FUTURE PERSPECTIVES IN CARDIOLOGY*

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Experts from complementary disciplines presented their experiences in planning effective cardiology services, and perspectives on coronary heart disease (CHD) policy from both north and south of the Border were given, in the morning sessions. The importance of communication and consultation with all, including patients, in planning and earmarking funding for delivering cardiac services was stressed. In the afternoon, three senior cardiologists outlined their approaches to three of the commoner cardiac presentations: breathlessness, palpitations and chest pain. The conference closed with a debate entitled 'Non-invasive cardiac investigations should only be performed in hospital and supervised by cardiologists.' This was as entertaining as it was informative, with both speakers making impassioned pleas for and against.

SESSION 1

NETWORKS FOR CHD

Chaired by: Dr David Newby, BHF Senior Lecturer in Cardiology, University of Edinburgh

Multidisciplinary networks in cardiac care

Ms Maree Barnett, Project Manager and Specialist Nurse Adviser, Department of Health, London

The World Health Organisation defines a network as 'a grouping of individuals, organisations and agencies organised on a non-hierarchical basis around common issues and concerns which are pursued proactively and systematically based on commitment and trust'. The emerging importance of networks for patients and clinical staff in the UK was emphasised in the light of decentralisation of health service provision. The traditional model of cardiac care provision, centred on tertiary referral centres, will be replaced with a patient-centred ethos and greater emphasis on the role of primary care.

For a network to function successfully, effective communication must be ensured between different disciplines. This was illustrated with the example of a heart failure network that involves heart failure nurses, cardiologists, primary care team members, pharmacists, a palliative care team, physiotherapists, a dietitian and rehabilitation specialists. The aim of the network is to manage and integrate different disciplines into the patient-care pathway, breaking down barriers, engendering teamwork and harmonising services. Benefits include coordinated care and a standardised approach that is applicable throughout the country, which obviates the development of brand-new networks and provides a benchmark for quality assurance and audit. Networks also provide rational frameworks for service planning, funding, research and development.

When developing a network it is important to have an all-inclusive approach with links to all relevant disciplines, which should be represented on the advisory board. The boundaries and priorities must be clearly established, and all potential resources identified. Keys to successful implementation include having clearly identified objectives, the development of standards and protocols, and agreed accountabilities between different contributors. It is also important to have the support from influential parties including the Chief Executive and Strategic Health Authority (or Health Board). Strong leadership and good communication remain at the heart of a successful network.

Extended nursing roles and nurse prescribing were identified as examples of the future potential contribution of networks to cardiac care. By achieving universal agreement, apparent legal or bureaucratic hurdles can be overcome, allowing nurses to perform procedures or administer drugs such as thrombolytics within the context of clearly pre-defined clinical scenarios and guidelines. Supplementary prescribing by nurses and pharmacists was introduced in April 2002 and has been successful, allowing the prescription of specific medicines within a defined management plan and with the agreement of the patient and supervision of a named independent prescriber.

The session ended by presenting 'The Coronary Heart Disease Collaborative' as an example of a successful cardiac network based in England, which is working in five areas: secondary prevention, patients with suspected or confirmed myocardial infarction (MI), heart failure, revascularisation and cardiac rehabilitation. This collaborative approach is based on the principles of coordinating patient care, improving delivery and matching demand with capacity. It focuses on drawing together contributors from different disciplines, making

*This is the report of the lectures given at the Future Perspectives in Cardiology Symposium held at the Royal College of Physicians of Edinburgh on 10 April 2003.

them feel part of one system with a shared goal. This concept for healthcare delivery aligns closely with the managed clinical networks in Scotland.

