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Cabbages and CABG

The risk of serious neurological damage from bypass operations is small

Coronary artery bypass grafts relieve angina and prolong some patients' lives with an operative mortality of 1-4%. The grafting requires temporary artificial maintenance of the blood's circulation and oxygenation with cardiopulmonary bypass. In the United States some 200 000 people yearly have a coronary artery bypass graft—six times as many in relation to the population as in Britain. The greatest risk of the operation is to the brain, and the size of the risk has been explored by the superb prospective study of Pamela Shaw and her colleagues from Newcastle.[1-3] Other prospective studies have been published,[4 5] and we now have a clearer picture of the risk of cerebral complications from cardiopulmonary bypass.

The two most serious complications of cardiopulmonary bypass are diffuse encephalopathy from global cerebral anoxia and focal cerebral infarction. Shaw *et al* reported that 3% of their series of 312 patients had such a prolonged depression of consciousness, whereas 12% of Breuer's 421 patients had an encephalopathy on the fourth day after the operation.[4] Focal stroke occurred in about 5% of patients in both series and was described as severe in 2%.

Patients may also suffer more subtle neuropsychological deficits after cardiopulmonary bypass, probably from patchy ischaemia in the cerebral arterial border zones. Four fifths of the patients in the Newcastle series showed deterioration in neuropsychological tests performed seven days after the operation compared with those performed before. Similarly, Smith *et al* found that two thirds of their patients showed neurophysiological impairment after seven days.[5] But only about one third of the patients in both series had symptoms, and only 9% of the Newcastle patients were disabled from neurophysiological deficit. The patients' performance deteriorated in tests of psychomotor speed, perceptual attention, concentration, short term memory, new learning ability, and visuospatial organisation—tests that are sensitive to organic cerebral injury. Further evidence that cerebral damage occurs during cardiopulmonary bypass comes from Aberg *et al*, who correlated neurophysiological deficits with increases in adenylate kinase in cerebrospinal fluid, which were present in 91% of patients.[6]

How much of this cognitive carnage arises from cardiopulmonary bypass and how much from other components of these large operations? Shaw *et al* found that a third of the 50 patients who had had large chest or vascular operations without cardiopulmonary bypass had neuropsychological deficit—but none had symptoms or were disabled. Smith's group, on the other hand, found that almost three fifths of the patients who had had large operations had deficits regardless of whether cardiopulmonary bypass had been used; abnormal neurological signs and severe deficits were, however, more common after bypass. These neuropsychological deficits do not seem to be so important in the long term: among the Newcastle cohort 57% had detectable deterioration after six months, but the impairment was mostly mild. A quarter had minor symptoms, 2% were disabled from neuropsychological deficit, and only one person was out of work because of intellectual deterioration attributable to the operation.

Focal infarction of the brain may occur with cardiopulmonary bypass from macroembolism of left ventricular thrombus, atheromatous debris shed from crunchy aortas on clamping, valve debris, or air. Multiple small areas of ischaemia might arise from microemboli of gas, fat, aggregates of red cells, platelets, or fibrin and particulate debris from silicone antifoam and plastic tubing. Global hypoperfusion might occur from low arterial pressure, reduced cerebral blood flow, and the non-pulsatile flow of most bypass systems. It is, however, difficult to find consistent risk factors for the cerebral complications of bypass. The Newcastle group found that the operations that used bypass were a little longer and associated with a greater drop in haemoglobin concentrations than major operations with bypass. There were no significant differences in haemodynamic factors. In the study of long term cerebral damage only poor left ventricular function before the operation was a risk factor. Age, low arterial pressures during bypass, long bypass times, and previous neurological disease had no effect. Smith *et al*, in contrast, found that bypass times had been prolonged in patients with early deficits. Neither they nor the Newcastle group found that carotid artery disease was a risk factor. Similarly, Breuer *et al* could find no clear risk pattern.

The importance of microembolism is shown by examining the ocular fundi. In Shaw's study a quarter of the patients who had been on bypass but none of the surgical control group had evidence of focal retinal ischaemia. Showering of the cerebral arterial border zones with other small emboli travelling up the carotid artery would be a plausible cause of some of the cognitive impairment. Atheromatous debris has been found scattered in the border zones of at least one patient who died of ischaemic brain damage after cardiopulmonary bypass. Arterial line filters have been suggested to reduce microembolism to the brain during bypass, but they add yet another foreign surface for the activation of platelets, comple-

ment (with generation of damaging free radicals by leucocytes), and the thrombotic cascade. At least one study has shown that small pore filters in the arterial line do not prevent the cerebral complications of bypass.[7] The importance of scrupulous control of the cerebral circulation just before the patient is put on to bypass has been shown by Nevin *et al.*[8] They found that avoiding hypocapnia just before bypass significantly reduced the incidence of persistent neuropsychological deficits (carbon dioxide is a potent cerebral vasodilator).

