CARDIOVASCULAR DISEASE IN THE ELDERLY

C.R. Langridge, LAT in Geriatric Medicine, Royal Victoria Hospital, Edinburgh

INTRODUCTION

With the increase in the elderly population and the improvement in the health of the very elderly it is increasingly important that all specialities are aware of the needs of these patients and the treatment options available. It is vital that individual medical specialists view the elderly patient holistically, and also that the geriatrician is updated about the latest therapeutic options available for various conditions.

On Friday 18 May 2001 a joint symposium on geriatric medicine and cardiology, entitled *Cardiovascular Disease in the Elderly*, took place at the Royal College of Physicians in Edinburgh. It provided a forum for a panel of renowned speakers to present the recent evidence base in managing the most common cardiovascular problems in the elderly. In the afternoon session an interactive debate on 'Not for attempted resuscitation' provoked, as might be expected, lively discussion and commentary. The report endeavours to summarise the salient points made from each of the lectures.

SESSION 1

DIAGNOSIS OF HYPERTENSION IN OLDER PATIENTS

Professor John Potter, Professor of Medicine for the Elderly, Glenfield Hospital, Leicester

The majority of deaths in the 75–84 year age group are from coronary artery disease and stroke,[1] with hypertension being the biggest treatable risk factor. Results from various studies in the past have shown a linear relationship between increasing systolic and diastolic blood pressure and risk of stroke and coronary artery disease.[2, 3] More recently, the Framingham data has been re-analysed and it has been found that there may, alternatively, be a threshold effect with a sudden take off in risk at a systolic blood pressure of 160 mmHg in men and 180 mmHg in women.[4]

Recent attention has been turned to examining the effects of pulse pressure and mean arterial pressure.[5] In the elderly, pulse pressure is the strongest predictor of coronary heart disease and should be measured; unfortunately there are as yet no drugs available to modify it specifically.

Accurate recording of blood pressure (BP) readings is obviously necessary to aid treatment. There are several specific problems that are encountered in the elderly (Table 1). Pseudohypertension affects one to two per cent of the population and is probably overdiagnosed in clinical practice. In patients with a large variability of BP readings between clinic visits it is recommended that ambulatory BP monitoring be used. This type of continuous monitoring is useful in other circumstances also (Table 2). The British Hypertensive Society Guidelines[6] for ambulatory BP monitoring have given arbitrary normal daytime BP readings as 135/85 and night-time as 120/70 as targets on treatment.

Hypertension caused by renal artery stenosis should be suspected in several circumstances (Table 3). In the elderly this should be investigated using, for example, magnetic resonance imaging (MRI) angiography or carotid duplex ultrasonography. Professor Potter suggested the aim would be to improve BP control as it is difficult to cure

TABLE 1
Specific problems with measuring BP in the elderly.

• Wrong size cuff
• Ausculatory gap
• Rigid arteries and pseudohypertension
• Rapid cuff inflation
• Large BP variability between visits

TABLE 2
Ambulatory BP monitoring.

• Suspected white coat hypertension
• Drug resistant hypertension
• Orthostatic/post-prandial/drug-induced hypotension
• Borderline hypertension without other risk factors

TABLE 3
Clues to the presence of renal artery stenosis.

• Sudden onset and rapid deterioration in control of BP
• Multiple drug resistant hypertension
• Deterioration in renal function with ACE1 inhibitor
• Malignant hypertension
• Abdominal bruits
• Flash pulmonary oedema

with angioplasty or stents and there is little place for surgery.

There is some evidence of benefit of treatment with antihypertensives up to age 82.[7] A meta-analysis[8] showed a decrease in all major cardiovascular events in >80 year olds with treatment but, paradoxically, the total mortality was actually increased with treatment. Therefore, there is no firm evidence of when and what level of BP to treat in this age group. The HYVET[9] study should provide valuable evidence.