Primary–secondary care: bridging the gap for cardiac patients

Dr Chris Baker, Lead Clinician, Dumfries and Galloway Cardiac Managed Clinical Network

A managed clinical network is a linked group of health professionals from primary, secondary and tertiary care, working in a coordinated way, unconstrained by existing professional or Health Board boundaries, to ensure provision of high-quality, clinically effective services.[1] Recognition that managed clinical networks are important for managing patient movement into and out of hospitals is not new, and was highlighted in the definition (above) taken from the Acute Services Review for Scottish hospitals in 1998. In Scotland, the aim was to develop a CHD network consisting of local networks made up of primary and secondary care teams, and feeding into a national intervention network. The experience of a pilot local network in Dumfries and Galloway was presented (Figure 1). The importance of involving representatives from all disciplines from the outset was underlined. This produced a sense of cooperation and allowed the waiting list for cardiology appointments to be reduced by transferring suitable referrals to the general physicians. Perhaps the most significant change is that patients and clinicians are now involved in the planning of cardiac services and resource allocation in Dumfries and Galloway. Patient representatives received training to enable them to participate and be effective in this role. The personal experiences of patients have been used to identify areas that need improvement and patient representatives have become powerful advocates for change, helping to shape health policy. The managed clinical network has facilitated the development of out-of-hospital thrombolysis, particularly relevant for a rural community, and has produced unified care protocols, standardising the quality of care throughout the region. Clinical effectiveness is monitored by an annual quality assurance programme that is evidence-based, involves patient representatives and is peer-reviewed. More details on the Dumfries and Galloway managed clinical network for CHD are available from the website (www.show.scot.nhs.uk/mcn).

Networks in secondary care

Dr James McLenachan, Consultant Interventional Cardiologist, Yorkshire Heart Centre, Leeds

The interventional centre perspective on clinical networks was delivered in this lecture. Before moving to its new facility in 2000, the Leeds angioplasty service suffered from having too few cases, too many operators, no on-call rota for interventionists, no formal referral system and a highly variable length of time for patient transfer to district general hospitals (DGHs). The