The main pharmacological candidates for protecting the brain from ischaemia are barbiturates, calcium entry blockers (nimodipine, flunarizine), free radical scavengers (vitamins E and C), and excitatory amino acid inhibitors. Recently excitement was generated when Nussmeier *et al* showed that thiopental given in a high enough dose to maintain cerebral electrical silence may protect patients undergoing cardiopulmonary bypass from persistent neuropsychiatric deficit.[9]

Stroke and disabling neuropsychological deficit do occur after coronary artery bypass grafting, but the long term risk to function is small. Cardiopulmonary bypass is responsible for most but not all the risk, but as many of these operations continue to be performed even this small risk is important. Reduction of the risk seems to depend on even more complex care over anaesthetic technology and technique, perhaps with the addition of positive protection to limit the damaging effect of any unpreventable cerebral ischaemia.

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Oxygen concentrators in the home

Wrongly prescribed without expert advice

Respiratory failure is a common cause of death in chronic obstructive airways disease. The fall of arterial oxygen tension becomes important at values below 8 kPa (60 mm Hg); many patients develop hypercapnia, but it is oedema (when the combination is called cor pulmonale) that marks the turning point in the prognosis.[1,2] If, however, the inspired air can be enriched with oxygen sufficiently to raise the arterial oxygen tension beyond 8 kPa survival is improved.[3,4]

The Medical Research Council study carefully selected bronchitic patients with a forced expiratory volume of less than 1·5 litres in one second and an arterial oxygen tension less than 8 kPa who had had oedema with or without hypercapnia.[3] They were then randomised into two groups. The first group was given oxygen through nasal cannulas at flow rates of about 2 l/minute for at least 15 hours a day, including during the night. The second group of patients were given merely the usual supportive treatment. After three years roughly half as many patients again in the first group had survived compared with those in the second. A second, American, study of patients given 12 hours of oxygen, largely at night, or 24 hours' treatment was stopped after just under two years because of the greatly enhanced survival in the latter group. Combining the results of the two studies showed clearly that oxygen had to be given for at least 15 hours a day to improve survival.

After these studies the health services of many Western countries were faced with implementing their implications. Several questions had to be asked about long term domiciliary oxygen treatment. How should it be given? How should patients be selected? And who was responsible for prescribing the oxygen, a consultant or a general practitioner?

The two national studies indicated that the oxygen concentrator, which separates oxygen from the ambient air, would be the cheapest and easiest means of supplying low flow oxygen for long periods during the day. Patients with bronchitis with an arterial oxygen tension of less than 7·3 kPa (55 mm Hg), hypercapnia, and oedema were the ideal candidates for treatment but there was less knowledge about its value in other patients—for example, those with respiratory failure from other causes such as fibrosis of the lung. Three categories were defined in the *Drug Tariff*: patients with obstructive airways disease, chronic respiratory failure, hypercapnia, and oedema (category 1); patients with obstructive airways disease and hypoxia but no oedema or hypercapnia (category 2); and patients with respiratory failure from other causes (category 3).[5] All patients must be in a stable phase of their disease with persistent respiratory failure for at least three to four weeks.

In other countries the use of oxygen varies widely, from roughly 10 000 per 50 million of the population in France to as high as 50 000 per 50 million people in the United States. Nevertheless, not all patients receive genuine long term oxygen treatment. In Britain we have no idea how many patients are likely to benefit, but in Sheffield a random survey of people over 45 showed that roughly three in 1000 had a forced expiratory volume of less than 1·5 litres in one second and an arterial oxygen tension of less than 7·3 kPa (55 mm Hg).[6] If such a proportion is general throughout England and Wales then as many as 60 000 oxygen concentrators might be needed.

As long term oxygen treatment is a specialised treatment it was recommended that consultant chest physicians should assess patients for suitability. Nevertheless, as treatment was based entirely in the home general practitioners had an important role and the decision was taken that they would prescribe the concentrator, though they were urged to seek a consultant's opinion before doing so.

The service started in 1985, and several studies have now examined the outcome. Several weeks ago in the *BMJ* Walshaw and his colleagues reviewed 61 patients prescribed