Professor Potter's own recommendations for the >80 year olds are:

- with target organ damage +/- other risk factors – for treatment (but no evidence);
- without target organ damage +/- other risk factors – probably don't treat;
- on treatment already with target organ damage/risk factors – continue treatment; and
- on treatment without target organ damage/other risk factors – unsure as to whether treatment should proceed.

DIAGNOSIS OF HEART FAILURE IN ELDERLY PATIENTS – IS IT HEART FAILURE?

Professor Henry Dargie, Consultant Cardiologist, Western Infirmary, Glasgow

The average age of a patient presenting with heart failure is 75 and, although many patients present with unmistakable signs and symptoms, a proportion do have more non-specific complaints. This lecture helped to clarify which investigations are vital for the diagnosis of heart failure and discussed newer strategies to identify heart failure more promptly to enable earlier treatment and improve prognosis.

In 1994 the European Society of Cardiology Task Force[10] produced a guideline which stated that most heart failure was diagnosed on appropriate symptoms and evidence of cardiac dysfunction. Their algorithm, included in the guideline, has since been updated.[11]

The Scottish Intercollegiate Guidelines Network[12] guideline is also useful and reviews two aspects of heart failure: firstly, the need to confirm the presence of cardiac dysfunction and secondly, the need to determine the aetiology of the underlying heart failure. If there is a clinical suspicion of heart failure an echocardiogram should be performed to demonstrate cardiac dysfunction, or radionuclide ventriculography if echocardiogram is not available. Routine blood tests should be performed including FBC, U & E, GLUCOSE, LFT, Bilirubin, Albumin, TFTS and cholesterol. A 12 lead electrocardiogram (ECG) may give a clue to the underlying aetiology. If the ECG is normal then left ventricular dysfunction would be unusual. A chest X-ray is of limited value but should be done to identify pulmonary pathology as a cause of shortness of breath.

Other investigations which can be performed include an ambulatory ECG, but only where an arrhythmia is suspected of causing symptoms; an exercise test (not of particular value in diagnosis, but can rather be used as an objective assessment of functional capacity with oxygen uptake as a measure of prognosis or for ischaemia detection); and radionuclide imaging, which can be used to detect ischaemia and is more accurate for left ventricle (LV) function than echocardiography. Coronary angiography is not normally performed to make a diagnosis of coronary artery disease or aortic valve problems except in those with angina. Finally, an endomyocardial biopsy can be used if no cause is found or where an infiltrative cause is suspected, e.g. amyloid or haemochromatosis.

Left ventricular dysfunction is probably the commonest type of cardiac failure. It is an abnormality of contraction and is most commonly due to coronary heart disease. The most widely used measure is left ventricular ejection fraction (LVEF), but it is not without problems. A study in north Glasgow[13] has shown a wide variation of LVEF measured using two different methods. Comparisons of various other methods, including LV angiogram, radionuclide angiogram, radionuclide ventriculogram and MRI have also shown considerable disparity.

Therefore, identification of other possible predictors of the presence of left ventricular systolic dysfunction (LVSD) would be of benefit. The predictor discussed was natriuretic peptide. Atrial natriuretic peptide (C-ANP and N-ANP) and ventricular peptide (BNP) are produced in response to stretch as a result of the left ventricular end diastolic pressure (LVEDP) and left atrium (LA) pressure in left ventricular dysfunction (LVD). Studies have shown that they have a very high negative predictive value which enables their use in 'ruling out' heart failure and therefore may be useful as a means to exclude heart failure and decrease the need for referral for echocardiography.[14, 15]

The term preserved systolic function (or 'diastolic dysfunction') is used for patients with 'clinical heart failure' who do not seem to have a reduced ejection fraction. It is a concept that is not entirely understood and it may be the case that these patients actually have a relative, and functionally important, reduction in ejection fraction, although the actual ejection fraction is within the 'normal range'.[16, 17]

Professor Dargie suggested that screening of heart failure should be targeted at high-risk subgroups as, although asymptomatic, they do benefit from treatment, e.g. the elderly, those with previous myocardial infarction (MI), evidence

of ischaemic heart disease, hypertensives and diabetics.