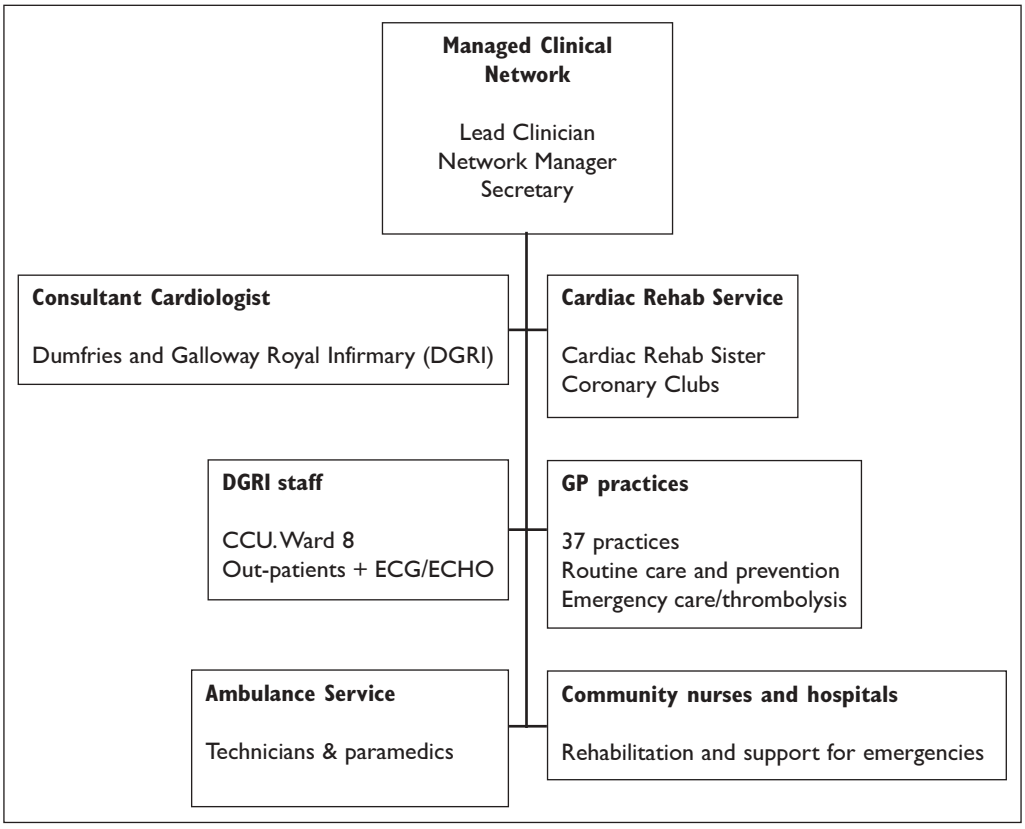

FIGURE 1

The structure of the Dumfries and Galloway Managed Clinical Network for Coronary Heart Disease, launched in July 2001.

operators in Leeds wanted to increase their interventional workload and shorten the waiting time for both elective and acute cases, in part by reducing the number of diagnostic coronary angiograms. The DGH cardiologists wanted more rapid transfer of patients for intervention, but wanted to continue care and follow-up of the patients that were transferred. These targets were met by utilising the facilities in DGHs for diagnostic coronary angiography, and shifting the workload for this investigation from the tertiary centre to the referring hospital. A formal referral protocol was devised and managed by a trained nurse interventional coordinator. Dedicated lists and beds were set aside for acute intervention, and these were managed according to clinical need and waiting time. The nursing role was also extended to patient clerking and a pre-admission clinic for elective angioplasties. Consultants who did not want to perform the minimum 75–80 percutaneous interventions per year recommended by the British Cardiac Intervention Society, to maintain competency, dropped out of the intervention on-call rota.

These changes resulted in fewer remaining interventionists but each was performing a higher volume of procedures. To maximise throughput, patients transferred from DGHs were often transferred back on the day of the procedure, using a dedicated ambulance service. This ensured continuity of care by the DGH cardiology team. An angiogram had already been carried out in over 50% of DGH referrals by the time they reached the interventional centre, allowing the case-load to be more efficiently managed. An immediate benefit of the new referral system was better representation of patients from outside the Leeds catchment area. More patients from the much bigger combined DGH population were coming through for angioplasty. One drawback of this approach was that audit and research became more difficult because the interventional cardiologist who performed their procedure did not follow-up patients. This problem was partly circumvented by having dedicated research nurses who travelled out to DGHs and collected information on patient demographics and outcome. The importance of good communication and detailed handover information was emphasised to ensure continuity for patients who were receiving care from more than one consultant.

SESSION 2

WORDS INTO ACTION – DELIVERING THE GOODS FOR CHD

Chaired by Dr Adrian Brady, Consultant Cardiologist, Glasgow Royal Infirmary

Delivering CHD policy in primary care

Dr Caroline Morrison, Consultant in Public Health, Greater Glasgow Health Board

The audience was reminded that the greatest impact on CHD mortality figures would be made through primary and secondary prevention directed and managed in primary care. Gaining the agreement of primary care trusts to implement secondary prevention is a fundamental step, and this should involve both allocation of adequate funding and resources and a clear outline of what is to be done within the framework of the new GP contract. Secondary preventive policies should be uniform throughout secondary and primary care organisations with agreed guidelines and protocols to avoid confusing patients with slightly differing messages. The costing of policies should be well-planned and any hidden costs identified, such as the cost of transport to the practice surgery for more frequent health checks. These costs need to be placed in the context of the overall health budget remembering that health priorities may differ slightly between different regions. The necessary support services must also be in place, such as help groups for smoking cessation and dietary advice.

A plan for secondary prevention provision will need to identify a target population and the person responsible for delivering it. Registers of patients and their medical conditions in GP practices are of varying quality. One difficulty is the multiple ways that patients with CHD suitable for secondary preventive measures can be coded or listed. Strategies are required for progressing through the list, identifying priorities and also making sure that new patients are continuously added. A system for recording health promotion activity needs to be established. This should use nationally agreed codes such as continuous morbidity recording, and should also interface easily between primary and secondary care. Computer databases are the most attractive in this context but have disadvantages, being costly, requiring extensive technical support and taking a long time to set up.

