rates are 82% and 91% respectively.[7] Thallium images are not easy to interpret and the isotope is expensive (about £50 a patient); again, the technique has not caught on widely in Britain. Radionuclide angiography, on the other hand, is proving a useful method of assessing cardiac function. For this purpose the relatively inexpensive isotope technetium-99 (about £5 a patient) is generally used. It may be detected either as it first passes through the heart after an intravenous injection or, more commonly, it can be bound to red cells and allowed to equilibrate in the blood so that an electrocardiographic-gated image can be built up from 100 or more cardiac cycles with 20-50 frames a cycle. The resulting nuclear angiogram may look just like a conventional contrast angiogram but it has the great advantage of being repeatable. Thus global and regional abnormalities of left ventricular function can be detected both at rest and on exercise. An abnormal contraction pattern, or a normal one that becomes abnormal on exercise, may be the best basis for a screening test for suspected cardiac disease. In patients with proved cardiac lesions a nuclear angiogram assesses the functional importance of the lesion, detects change with the passage of time, and monitors the effects of treatment. For example, patients with coronary artery disease and angina show a deterioration in ventricular function on exercise that can be restored to normal with effective medical or surgical treatment.[8]

Nuclear techniques also have an established place in assessing pulmonary perfusion—for example, in suspected pulmonary embolism or congenital heart disease—and in detecting myocardial injury with infarct-avid agents such as technetium-99 pyrophosphate.[9] As a research procedure, positron-emission tomography is providing a measure of regional myocardial blood flow and metabolism; but the specialised equipment necessary for these studies, including a cyclotron, will limit its application. Nevertheless, it should be possible to correlate myocardial perfusion with coronary arterial stenoses in patients and thus to improve our understanding of this common problem. Nuclear magnetic resonance is yet another (totally different) imaging technique that is now being applied in neurology, producing images that can be superior to those of computed tomography.[10] Its role in cardiology is impossible to predict at this stage.

During the past few months the *BMJ* has published several reviews of the newer imaging techniques.[11–13] In the diagnosis of heart disease echocardiography and nuclear angiography are useful now. Both may confirm a clinical diagnosis and provide an estimate of left ventricular function, which is important because the state of the left ventricle is the best predictor of survival in adult heart disease: poor function equals poor prognosis.[14 15] The wider availability of these and other imaging techniques will be determined by their cost and diagnostic value. At present, given that echocardiography is relatively cheap and that nuclear medicine departments are widespread, a reasonable arrangement for the consumer-physician would be as follows. Every doctor should have access to chest radiography. Every district general hospital should have M-mode echocardiography and a physician capable of interpreting the results. Every cardiac centre with a regional responsibility should be able to undertake two-dimensional echocardiography and nuclear studies, certainly blood-pool imaging as a routine with thallium scintigraphy on occasion. Facilities for cardiac catheterisation should usually be confined to one or two such regional centres. Despite doubts about the future of cardiac catheterisation[1] there are strong reasons for believing that it will be with us for years to come,[3] particularly with the advent of interventional cardiac radiology.[16] In the next year or two digital subtraction techniques will become available, and in the more distant future who knows? Three-dimensional echocardiography? Coronary arteriography without catheterisation? One thing seems certain: physicians must continue to work closely with their colleagues in departments of diagnostic imaging so that patients can take advantage of whichever imaging technique gains ascendency in the next decade. Isolated cardiac units are doomed to provide a second-rate diagnostic service.

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Obstructive sleep apnoea syndrome

Obstructive sleep apnoea syndrome is a bizarre disorder first described relatively recently.[1] During sleep the upper airway becomes obstructed, sometimes for well over a minute, despite vigorous attempts by the respiratory muscles to draw in a breath.[2]

The obstruction is probably due to passive collapse of the pharyngeal walls. The muscular activity holding open the pharynx is reduced during sleep[3] and may be unable to overcome the subatmospheric pressures generated in the airway during inspiration.[4 5] Much less commonly laryngeal dysfunction is responsible, as proposed in the Shy-Drager syndrome.[6] Whatever the cause the consequences are hypoxaemia, sometimes incredibly severe, and hypercapnia;

these increase the drive to the respiratory muscles including the pharynx, which is finally pulled open with loud explosive snoring and snorting. At this point the sufferer may wake, partially or completely—a feature that may be important in terminating the apnoea. The blood gas tensions return to normal after a few noisy breaths. Next the obstruction gradually returns with even louder snoring until the apnoea recurs with complete silence again.[7]