DIAGNOSING DIZZINESS – WHAT IS CARDIOVASCULAR AND WHAT ISN'T

Dr Andrew Davies, Consultant Geriatrician, Newcastle General Hospital

Dizziness means different things to different people – from light-headedness to an epileptic aura to panic attacks.

Patients can present to a variety of specialists due to the different pathophysiological systems involved. Certainly, in a variety of trials the cause of dizziness found in the clinic or community setting may be somewhat dependent on the type of specialist clinic the patient was referred to.[18–20] Tinetti recently suggested that dizziness should be thought of as a possible 'geriatric syndrome'.[21]

Even a detailed accurate history in the elderly may not be very revealing: cognitive impairment, amnesia of syncope, postictal confusion in the patient or the lack of an available account from a witness can all be factors.

Dizziness as a symptom has a significant influence on the quality of people's lives; patients with dizziness are more prone to depression and anxiety than controls matched for age and sex,[20] and dizziness has been described as being one of the most important single symptoms with a negative influence on well-being in old age.[22]

Although the causes of dizziness are often multiple and cannot be put into neat categories, they can be divided up into various groups on the basis of the presenting symptoms.

Vestibular dizziness

Patients describe symptoms of vertigo which is rotational, often on movement.

It is important to examine the ear (looking specifically for a fistula), and perform Rinnes and Weber's tests along with more specific tests, e.g. Hallpike's manoeuvre and Unterberger's test.

The causes include:

- benign positional paroxysmal vertigo (BPPV) – the dizziness is short-lived (seconds) and classically occurs when lying down in bed and turning in one direction; it is also associated with head movement, e.g. looking up; it is diagnosed with the Hallpike's manoeuvre and is treated using the Epley technique for particle repositioning;
- viral labyrinthitis – this initially presents with vertigo of several days' duration; if uncompensated, it continues with intermittent vertigo;
- Ménière's disease – there is intermittent vertigo with associated tinnitus and deafness which is progressive; if intractable, saccus decompression, vestibular nerve section or ablative surgery can be tried;
- perilymph fistula; and
- acoustic neuroma.

Vestibular sedatives are used to treat but should only be used during the acute event as they may delay recovery if used long-term.

Neurological dizziness

Patients complain of 'unsteadiness' and 'feeling drunk'. It is necessary to examine specifically for nystagmus, pastpointing, dysdiadochokinesis, loss of smooth visual pursuit, poor static balance, broad based gait and staggering.

The causes include:

- cerebrovascular disease;
- cerebellar disorders;
- vertebro-basilar migraine; and
- subclavian steal syndrome.

The cause remains unknown in 25% of the elderly.

Treatment often proves difficult and may involve specific exercises to improve balance.[23]

Cardiovascular dizziness

This can be thought of as being caused by 'blood pressure' and 'heart problems'.

1. *Blood pressure*

Patients can describe 'feeling like I'm draining away' and a 'blackness of vision'. The symptoms are relieved by sitting or lying down and are brought on by a rapid change in posture or prolonged standing. There is often associated loss of consciousness with witnessed pallor or sweating.

The causes include:

- orthostatic hypotension – best assessed in the morning; get the patient to rest supine for 15 minutes and then stand as quickly as possible; record the patient's BP every 30 seconds for three minutes; a sustained drop of 20 mmHg that is reproducible is significant;
- vasovagal syncope – specific symptoms to ask about include a slow prodrome of tiredness with gradually increasing dizziness, and afterwards nausea with or without vomiting and feeling presyncopal for several hours; it can be sought with head-up tilt testing for 40 minutes at 70 degrees (can also use isoprenaline/GTN), simultaneously measuring pulse and BP; the test is positive if symptoms occur in association with a drop in BP, bradycardia or asystole; and
- carotid sinus hypersensitivity – specific symptoms

include sudden falls or loss of consciousness or transient dizziness; the symptoms are often short-lived (<20 seconds); to investigate, it is necessary to perform longitudinal massage over the carotid sinus for five seconds, performed with the patient supine and at 70 degrees; there needs to be simultaneous beat to beat BP recording and ECG monitoring; a positive result is >3 seconds asystole or a >50 mmHg drop in BP, or there can be both.