A variety of health professionals may be responsible for delivering secondary prevention. Most will be specialised nurses who have received specific training. These nurses may move between different GP practices within a city. In rural areas, existing community nurses may need to be trained further to take on the role. In some cases, secondary prevention may be funded and delivered within a primary care environment as an outreach service from the hospital. Potential for overlap with other services, such as cardiac rehabilitation, should be recognised.

SYDNEY WATSON SMITH LECTURE

Delivering the National Service Framework for CHD

Dr Roger Boyle, National Clinical Director for CHD, Department of Health, London

The National Service Framework for CHD was launched in March 2000.[2] This identified CHD as a government priority area aiming at tackling the high disease burden. Additional driving forces for this were the variability in standards of heart disease care throughout England and unfavourable comparisons with Europe made by the media. National standards of care were produced with service models, targets and milestones to be reached.

Immediate priorities for the framework were identified, including smoking cessation, rapid-access chest pain clinics and reducing the 'call-to-needle' time for thrombolysis in acute myocardial infarcts. Local implementation teams involving primary care trusts were developed; a national audit programme to monitor and compare standards was established; and multidisciplinary collaboratives in such areas as secondary prevention, management of angina, acute MI, heart failure, cardiac rehabilitation and revascularisation have all proved powerful levers of change. In other areas, collecting and disseminating information, and workforce planning and development have proven more challenging largely because of technical difficulties in disease-recording and problems with generalising plans to regions with different needs.

To date, over £800 million has been spent on the CHD national service frameworks in England: this has come from the Treasury as part of increased spending on the NHS, as well as from other sources such as lottery funding. The target of a 40% reduction in standardised cardiovascular mortality by 2010 is projected to be on course at the halfway point. Important successes have included the 'school fruit' scheme, a complete ban on smoking advertising, and primary care collaboratives for secondary prevention through dedicated practice-nurses.

Diagnosis and access have been improved by the widespread introduction of rapid-access chest pain clinics, initiatives to reduce out-patient waiting times and expansion of hospitals with new or replacement cardiac catheterisation laboratories (86 new facilities in England over the last three years). The waiting time for bypass surgery has been reduced by a combination of investment and the patient choice scheme, allowing patients to shorten their time to surgery by offering them the choice of having their operation in a different centre.

Parallel improvements have taken place in acute care. Ambulance response times have been reduced and the introduction of telemetry and paramedic-led thrombolysis have impacted on 'door-to-needle' time allowing ambulance crews to alert hospitals of incoming 'hot cases' or instigate early treatment. Public access defibrillation has been introduced and most successes from it have been in major stations and airports; the cost efficacy of public access defibrillators remains to be clearly established.

Continuing care and follow-up care strategies are also being implemented for heart failure and cardiac rehabilitation. The National Institute for Clinical Excellence published a guideline for heart failure in July 2003 and England has adopted the Scottish SIGN guidelines for cardiac rehabilitation.

SESSION 3

HOW TO MANAGE THE CARDIAC PATIENT

Chaired by: Dr Neil Grubb, Consultant Cardiologist, Royal Infirmary of Edinburgh

The breathless patient

Dr Stuart Shaw, Consultant Cardiologist, Western General Hospital, Edinburgh

In order not to feel breathless, patients require 'good air', adequate ventilation-perfusion of the lungs, effective oxygen diffusion across the alveolar-arterial barrier and adequate oxygen carriage capacity within the blood.

