cause bronchoconstriction and would therefore be contraindicated in this scenario, as Mr Smith is asthmatic. Beta blockers may be considered for use as an antihypertensive agent for:

- Women of childbearing age
- Individuals with an increased sympathetic drive
- Individuals with an intolerance or contraindication to ACE inhibitors

5. You have established that Mr Smith has a 5% cardiovascular risk profile and has been commenced on antihypertensive medication as appropriate. Which of the following blood pressure readings is his treatment target?

a. <130/75
b. <130/80
c. <130/85
d. <140/80
e. <140/90

Answer: e

NICE guidance advocates a treatment target of <140/90mm Hg for non-diabetic patients. Lower targets are recommended for those with diabetes:

- Type 2 diabetes: <140/80mm Hg or <135/75mm Hg
  if microalbuminuria or proteinuria present in urine
- Type 1 diabetes: <135/85mm Hg or <130/80mm Hg
  with renovascular disease (highest risk of cardiovascular disease)

Although it may not be possible to achieve target values in all patients, any lowering of blood pressure is beneficial to reduce mortality from cardiovascular disease.

Following the introduction of antihypertensive medication, progress must be monitored. The frequency of follow-up is dependent upon blood pressure severity, patient compliance, complexity of the treatment regime and development of side effects and complications.

An annual review should be performed once hypertension is controlled; blood pressure and weight should be documented and urine tested for proteinuria. This consultation also provides the opportunity to reinforce lifestyle modification, adherence to therapy and to reassess cardiovascular risk and so optimising patient care.

References

1. Williams B, Poulter NR, Brown MJ, Davis M, McInnes GT, Potter JF, et al. Guidelines for management of hypertension: report of the fourth working party of the British Hypertension Society, 2004–BHS IV. J Hum Hypertension, 2004, 18:139–185.

2. NICE. Management of adults with hypertension in primary care. June 2006.

3. The management of hypertension in primary care: updated guidance from NICE part 1. MeReC Bulletin, October 2006, 17:1–2.

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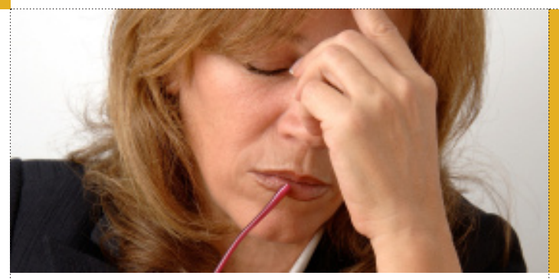
CHRONIC FATIGUE SYNDROME: AN UPDATE

K Khanna, H Polacarz and M Kalla



Women suffer from Chronic Fatigue Syndrome more commonly than men for reasons which are unknown. Good Clinical Care.

Introduction

Fatigue is a feeling of weariness, tiredness or lack of energy and is an extremely common presenting complaint in primary care and clinical medicine. It is difficult to define and can be due to systemic pathology or underlying psychiatric illness. The term "chronic fatigue" is frequently used, and has been defined as fatigue lasting more than 6 months but without any associated symptoms that are characteristic of chronic fatigue syndrome[1].

Chronic fatigue syndrome (CFS) is a controversial diagnosis and the definition has been much debated in recent years. It is a medically unexplained illness characterised by "chronic (often relapsing but always debilitating) fatigue, lasting for at least 6 months and, in some cases, for much greater lengths of time, causing impaired overall physical and mental functioning"[1].

A UK study of chronic fatigue in primary care has shown the symptom to impose substantial economic costs on society, mainly in the form of care provided by friends and family and lost employment2. A similar US study estimated a 37% decline in household productivity and a 54% reduction in labour force productivity among people with CFS. The estimated annual total value of lost productivity in the USA was $9.1 billion, representing approximately $20,000 per person with CFS3.