General treatment should include withdrawal of all culprit medication, with simple advice given to increase fluid intake, increase salt intake, take care on rising and wear support hosiery. More specific medication includes fludrocortisone, desmopressin nasal spray. Insertion of a pacemaker can be used in certain circumstances for carotid sinus hypersensitivity. These have been shown to reduce the incidence of falls and loss of consciousness but not the feeling of dizziness.

2. *Heart problems*

Patients complain of palpitations with associated loss of consciousness. These symptoms can be exercise-induced and produce sudden and unexplained falls. Other symptoms include chest pain-induced dizziness and witnessed pallor or sweating.

The causes include:

- tachyarrhythmia/bradyarrhythmia – the investigation starts with a 12 lead ECG; if this is normal there is a low risk of significant cardiac arrhythmia or sudden cardiac death; an ambulatory ECG can be useful, but it is worth remembering that 24% of elderly people have an asymptomatic bradycardia, and often symptoms do not correlate with arrhythmias on the tape; other patient activated recorders, including the implantable loop recorder, can provide valuable information;
- cardioinhibitory carotid sinus hypersensitivity; and
- mechanical, e.g. outflow tract obstruction, mitral stenosis or atrial myxoma; investigations can include echocardiography with or without transoesophageal echocardiogram (TOE) and angiography.

Treatment will be determined by the precise aetiology of the underlying cause.

SESSION 2

DRUG TREATMENT OF HEART FAILURE – IMPROVING SYMPTOMS OR EXTENDING LIFE?

Professor John Cleland, Professor of Cardiology, University of Hull

In Scotland, despite advances in available treatment, there has been a steady increase in hospital discharges with a diagnosis of heart failure.[24] The prognosis is poor, with 35–45% of patients dead within six months, and those surviving over six months having a seven to ten per cent annual mortality.[25]

Recommendations regarding best treatments are made difficult as few trials[26, 27] specifically included patients over 70 years of age – subgroup analysis from larger trials is therefore necessary. Professor Cleland reviewed the literature and available randomised control trials (RCTs) for various treatment options for congestive heart failure, the results of which are summarised below.

Diuretics

In comparing thiazide and loop diuretics it is found that thiazides, although more likely to cause hypokalaemia, do cause less urgency of micturition, but they are less effective than loop diuretics, particularly if the glomerular filtration rate (GFR) is low (as it can be in the elderly). An alternative is torasemide (a long acting loop diuretic).

Digoxin

Its benefit in heart failure in the elderly is doubtful. In the large DIG study the mean age was 63 years with no published subgroup data. However, in one group of elderly patients with clinical heart failure but with preserved systolic function and in sinus rhythm there was a decrease in hospital readmission whilst on digoxin.[28] Two other studies[29, 30] in elderly patients showed no evidence of benefit if the patient is in sinus rhythm, but it is probably useful if they are in atrial fibrillation (AF).

ACE inhibitors

Trial based evidence regarding symptomatic benefit is surprisingly limited. A small study[31] (with a mean age of 75) compared captopril with placebo, and this did show some improvement in a six minute walk and a decrease in New York Heart Association (NYHA) classification.