Air quality becomes important at high altitude (>2,500 m) where the partial pressure of oxygen is reduced to a degree that exposes climbers to the risk of altitude sickness;[3] all people moving to high altitude are at risk and 25% will develop signs of acute mountain sickness within three days. Usually the condition is mild and settles, but patients with underlying heart or lung conditions may suffer aggravation of hypoxia, and patients with primary pulmonary hypertension in particular should not leave sea level. Risk factors for developing more severe disease include rapid ascent, higher altitude, vigorous exercise, younger age, hypothermia and alcohol consumption.[3] In the course of air travel, planes ascend to 10,000 m and the cabin is pressurised to 2,438 m. Healthy subjects may desaturate to 85% and patients with a resting oxygen saturation at sea level of less than 91%, or 91–5% with such added risk factors of CO_2 retention, FEV_1 less than 50%, restrictive lung disease or severe cardiac disease, should be considered for supplemental in-flight oxygen.[4] Cyanosed patients should probably not go on long-haul flights, and where there is doubt, flight simulation tests can be performed at fitness-to-fly clinics where the patient is monitored while breathing a reduced oxygen concentration. These guidelines are not rigorously applied and many patients are not assessed or advised before travelling by air. There then is potential for conflict between interfering with the patient's right to travel freely and ensuring the safety of the flight through

not having to divert, at significant cost and inconvenience, because of a sick patient. Guidelines for fitness to fly can be found on the British Thoracic Society website (http://www.brit-thoracic.org.uk/docs/flyingguidelines.pdf).[2]

At the cardiology clinic, the history examination and investigations must exclude underlying or associated respiratory disease, anaemia and thromboembolic disease; psychological causes of breathlessness must also be considered Underlying cardiac conditions are detected by eliciting a history of cardiac symptoms and clinical signs of heart disease, and taking into account the rhythm and electrocardiogram (ECG) complex morphology and echocardiography. The chest X-ray is only usually required if no underlying cause of dyspnoea can be found, or the clinical findings suggest a respiratory cause.

Cardiac patients develop dyspnoea when pressure and stretch receptors located in the airways are activated following pulmonary venous congestion and expansion.[5] This may be compounded by hypoxia, detected by chemoreceptors, which is caused by a combination of ventilation/perfusion mismatch and air trapping in patients with pulmonary oedema. Fluid shift from the extremities and splinting of the diaphragm when lying flat exacerbates these processes resulting in the symptom of orthopnoea.

The concept of the cardiac failure 'staircase' is helpful when investigating a patient for a cardiac cause of dyspnoea (Figure 2). The cardiac rhythm and all components of the 'staircase' must be evaluated and often there will be several jointly occuring abnormalities.

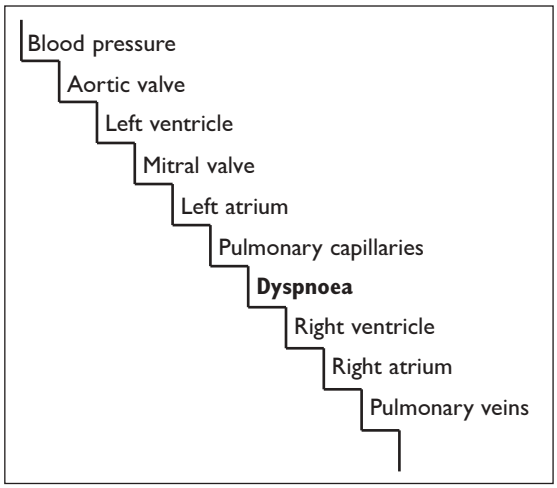

FIGURE 2
Cardiac failure staircase. Each component should be considered when evaluating a patient with a possible cardiac cause of breathlessness.

Echocardiography has largely replaced ventricular angiography as the 'gold standard' for assessment of cardiac function. Left ventricular function, valvular stenosis, or reflux and pulmonary artery pressures can also be reliably estimated by echocardiography. Patients presenting with signs and symptoms of cardiac failure and normal systolic function frequently have abnormal measurements during diastole: widespread use of echocardiography has led to increased recognition of patients with isolated diastolic dysfunction.[6] The inflow across the mitral valve during diastole can be split into early (E) and late atrial (A) components: normally the E wave velocity is slightly greater than the A wave. A dominant E wave velocity with a short deceleration and diminished A wave velocity indicate a restrictive pattern and elevated left atrial pressure.[6] Patients with this pattern may benefit from diuretic therapy even if systolic function appears preserved.