Prevalence

Studies based in primary care have reported the prevalence of chronic fatigue syndrome to be between 0.2–2.6% depending on the criteria on which the diagnosis is based[4]. It is primarily a disorder of young to middle aged adults, but cases in children have been recognised. Population surveys found similar rates across ethnic groups and socio-economic classes[5]. Women suffer from CFS more commonly than men for reasons which are unknown, although increasing evidence suggests a genetic influence on the illness[5].

Aetiology

Chronic fatigue syndrome, also known as myalgic encephalomyelitis, is an illness of unknown aetiology. Indeed, the existence of CFS as a true medical illness is often questioned. Certain infectious illnesses (such as Q fever, Chlamydia pneumoniae and viral meningitis) can trigger CFS and it has been suggested that CFS is in fact an immunological manifestation of these infectious diseases6. This proposed aetiology continues to be questioned due to the heterogeneous nature of the syndrome and the lack of diagnostic biological markers. However, preliminary research has shown that a low cortisol level has repeatedly been found to be associated with chronic fatigue syndrome though this may be secondary to the physical inactivity and sleep disturbance associated with the syndrome[7].

Clinical Features & Diagnosis

The International Centre for Disease Control first published a definition of CFS in 1988. Major and minor criteria were as follows[8]:

Major criteria:

1. New onset of persistent or relapsing fatigue that does not resolve with bed rest and that is severe enough to reduce average daily activity to less than 50% of the patient's pre-morbid activity level for at least 6 months
2. Fatigue that is not explained by the presence of other evident medical or psychiatric illnesses

Minor criteria:

Symptom criteria:

1. Mild fever
2. Sore throat
3. Painful lymph nodes in the anterior or posterior cervical or axillary distribution
4. Unexplained generalised muscle weakness
5. Muscle discomfort or myalgia
6. Prolonged (≥24 hours) generalised fatigue after exercise
7. Generalised headaches
8. Migratory arthralgia without joint swelling or redness
9. Neuropsychologic complaints
10. Sleep disturbance

CHRONIC FATIGUE SYNDROME: AN UPDATE

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Physical criteria:

1. Non-exudative pharyngitis
2. Low-grade fever

A diagnosis of CFS required 2 major criteria along with either 6 symptom criteria + 2 physical criteria or 8 symptom criteria.

A large number of definitions have since been created in an attempt to clarify the diagnosis of CFS. The key points prevail, however, and it is essential to exclude other organic causes before diagnosing chronic fatigue.

In practice, certain clinical features are common to chronic fatigue syndrome, and if recognised early, can lead to early diagnosis of the condition. These are:

1) Typical upper respiratory tract infection or viral infection followed by relatively sudden onset of profound fatigue
2) Fatigue persists after acute phase of infection, accompanied by sleep and cognitive disturbance
3) Symptoms exacerbated by attempts at strenuous physical activity
4) Sudden onset of the disease in highly functioning individuals, often with a past psychiatric history of note

Patients with CSF are often diagnosed with depression but this may be secondary to their inability to perform activities of daily living without true psychiatric illness. Patients with CFS also typically complain of post-exertional fatigue following normal activities and even after prolonged periods of rest or sleep[6].

It is important to note that any psychiatric condition including alcohol misuse and anorexia are excluded from a diagnosis of CFS.

Laboratory tests neither confirm nor definitely exclude CFS but are essential in first line investigation to exclude true pathological illness[9].

First Line Investigations For CFS
Full blood count
C-reactive protein
Erythrocyte sedimentation rate
Urea and electrolytes
Thyroid function tests

The most consistent laboratory abnormality in patients with CFS is an extremely low erythrocyte sedimentation rate (ESR), which approaches zero. Typically, patients with CFS have an ESR of 0–3mm/h. If the ESR is elevated or even in the high-normal range, another diagnosis is suggested. There may also be an elevated IgM/IgG Coxsackie B virus titer, elevated IgM/IgG HHV-6 titer or elevated IgM/IgG Clamydia pneumoniae titer, but these results can be inconsistent[9].