Evidence regarding the effects of ACE inhibitors on prognosis is more extensive. The SOLVD trial[32] (exclusively looking at LVSD) had an age cut off of 80 years and followed up patients over three years. There was a 24% reduction in mortality and a 29% reduction in recurrent hospitalisation at one year. The CONSENSUS trial[26] showed that using an ACE inhibitor could improve prognosis in patients with severe heart failure and LVSD. The ATLAS trial[33] (which included patients over the age of 90) investigated the possible value of higher dose ACE inhibitor treatment (35 mg lisinopril vs 5 mg lisinopril). No difference in total mortality was found, but the specific endpoints of death from, or hospitalisation for, heart failure decreased amongst patients on the higher dose of ACE inhibitor. Concern does obviously exist regarding the use of such very high doses in frail elderly patients with multiple comorbid factors in everyday clinical practice.

AII antagonists

The ELITE trial[27] (losartan vs captopril) showed little difference between ACE I and AII antagonists in >70 year

olds (compared with younger patients, when captopril is better). However, it is well tolerated and is a reasonable second choice. VAL–HEF[34] (valsartan versus placebo in addition to ACE I inhibitors) showed no improvement in mortality but did improve symptoms (NYHA–I class improvement and 27% decrease in recurrent hospitalisation).

Beta blockers

There are probably more studies in patients up to the age of 80 on beta blockers than ACE inhibitors. There is probably synergy between beta blockers and ACE inhibitors. The CAFÉ[35] study suggested possible long-term symptomatic benefit in selected patients. The COPERNICUS[36] study found there to be a decrease in mortality in association with ACE inhibitors, while MERT-HF[37] found a reduction in mortality in patients aged >70 years. Optimal dose in the elderly remains uncertain. Throughout the trials there is a consistent finding that beta blockers are better tolerated than placebo. This is particularly interesting given the concern about side-effects expressed by doctors themselves, especially when administering these drugs to elderly patients.

Aspirin

The SOLVD[32] trial showed a highly significant adverse interaction with ACE and aspirin with an increase in gastrointestinal (GI) bleeds in patients with heart failure.

Spironolactone

The RALES study[38] compared spironolactone with placebo in addition to ACE I and demonstrated benefit in the elderly.

Implantable defibrillators

In the Mayo Clinic 20% of patients who receive implantable defibrillators are over the age of 80. It is noteworthy that they improve 'quantity' of life only.

Heart failure services

Heart failure services may be beneficial as a means of monitoring and coordinating the care of heart failure patients in the community.

SESSION 3

INTERACTIVE DEBATE – RESUSCITATION

During the last year the issue of resusitation, or rather 'Do Not Resuscitate' (DNR), has been highlighted by the media and is firmly in the public eye. This has lead healthcare professionals to have to clarify their own personal views, the views of their health teams and that of the hospitals they work in. Guidance has been given by the British Medical Association and nursing profesionals.[39] The two case histories in this interactive debate were organised to encompass many of the problems surrounding the DNR order and enabled the two invited guests, Professor Shah Ebrahim, Professor of Epidemiology and Ageing, University of Bristol, and Professor Marion McMurdo, Professor of Ageing and Health Medicine, University of Dundee to give their views along with the audience.

As expected there was disagreement among the various responses from both the audience and the invited discussers. Several points were made.

- When faced with an incompetent patient (i.e. one who is unable, in law, to decide treatment for him- or herself) it is important to gain as much information surrounding the patient as possible by telephoning relatives, discussing the case with the GP, etc. However, although it would be best to have all family members in agreement with the decision of health professionals and medical staff, legally it is the doctor's decision as to whether he or she feels that it is appropriate and in the patient's best interests for that patient to be resuscitated. This may have to be justified if any such decision is in opposition to family wishes.
- Resuscitation decisions about a competent patient should take place with the patient and not the relatives.
- There is nothing in the guidelines to say that routine discussion about resusitation must take place with each patient.
- Although making a resuscitation decision when a patient is first admitted seems to be becoming of increasing priority, it is much more important for decisions of how the patient is to be managed to be discussed and decided, with the knowledge that these decisions need to be reviewed regularly and can be changed as the patient's clinical state alters.
- Professor McMurdo believed that hospitals and wards should provide written information about resuscitation to all patients and their relatives on admission to hospital. This leaflet would give information on what resuscitation means, the outcomes for resuscitation and the policy for that unit and encourage family and patients to discuss this with the medical and nursing staff, including any advance directives that may have been written.
- There needs to be research in the UK on what is currently happening, an improvement in communication skills for all healthcare workers and advance directives may be of some help.