When assessing the breathless patient, it is the task of the cardiologist to detect rare causes of cardiac failure: all patients with unexplained cardiomyopathy should have ferritin levels measured (haemachromatosis), thyroid function tests (altered function) and 24-hour urinary collections for catecholamines (phaeochromocytoma); the latter may develop cardiomyopathy despite having normal blood pressure recordings in a clinic situation. Further pitfalls include pleural effusions (best quantified by ultrasonography); constrictive pericarditis (difficult to pick up on echocardiography); and para-prosthetic mitral valve regurgitation (often silent on chest auscultation and missed on transthoracic echocardiography). In addition, patients with extensive three-vessel coronary artery disease can present with breathlessness on exertion and there may be no specific ST segment changes before they have to stop during treadmill testing.

Testing for N-terminal pro-brain natriuretic peptide (released into the circulation in response to ventricular overload) will help in future to assess patients with possible cardiac failure. This test is close to 90% sensitive and specific for left ventricular systolic dysfunction; it can be applied as a screening tool in patients with existing clinical labels of heart failure who have never had an echocardiogram, in triaging patients with symptoms of cardiac failure before referring for echocardiography and in screening patients at high risk of cardiac failure.[7]

The patient with palpitations

Dr Mike Griffith, Cardiologist and Electrophysiologist, University Hospital, Birmingham

It is important to elicit carefully the duration of symptoms when taking a history in a patient with palpitations. Momentary episodes lasting less than three to four seconds are usually caused by ectopy. Brief episodes lasting less than 30 seconds, defined as non-

sustained tachyarrthymias, are most frequently caused by ventricular tachycardia (VT) or atrial fibrillation (AF). The differential diagnosis of sustained tachyarrthymias is wide including supra-ventricular tachycardias.

Ectopic beats may be felt as a missed beat, an extra beat or a thud in the chest; the missed beat is caused by contraction of a poorly filled ventricle and the patient senses the transient drop in cardiac output; the thud results from the post-ectopic compensatory pause and increased filling of the ventricle before the next contraction. Ectopic beats are generally benign and require re-assurance only. Ventricular ectopics following MI or after exercise are markers of a diseased myocardium and are associated with a small increase in the risk of sudden cardiac death.[8] Suppression with medication is not helpful and indeed trials conducted in the 1980s in survivors of MI, using class I anti-arrhythmics, although achieving effective suppression of ectopy conferred increased mortality in the treated groups.[9] Investigation or treatment of patients with ectopy is generally not required, although occasionally a 24-hour ambulatory ECG will allow the patient to connect their symptom with an extra beat on the recording and can provide helpful reassurance.

Patients with non-sustained tachycardias have symptoms for more than a few seconds. Occasionally the resting 12-lead ECG has helpful clues in it such as atrial ectopics (suggesting underlying paroxysmal AF) or ventricular ectopics (indicating the possibility of non-sustained VT). Symptoms of presyncope or syncope are worrisome, indicating VT, and the 24-hour recording is often diagnostic. Occasionally, event recorders worn for a longer period or an implantable loop recorder, inserted subcutaneously under local anaesthetic, may be required to pick up less frequent episodes.

Consideration should always be given to the risk of stroke and need for anticoagulation in patients with paroxysmal AF. Patients under the age of 65 with structurally normal hearts derive no benefit from warfarin and can be maintained on aspirin. Older patients with risk factors for cerebrovascular disease including hypertension and diabetes mellitus or patients with structural heart disease should be considered for warfarin treatment. Previous strokes or transient ischaemic attacks are a compelling indication for warfarin.[10] Beta-blockers are the first-choice therapy for suppression in patients with troublesome symptoms during the attacks.