Differential Diagnosis

As chronic fatigue syndrome is a diagnosis of exclusion, a number of differential diagnoses need initial ruling out before a patient is labelled with CFS. These differentials fall across a wide spectrum of health[10].

Infectious	Haematological	Rheumatological
Chronic Epstein-Barr virus	Anaemia	Fibromyalgia
Influenza	Lymphoma	Systemic lupus erythematosus
HIV infection	Malignancy	Polymyalgia rheumatica

Endocrine	Psychiatric	Other
Myxoedema	Depression	Chronic disease (e.g. liver)
Diabetes	Schizophrenia	Alcohol/substance misuse
Adrenal insufficiency	Bipolar affective disorder	Iatrogenic

Table 2: Differential diagnosis of CFS.

Management

Continuing controversy over the existence of CFS, together with the contradictory research findings over aetiology and treatment had made the management of CFS extremely complicated. Current management is largely supportive. The only medical treatment that is considered curative is antichlamydial therapy in patients with elevated Clamydia pneumoniae levels.

Antidepressants, namely SSRIs are common therapy for patients with CFS. Although clinical trials of tricyclic antidepressants have not produced definitive results it is believed that they do promote an acceptable sleep pattern and alleviate pain9.

Cognitive behavioural therapy (CBT) has had promising effects on the severity of CFS as it seems to work by addressing patient's cognition and behaviour to identify coping strategies. The role of exercise in patients with CFS is also crucial as long-term physical inactivity can lead to physical deconditioning that further complicates the symptoms of the syndrome9. Trials have shown that 25% of individuals treated with CBT regarded themselves as cured 5 years after treatment9.

Due to the ambiguity surrounding CFS it is essential that management involves all medical and lifestyle aspects including exercise, optimum diet, sleep, hygiene, antidepressants and cognitive behavioural therapy.

CHRONIC FATIGUE SYNDROME: AN UPDATE

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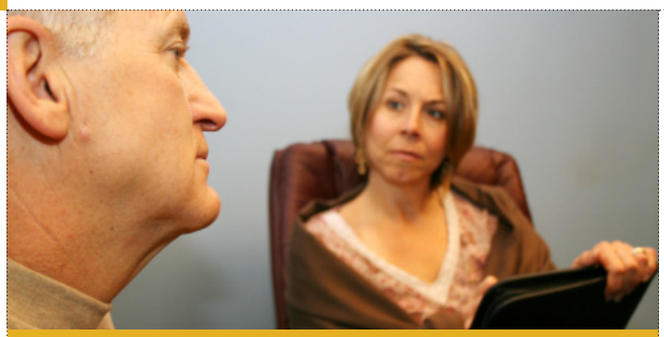

The need for careful diagnosis is further emphasised by the poor prognosis of the syndrome and the need for a multifaceted approach to management through drug therapy, psychological therapy and lifestyle changes.
Good Clinical Care.

Prognosis

Among those presenting in primary care with fatigue lasting less than 6 months, 40% improve6. However, without treatment, full recovery from CFS is rare. Furthermore, any coexisting psychiatric disorder reduces the chances of recovery6. Only 6% of those with CFS will return to pre-morbid levels of functioning with the majority remaining significantly impaired11. Prognosis is better in children with >50% of children showing definite improvement as in the medium term[11].

Conclusion

CFS is a complex phenomenon which is poorly understood and often questioned in relation to its true pathological existence. Fatigue, as a symptom, is extremely common in primary care and medical or psychiatric pathology needs to be excluded before a patient is "labelled" with CFS. The need for careful diagnosis is further emphasised by the poor prognosis of the syndrome and the need for a multifaceted approach to management through drug therapy, psychological therapy and lifestyle changes.

References

1. Fukuda K, Straus SE, Hickie I, Sharpe MC, Dobbins JG, Komaroff A and the International Chronic Fatigue Syndrome Study Group. The chronic fatigue syndrome: a comprehensive approach to its definition and study. Ann Intern Med, 1994, 121:953–959.