SESSION 4

AORTIC STENOSIS IN THE ELDERLY – THE QUIET EPIDEMIC

Dr Colin Forfar, Consultant Cardiologist, the John Radcliffe Hospital, Oxford

Clinicians are often confronted by elderly patients with both symptomatic and asymptomatic aortic stenosis (AS). Dr Forfar outlined which patients should be referred – and when – for aortic valve surgery.

It can be estimated that, in the UK, approximately 4,000 patients over the age of 80 have AS of sufficient severity to warrant surgery (extrapolating results from Finland).[40] However, only 5,000 aortic valve replacements (AVRs) in total are undertaken on patients (of all ages) every year in the UK. There is therefore a large discrepancy between those with significant AS and those actually being operated on.

The assessment of a patient with AS should include a full history, examination and echo-doppler evaluation. Severity is best assessed by measurement of aortic valve area rather than by peak velocity measurements which measure flow per beat and are dependent on stroke volume and systemic vascular resistance (SVR).

Severity of AS can be simply classified as follows:

- mild: aortic valve area >1·5 cm^2;
- moderate: aortic valve area 0·8–1·5 cm^2; and
- severe: aortic valve area <0·8cm^2.

Management strategy

Symptomatic patients over 80 years of age

There are mainly retrospective studies with few prospective and no RCTs of AVRs. In summary, retrospective trials (in the 1970s and 80s) following AVRs for isolated calcific disease demonstrate similar mortality over five years compared with unselected 80 year olds without disease.

If there is coexisting ischaemic heart disease and both coronary artery bypass graft (CABG) and AVR were performed, after an initial increase in mortality post-operatively there is similar long-term outcome as for only AVR. The prognosis for unoperated severe AS is very poor. There are similarly poor results for balloon valvuloplasty, which is now virtually abandoned worldwide.

Looking at results from Oxford[41] retrospectively over nine years showed a 30 day mortality high at 19% with longer intensive therapy unit (ITU) and in-patient stays, but the mean survival was equivalent to age matched controls with 92% of patients at NYHA I or II. Another review[42] compared patients at low/intermediate/high risk based on their age/LV function/NYHA and compared medical and surgical treatments. This demonstrated that 'sicker' patients were at higher risk but gained greater survival advantage with AVR compared to medical treatment alone.

The preferred AVR implant in patients over the age of 70 is a biological rather than mechanical valve, due to heightened risks of warfarin and less concern with valve durability in the over 70s.

Asymptomatic patients with moderate to severe aortic stenosis

This is a difficult issue with no good longitudinal data.[43–5] Dr Forfar suggested that high risk patients with severe asymptomatic AS can be identified by regular echo follow-up looking at the degree of aortic valve calcification (calcification is a good predictor) and those with yearly progression of aortic jet velocity of >0·3 mls. The monitoring of symptoms is obviously crucial as symptoms identify the critical point in the natural history of AS with a poor prognosis.

Finally, should an AVR be performed at the time of CABG when the primary symptoms are due to angina? This would of course avoid the need for a further surgery but it does add unnecessary risk.

Results show that, for patients with mild AS with coronary artery disease, by doing a combined operation at the onset the excess mortality does significantly increase over a ten year period.[46] There are similar data with excess mortality for doing the combined operation at the start for moderate AS, even allowing for the fact that over half the patients will go on to develop severe AS.

Dr Forfar felt it was difficult to justify combined AVR and CABG in patients with mild/moderate AS, but important not to miss the patient with severe AS which did not feature in his analysis.