This cycling of oxygen and carbon dioxide concentrations with recurrent loud snoring and arousal usually persists throughout the night, grossly disturbing both cardiorespiratory physiology and sleep. Thus excessive daytime sleepiness is a prime symptom. The worst disturbances usually occur during rapid-eye-movement sleep, a particularly vulnerable period.[7] The physiological changes during sleep have been well reviewed by Phillipson.[3]

Many conditions may contribute to the pharyngeal collapse at night. Simple obesity with the deposition of fat in the pharynx may cause critical narrowing,[2] as may other conditions such as acromegaly,[8] myxoedema,[9] enlarged tonsils,[10] Scheie syndrome (mucopolysaccharidosis),[11] and superior vena caval obstruction.[12] In some people no apparent abnormality can be found, and they are assumed to have inherently poor pharyngeal dilator activity during sleep.[7] In addition, partial nasal blockage or adenoidal enlargement can lead to even lower pressures in the pharynx during inspiration and provoke collapse when it would not occur otherwise.[13] Combinations of all these factors may tip the balance to produce obstructive sleep apnoea, as may excessive tiredness, sedatives, and alcohol.[7] Loud snoring without recurrent apnoea and symptoms is probably a "forme fruste" that may precede development of the full syndrome.[14]

The incidence of the syndrome is not known. The Stanford Sleep Institute has seen over 300 cases.[7] Though the incidence in Britain has been said to be lower than in the United States,[15] the difference probably reflects failure to recognise the condition.[16] From our experience in about 10 cases the diagnosis has been made only after referrals to many different departments.

Sir William Osler recognised the connection between obesity and hypersomnolence and commented that Joe, the fat boy in Dickens's *Pickwick Papers*, had this problem.[17] Osler would have approved of the fact that sleep apnoea syndrome can be diagnosed entirely on history and observation. The symptoms and signs were first fully described in obese subjects by Burwell *et al* in 1956,[18] who labelled it a Pickwickian syndrome, though they did not appreciate the cause. Eleven years later Gastaut *et al*[1] recognised that sleep apnoea was the primary cause. The patient rarely reports disturbed sleep but complains of overwhelming and often crippling daytime sleepiness while the spouse bemoans the patient's snoring. The consequences of the lack of sleep include apparent intellectual deterioration, personality changes, car accidents, and marital problems. Other symptoms noted less often include impotence, nocturnal enuresis, morning headaches, and abnormal motor activity during sleep to the point of sleep-walking.[2 7] Because of some similarities narcolepsy is often the initial diagnosis.

In the later stages of this condition other complications may appear, such as daytime hypercapnia and hypoxaemia with normal or near-normal lungs, polycythaemia, cor pulmonale, and hypertension. The usual modes of death are intractable heart failure or sudden death at night.[2 7 19]

Complicated and expensive recording equipment is not necessary to confirm the diagnosis; simple observation during a period of sleep will show the recurrent cycling of snoring and apnoea. In milder cases poor sleep in hospital may make it a little less obvious and longer periods of observation on more than one night may be necessary. A tape recording made at home of the snoring and apnoea may be diagnostic when the history is inadequate. Twenty-four-hour electrocardiographic monitoring may show sinus arrhythmia during sleep, cycling with the apnoea, as well as other more disturbing arrhythmias.[20] Ear-lobe oximetry is useful to measure the degree of consequent hypoxaemia and to show any response to treatment.

