be hidden. A full disclosure of all important aspects of disability in older people is often best undertaken by a rehabilitation team. This is most usually available via the day hospital.

It has been increasingly recognised that assessment and treatment of older people in their own home has advantages over departmental based assessments. Real daily life problems are more apparent; there is greater opportunity to involve the family; and there is greater opportunity to involve home care and community nursing staff. Some districts now provide separate home rehabilitation teams to achieve this, while in other districts the therapy team is based in the day hospital but much of the assessment and treatment takes place in the patient's home.

COORDINATION OF REHABILITATION

Coordination of effort between the members of a multidisciplinary rehabilitation team and between rehabilitation services is important but can be difficult to achieve. It should be easier in hospitals: elderly care wards, rehabilitation wards, stroke, and orthogeriatric units are structures which greatly facilitate team working and coordination between disciplines and agencies. It can be considerably more difficult outside the hospital as natural teams do not usually exist but need to be purposefully formed around individual patients. Consider the common situation of an elderly housebound woman disabled by osteoarthritis who wishes to remain in her terraced council house. A range of services and equipment is required, the access to which may defeat all but the most tenacious of rehabilitation staff. The box on the previous page provides an example.

Rehabilitation research

Although the research base for rehabilitation has expanded considerably, evidence for effectiveness remains patchy. There is evidence to support the general concepts of the rehabilitation process for older

Findings of studies of rehabilitation in the home

<i>Occupational therapy</i>	
Bathing aids	Improved independence
Simple aids	Improved independence
Rheumatoid arthritis	Improved independence
<i>Physiotherapy</i>	
Falls	Improved balance
Osteoarthritis	Less pain greater mobility
Stroke	Improved mobility and independence
Parkinson's disease	Improved mobility

people, for stroke units and some evidence to support orthogeriatric units. Several randomised trials of domiciliary rehabilitation have been undertaken with encouragingly positive results.

Perhaps the most surprising finding from the domiciliary rehabilitation studies is how little therapy input is required. Several of the studies used only two or three visits, yet the health gain for the patient was considerable, especially for interventions consisting of occupational therapy.

Further reading

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Lesson of the Week

Reversible cardiogenic shock complicating subarachnoid haemorrhage

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Patients with subarachnoid haemorrhage who develop profound cardiogenic shock should have aggressive intensive care as good recovery is likely

When rupture of a cerebral aneurysm results in cardiovascular collapse the prognosis may seem hopeless and active management may be withheld. We report five cases of aneurysmal subarachnoid haemorrhage associated with severe myocardial dysfunction and cardiogenic shock. Intensive treatment, including intravenous inotropes and intra-aortic balloon counterpulsation, resulted in good neurological and cardiovascular recovery.

Case reports

Case 1—A 41 year old woman was admitted following a collapse preceded by a headache. Her initial Glasgow coma scale score was 6 and she had a fixed dilated right pupil. On intubation of the trachea, frank pulmonary oedema issued from the tracheal tube, and the chest radiograph confirmed pulmonary oedema. A cranial computed tomogram showed subarachnoid haemorrhage and a right subdural haematoma (fig 1). She

immediately underwent evacuation of the subdural haematoma. After fluid resuscitation she required infusions of noradrenaline (1 µg/kg/minute), adrenaline (0.6 µg/kg/minute), and dobutamine (14 µg/kg/minute) to maintain adequate blood pressure. A pulmonary artery catheter was inserted and confirmed cardiogenic shock (table 1). The following day cerebral angiography showed an aneurysm of the pericallosal branch of the right anterior cerebral artery. An echocardiogram showed global impairment of left ventricular function. Estimated left ventricular ejection fraction was 25-30%; this deteriorated to 20% the following day. Mechanical ventilation and inotropic support were continued, and five days later the ejection fraction had improved to 40%. The aneurysm was clipped the following day, and she was discharged from intensive care seven days later. She remained in hospital for a further nine days and was then discharged to rehabilitation. At follow up, three

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Table 1—Initial cardiac index, pulmonary artery occlusion pressure, worst left ventricular ejection fraction, and time spent in intensive care unit

	Cardiac index (l/min/m ²)	Pulmonary artery occlusion pressure (mm Hg)	Left ventricular ejection fraction (%)	Days in intensive care unit
Normal range	2.5-3.6	6-15	55-75	—
Case 1	3.9	21	20	13
Case 2	1.9	17	30	26
Case 3	3.1	30	35	22
Case 4	1.9	20	30	14
Case 5	2.3	36	20	41

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months after discharge from our hospital, she was neurologically normal with no evidence of cardiac failure.