PACEMAKERS IN THE ELDERLY – WHO NEEDS THEM AND WHO GETS THEM?

Dr Rod Bexton, Consultant Cardiologist, Freeman Hospital, Newcastle upon Tyne

There has been a significant increase in the number of new pacemakers inserted in patients in Great Britain over the past 20 years; in Scotland it has been estimated that there were approximately 25 implants per million population in 1980 rising to approximately 280 implants per million in 1998.

Permanent pacemakers are inserted for various reasons and Dr Bexton provided clear guidelines and indications for permanent pacing (Table 4).

Of particular interest to the geriatrician is the group of patients with falls and cardioinhibitory carotid sinus hypersensitivity (CICSH) and whether these patients benefit from a permanent pacemaker (PPM). There is currently a multicentre trial underway which will help answer this question.[47]

In a typical pacemaker population 42% will have an atrioventricular (AV) block, 25% sinus node disease (SND), ten per cent SND and AV block, 13% AF and AV block, and ten per cent a combination of carotid sinus

TABLE 4
Guidelines and indications for permanent cardiac pacing.

Absolute indications
• Complete AV block (broad/narrow/any rate)
• Second degree Type II (Mobitz) AV block
• 2:1 AV block
• Second degree Type I (Wenkebach) AV block during waking hours
• Sinoatrial disease with pauses associated with symptoms
Relative indications (much greyer area)
• Patients with syncope and documented CICSH
• Symptomatic patients with sinus pauses of three seconds, unrelated to symptoms
• Symptomatic patients with lesser degrees of block, e.g. trifascicular block, bifascicular block where periods of asystole have not been documented
• Patients with falls and CICSH

sensitivity (CSS) and malignant vasovagal syncope (MVVS).

In simple terms there are two types of pacemaker, an 'ordinary' demand pacemaker with leads to either the atrium or the ventricle, or a 'physiological' pacemaker with either a single chamber and atrial (A) or ventricular (V) leads with rate responsiveness (R), or a dual chamber with atrial and ventricular leads +/- rate responsiveness. The three position pacemaker code is used in clinical practice to depict which heart chambers are being paced, sensed and the mode of response. Table 5 helps explain the nomenclature. In reality, the only ones made and used are VVI, AAI and DDD (also VDD).

There are general principles for the choice of pacemaker implanted. Firstly, the ventricle should be paced if there is actual or 'threatened' complete heart block. Secondly, the atrium should be paced/sensed unless contraindicated. A rate responsiveness pacemaker is not essential if the patient is very inactive or has a normal chronotrophic response.

The British Pacing and Elecrophysiological Group (BPEG) devised guidelines in 1992 as an aid to providing the correct pacemaker system for a variety of conditions which are listed down the left hand side of Table 6. However, Dr Bexton felt that all patients with SND should have a DDD (i.e. both heart chambers are paced and sensed) rather than the recommended AAI pacemaker where only the atrium is paced and sensed.

Although these are the recommended optimal pacing modes, Dr Bexton then reviewed what is actually being implanted. For example, in complete heart block back in 1980, 100% of patients had a VVI (i.e. only the ventricle is paced and sensed) inserted, in 1998 40% had a DDD inserted, which is obviously still suboptimal. Other heart blocks showed similar results.

So, despite BPEG recommendations, many patients are not getting the optimal pacing system inserted.

The age of first implant has been rising over the past 20 years and the relative need for inserting a pacemaker rises exponentially after the age of 60 due to the increase in the number of patients with SSS and AVB, with more men requiring PPM than women. However, it is noticeable that elderly patients are much more likely to have a VVI inserted rather than a DDD which they should have due to the higher incidence of sick sinus syndrome (SSS) and AV block.

Dr Bexton believes that not enough pacemakers are being inserted and that 'ageism' is still occurring in the choice of pacemaker for implantation.

THE OLDER PATIENT WITH ANGINA – WHEN IS MEDICAL TREATMENT NOT ENOUGH?