In obese patients simple loss of weight may be curative,[19] and this should always be attempted unless the condition is serious enough to warrant immediate active intervention. Excessive alcohol intake should be curbed and other reversible diseases treated. Drug treatment is worth trying; medroxyprogesterone acetate (for its respiratory-stimulant[21] or fat-redistributing properties[7]), and protriptyline[22] (which reduces rapid-eye-movement sleep and may alter respiratory muscle activation) have had some success. Strychnine has recently been tried because it prevents the reduction in postural muscle tone during rapid-eye-movement sleep, which is thought to be part of the problem.[23] Intubation of the oropharyngeal or nasopharyngeal airway and pharyngeal surgery have met with limited success.[24] Relief of nasal obstruction and tonsillectomy may be curative, particularly in children.[25 26] Tracheostomy is the most effective treatment,[7 27] but this operation has its own problems and can be socially disabling. Sedative premedication and general anaesthesia are hazardous in these patients and must be the responsibility of an expert anaesthetist.[10 27] With such a physical problem mechanical solutions seem most appropriate, and some success has been reported with continuous positive airway pressure through the nose.[28] After any successful treatment obese patients find that losing weight becomes easier and this may make continued treatment unnecessary.[29]

Obstructive sleep apnoea syndrome is easy to diagnose but harder to treat. The physician must be prepared to consider several lines of approach, tailoring his treatment—pharmacological, mechanical, or surgical—to the patient's disability, reserving tracheostomy for the more severe cases or when all else fails. The dramatic change in these patients after successful treatment is most rewarding.

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Deaths and anaesthesia

With all the recent talk about audit formal large-scale investigations of current practice remain rare. An investigation of deaths associated with anaesthesia in five NHS regions[1] commissioned jointly by the Association of Anaesthetists and the Nuffield Provincial Hospitals Trust is therefore welcome —despite its having provoked newspaper headlines[2] claiming that "anaesthesia could cost 900 lives each year."

What the survey showed was that 6 in 1000 of patients died within six days of operation but that only one in 10 000 died totally as a result of anaesthesia. To extrapolate from those deaths to the whole of Britain, around 280 deaths each year would be totally attributable to anaesthesia (though some of those would be in patients with other life-threatening disease). Anaesthesia would play some part in 1800 deaths. In both categories avoidable anaesthetic errors could be identified.

The picture that emerges from the report is depressing. Half the deaths occurred in the ward on the day of operation, which suggests that selection of patients for care in recovery rooms was poor. In nearly 10% of the deaths the anaesthetist had not made a preoperative assessment of the patient, which implies that each year in Britain 300 000 patients are anaesthetised without meeting their anaesthetists.

In contrast to the *Confidential Inquiries into Maternal Deaths in England and Wales* the report did not highlight and comment on the clinical problems most commonly associated with death. Nor did it look at anaesthetic disasters which may be just as serious: those in which a patient is left permanently handicapped from a prolonged period of anoxia. To some extent there are problems of scale—the sheer numbers of deaths made detailed analysis impossible.

Despite its lack of clinical detail, however, the report has drawn attention to the inadequate facilities and lack of monitoring equipment in some hospitals (20% have no recovery rooms), yet further evidence of the current underfunding of the NHS. It has also pointed to the failure by some anaesthetists to identify patients at risk long enough before operation for the necessary plans to be made. Trainee anaesthetists are too often left unsupported; medical assistants, clinical assistants, and general practitioner anaesthetists may either not feel at liberty to call for consultant help or have a false belief in their own competence.

In refuting some of the explanations advanced[2] for these findings, Professor J P Payne[3] pointed to some of the particular problems faced by anaesthesia: largely because of its rapid expansion, it is still a shortage specialty and it relies too much on junior doctors for routine services, with a lack of opportunity for proper instruction. Few would disagree, however, that anaesthetic deaths need to be investigated within the profession with the same vigour as maternal deaths have been studied for half a century. That means collection of reliable comprehensive data nationally and more effort locally. Indeed, audit should ideally identify specific failings and quickly bring them to the attention of the clinicians concerned. These aims are probably best achieved at regular "deaths and disasters" meetings in individual hospitals, with only those directly concerned attending. A balance can be drawn between the plain speaking that is necessary and any hint of a self-appointed kangaroo court; the outcome must be seen to be non-punitive. Public concern will be allayed only so long as professional efforts to make audit acceptable are seen to be effective. But that concern should also extend to correcting the underfunding of the National Health Service; until this is done the people of our country cannot be provided with the standards of care to which they are entitled.

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