2. McCrone P, Darbishire L, Ridsdale L, Seed P. The economic cost of chronic fatigue and chronic fatigue syndrome in UK primary care. Psychological Medicine, 2003, 33(2):253–261.

3. Reynolds KJ, Vernon SD, Bouchery E, Reeves WC. The economic impact of chronic fatigue syndrome. Cost Effectiveness and Resource Allocation, 2004, 2:4.

4. Wessely S, Chalder T, Hirsch S, Wallace P, Wright D. The prevalence and morbidity of chronic fatigue and chronic fatigue syndrome: a prospective primary care study. Am J Public Health, 1997, 87:1449–1455.

5. Ranjith G. Epidemiology of chronic fatigue syndrome. Occupational Medicine, 2005, 55:13–19.

6. Reig S, Chalder T. Chronic Fatigue Syndrome. BMJ, 2000, 320:292–296.

7. Cleare AJ. The HPA axis and the genesis of chronic fatigue syndrome. Trends Endocrinol Metabol, 2004, 15: 55–59.

8. Holmes GP, Kaplan JE, Gantz NM. Chronic fatigue syndrome: a working case definition. Annals of Internal Medicine, 1988, 108:387–389.

9. White P. What Causes Chronic Fatigue Syndrome? BMJ, 23 October 2004, 329:928–929 (reference no. doi:10.1136/bmj.329.7472.928).

10. Hawk C, Jason LA, Torres-Harding S. Differential diagnosis of chronic fatigue syndrome and major depressive disorder. Int J Behav Med, 2006, 13(3):244–251.

11. Joyce J, Hotopf M, Wessely S. The prognosis of chronic fatigue and chronic fatigue syndrome: a systematic review. Q J Med, 1997, 90:223–233.

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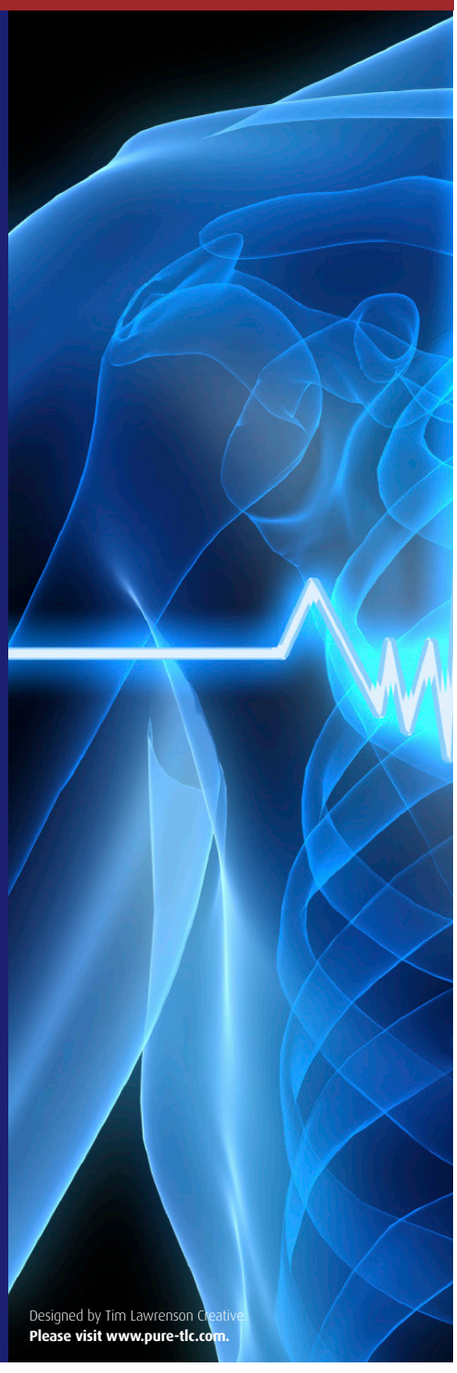

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