Dr Peter De Jaegere, University Medical Centre, Utrecht

Dr De Jaegere felt that it was important to maximise elderly patients' medication in order to improve symptom control of their angina, but if symptoms persisted and there were side-effects of pharmacological therapy then he would then consider PTCA or surgical treatment taking into account comorbidity factors. He then reviewed the literature surrounding PTCA and CABG describing the risks involved with these procedures in the elderly patient.

TABLE 5
Nomenclature used in the three position pacemaker code.

Position	I	II	III
Category	Chamber(s) paced	Chamber(s) sensed	Mode of response
Letters	V – Ventricle	V – Ventricle	T – Triggered
	A – Atrium	A – Atrium	I – Inhibited
	D – Double	D – Double	D – Double*
		O – None	O – None

**atrial triggered and ventricular inhibited*

TABLE 6
The British Pacing and Electrophysiological Group devised guidelines 1992.

Diagnosis	Optimal	Alternative	Inappropriate
SND	AAIR	AAI	VVI
AVB	DDD	DDD	AAI/DDI
SND+AVB	DDDR	DDD	AAI/VVI
AF+AVB	VVIR	VVI	AAI/DDD
CSS	DDI	DDD	VVI/AAI
MVVS	DDI	DDD	VVI/AAI

PTCA

The older the patients the more likely they are to die, suffer a stroke, have a Q wave myocardial infarction or develop renal and vascular impairment after PTCA compared to the younger patient.[48] The major predictive factor for these problems is the severity of coronary artery disease (the number of lesions >70%). Noteworthy is that age itself is less independently predictive.[48, 49] The completeness of revascularisation was the only independent predictor of event free survival.[49] With increasing experience of performing PTCA, the procedural outcome has improved with less devastating complications.[49]

With regard to symptom control, the majority of patients was asymptomatic or had mild angina after PTCA and there was significant reduction in the use of beta blockers and calcium antagonists, but the patients may have needed to have repeat attempts at revascularisation.[48]

Surgery

The number of elderly patients undergoing CABG is increasing. The age of the patient is an independent predictor for perioperative mortality. Other factors such as if the operation is performed in the acute situation and if it is a redo CABG affect the outcome adversely. However, the severity of the coronary artery disease is not such a big predictor, i.e. different to PTCA.

On an independent patient basis it is possible to assess the perioperative risk using the mathematical model devised by the Northern New England Cardiovascular Disease Study Group[51] and come up with a score (risk of stroke, etc.). Age itself is heavily weighted in this scoring system.

Stroke is a major risk perioperatively. A prospective study[52] looked at the incidence of Type I events (major focal deficits, stupor, coma) and Type II events (global, e.g. memory, intellectual). Both types of deficits occur in three per cent of the patients. Age is an independent predictor of a Type I event, but atherosclerosis of the ascending aorta and aortic arch is the most important predictor of perioperative stroke.

Again, in predicting a Type II event age is most important, followed by systolic BP and alcohol intake.

Cardiopulmonary bypass (CPB) has been held as most responsible for the above deficits. In Utrecht a trial is

currently underway to investigate the use of an octopus system (without the need for CPB compared with CPB). It has no results as yet from the elderly cohort but results from patients less than 65 years look encouraging.[53]

Coronary artery bypass graft can be used to palliate symptoms of angina, and although a higher mortality initially occurs in the elderly, once they have survived the hospital admission they have a reasonable outcome with mild angina.[54, 55]

CONCLUSION

This symposium provided all those who attended with an impressive wealth of information on the main aspects of cardiology in the elderly patient. The speakers were excellent and the content of their lectures outstanding. However, I felt it was noticeable that many of the speakers had to consistently refer to the 'lack of trial evidence in this elderly age group'. As the elderly population grows, it must be hoped that the number of trials that they are included in grows simultaneously so that future symposia can provide further evidence to improve clinical practice with the elderly patients that are being treated on a daily basis.

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