Patients with structurally normal hearts and non-sustained VT are most likely to have right ventricular outflow tachycardia. The ECG typically shows right axis deviation with a left bundle branch block pattern.[11] The tachycardia may be brief or sustained and is often catecholamine-sensitive, being triggered by exertion. Symptoms, including exertional syncope, may be alarming but the prognosis is excellent, and the arrhythmia can be curable by radiofrequency ablation of the re-entrant circuit in the right ventricular outflow tract. Patients with ischaemic left ventricular dysfunction and ventricular tachycardia have, by contrast, a poor prognosis. The Multicenter Automatic Defibrillator Implantation Trial (MADIT) demonstrated improved survival in patients receiving an implantable defibrillator who had a previous Q-wave MI, a left ventricular ejection fraction of less than 35%, documented non-sustained VT and inducible, non-suppressible VT on electrophysiological testing.[12] The MADIT II trial found a reduction in mortality in patients with ischaemic left ventricular dysfunction and an ejection fraction of less than 30%.[13] This trial did not require demonstration of non-sustained VT or electrophysiological testing as entry criteria and was more of a heart failure or post-MI trial than an arrhythmia trial. The results indicate that a much larger patient-population may benefit from implantable defibrillators and, if this is borne out by further trials awaiting publication, it will have important implications for health resources.

Sinus tachycardia is the commonest cause of a sustained tachycardia. Sudden onset and offset are suggestive of pathological tachycardias. Symptoms may range from minor palpitations to syncope or severe dyspnoea. There may be an associated diuresis; a complaint of lethargy for up to 48 hours after the episode is a useful discriminatory symptom indicating an arrhythmia. Beating out the rate and regularity of the rhythm by the patient may indicate irregularity in atrial fibrillation or sensation of a normal heart beat. Initial investigation includes the 12-lead ECG. Ventricular tachycardia should be considered as a more rare cause of sustained tachycardia in any patient with evidence of ischaemic or structural heart disease. An ECG will also demonstrate evidence of pre-excitation with a slurred delta-wave and shortened P-R interval in patients with an accessory conduction pathway and Wolf Parkinson White (WPW) syndrome. A 24-hour ambulatory ECG recording is an appropriate next step, but often multiple recordings fail to capture any arrhythmia and event recorders are frequently required. In a significant proportion of patients, no arrhythmia will be demonstrated and those with a clear history can be admitted for electrophysiological testing and ablation.

Supraventricular tachycardias (SVT) and AF, or atrial flutter, are the commonest causes of sustained tachycardias. Sustained VT should be referred to cardiologist for in-patient assessment. The potential mechanisms for SVT include nodal re-entrant tachycardias, atrial flutter, focal atrial tachycardia and accessory pathways. The prognosis of SVT is usually but not always benign. The important exception is the

WPW patient presenting in AF who is at risk of fast accessory pathway conduction, facilitating degeneration to ventricular fibrillation (Figure 3).

Simple reassurance that the symptoms, although bothersome, are not life-threatening is a treatment option in some patients and avoids the side-effects of pharmacological agents; amiodarone and flecainide have serious and occasionally life-threatening side-effects and their use should be supervised by a cardiologist. Beta-blockers and verapamil, are less effective suppressive agents and also produce troublesome side-effects including lethargy and constipation, which are poorly tolerated in a young and fit population. In highly symptomatic patients, radiofrequency catheter ablation offers an effective cure for SVT in 95% of cases of WPW in which this procedure is particularly indicated, and may be life-saving in the WPW patient with AF when combined with a fast-conducting accessory pathway (Figure 3). The risks of the procedure are low; the main complication is damage to the atrioventricular node, as this is located close to some re-entrant pathways, causing heart block in 1% of cases and requiring implantation of a permanent pacemaker.

The patient with chest pain

Dr Ian Findlay, Consultant Cardiologist, Royal Alexandra Hospital, Paisley

Angina is primarily diagnosed from the patient's history and his description of chest pain. Typical angina consists of chest discomfort or tightness of brief duration, coming on with exercise and being rapidly relieved by rest or GTN spray. Radiation to the arm and jaw are further typical features (Table I).[14]

Angiographic studies have indicated that the significant obstructive coronary artery disease can be predicted with some confidence from a typical history in men or older women.[15, 16] Middle-aged women present a more difficult diagnostic challenge largely because of their lower disease prevalence. Patients classified with non-anginal pain have a very low risk of significant coronary disease, and patients with atypical pain are at an intermediate risk. The presence of risk factors such as diabetes mellitus, hypertension, hyperlipidaemia and a positive family history are useful additional indicators of the likelihood of significant coronary artery disease, and various predictive algorithms are available to combine patient variables to produce a combined risk score. For example, a woman under the age of 55, with a typical history for angina but no risk factors, has a risk of coronary artery disease of less than 10%. A similar woman with diabetes, hyperlipidaemia and a smoking history has a risk of 40%.

Exercise stress testing is an established component of the evaluation of patients at chest pain clinics. However, its sensitivity in detecting patients who were subsequently demonstrated to have significant coronary angiography is poor, being positive in 58–71% cases. Similarly, the specificity of the test is weak, being negative in only 73–82% of patients with no angiographic disease. The main value of stress testing comes from the prognostic information that it can provide. Workers at Duke University have devised a treadmill score that incorporates time or distance managed on the treadmill,

FIGURE 3

An ECG demonstrating atrial fibrillation in a patient with an accessory pathway, pre-excitation and the Wolf Parkinson White syndrome. The rhythm is fast and irregularly irregular with broad pre-excited complexes. Patients who develop this rhythm and have a fast-conducting accessory pathway are at risk of progression to ventricular fibrillation.

TABLE 1
Criteria to classify chest discomfort.
A simple classification of chest pain modified from Patterson and Horowitz[14] that may be used in conjunction with an assessment of cardiac risk factors to determine a patient's likelihood of having significant coronary artery disease and the need for further investigation.

Criteria
1. Precipitated by exercise
2. Brief duration
3. Relieved promptly by rest or nitroglycerin
4. Substernal location
5. Radiation to jaw, left arm or neck.
6. Absence of other causes for pain

Classification
I. Typical angina
Criteria 1 to 3 all positive
Any four criteria positive

II. Atypical chest pain
Any two criteria positive
Only criteria 4 to 6 positive

III. Nonanginal chest pain
Only one criterion positive

the presence and magnitude of ST segment shift, the provocation of anginal symptoms by this first and whether these were limiting. The 'Duke treadmill score' has been repeatedly validated and can identify a high-risk subset of patients with an annual mortality of 5%.[17]

Coronary angiography is indicated in high-risk patients to further characterise those who may benefit from surgical revascularisation. Coronary artery bypass grafting improves mortality in patients with left main stem disease, two-vessel disease with left anterior descending artery involvement and three vessel disease, particularly if there is associated left ventricular impairment or exercise testing is strongly positive. Coronary angiography is also indicated where exercise testing is inconclusive or cannot be performed owing to mobility problems or resting ECG abnormalities, including left bundle branch block. It is important to establish a firm diagnosis of coronary artery disease before the patient embarks on a lifetime of multiple preventive therapies.

The National Service Framework recommends rapid-access chest pain clinics for the prompt assessment of patients presenting with angina.[2] Moreover, consultations in GPs' surgeries should be 'structured and guided by the active use of paper or electronic practice protocol/guideline which includes the indications and arrangements for accessing specialist advice and exercise testing'. The Royal Alexandra Hospital in Paisley has implemented a web-based referral system that facilitates protocol-driven access to exercise testing and consultation with a cardiologist.[18] This includes online entry of treadmill results and calculates the patient's risk of coronary artery disease from the GP's data and exercise results; the majority of patients referred are found to be at low risk from coronary artery disease partly because of website screening questions that suggest immediate hospital referral for patients with unstable angina or suspected MI. Other rapid access chest pain services have a much higher percentage of acute patients. Given the resources that have been directed towards the development of rapid-access chest pain clinics, there has been increased debate surrounding evidence for their effectiveness with a recent Cochrane review indicating little proof of benefit and a requirement for larger studies to address this question.[19] Rapid access chest pain clinics probably can be effective in reducing admissions to hospital with chest pain but more evidence is needed with improved dialogue between GPs and hospitals required, in selecting which patients benefit the most from referral.

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