Case 2—A 62 year old man was admitted to hospital after having collapsed. His family reported a seven day history of headache and vomiting. Initial Glasgow coma scale score was 14 and a computed tomogram showed an extensive subarachnoid haemorrhage with intraventricular haemorrhage and secondary hydrocephalus. Angiography showed an aneurysm of the right vertebral artery, intense spasm of the right vertebral artery, and mild spasm of the left vertebral artery. He was taken from the emergency department to the operating theatre, where a ventricular drain was inserted under general anaesthesia. Mechanical ventilation was continued because of a high requirement for oxygen and he was transferred to the intensive care unit. On admission a chest radiograph showed frank pulmonary oedema. Radial and pulmonary artery catheters were inserted and confirmed cardiogenic shock (table 1). Dobutamine (10 µg/kg/minute) and then adrenaline (0.3 µg/kg/minute) were given; noradrenaline (0.3 µg/kg/minute) was subsequently given for persistent hypotension. He remained hypoxic and hypotensive with a low cardiac index despite increasing the inotrope dose. Echocardiography showed severe diffuse left ventricular dysfunction, and the ejection fraction was 30%. As he was sedated for mechanical ventilation his neurological state could not be assessed accurately. After discussion with his family an intra-aortic balloon pump was inserted. Twenty four hours later dobutamine and noradrenaline were discontinued, but the adrenaline infusion was continued for a further 24 hours. The adrenaline was then discontin-

ued and the balloon pump removed. A repeat echocardiogram the next day showed normal left ventricular function. Eighteen days after admission the aneurysm was clipped. He remained in intensive care for a further eight days. He was discharged from hospital 41 days after admission. At follow up three months after discharge he was cognitively normal, able to look after himself without help, but had some difficulty swallowing. At the time of writing, he had no evidence of cardiac failure.

Case 3—A 56 year old woman was admitted after a collapse. Her admission Glasgow coma scale was 5 and she required intubation and ventilation for pulmonary oedema. After admission to the intensive care unit echocardiography showed diffuse left ventricular dysfunction (ejection fraction 35%), and pulmonary artery catheter data confirmed cardiogenic shock (table 1). She was treated with dobutamine (7 µg/kg/minute) and noradrenaline (0.4 µg/kg/minute). Angiography showed basilar tip and right posterior communicating artery aneurysms. The day after admission she underwent craniotomy and clipping of the basilar tip aneurysm. She received intravenous nimodipine for 16 days, dobutamine for 12 days, and noradrenaline for 18 days. She was discharged from intensive care 21 days after surgery. Repeat echocardiography showed some improvement in left ventricular function (ejection fraction 40%). She was discharged from the hospital on day 31. At three month follow up she was neurologically normal with no evidence of cardiac failure.

Case 4—A 50 year old woman was admitted after a collapse and respiratory arrest. She had regained consciousness after bag and mask ventilation but was confused, with pinpoint pupils, a right hemiplegia, and respiratory distress. Her admission Glasgow coma scale was 13, and she was profoundly hypoxic (arterial oxygen saturation (Sao₂) 80%) owing to pulmonary oedema. Mechanical ventilation was started and she was admitted to intensive care. Pulmonary artery catheter data confirmed cardiogenic shock (table 1). Echocardiography showed severe diffuse left ventricular dysfunction (ejection fraction 30%), and she was treated with an adrenaline infusion. Computed tomography and angiography showed a subarachnoid haemorrhage from a right vertebral artery aneurysm. At craniotomy a ruptured aneurysmal dilatation of the vertebral artery consistent with dissection was found, and trapping of the right vertebral artery aneurysm was performed. She received infusions of adrenaline (0.2 µg/kg/minute) for three days, noradrenaline (0.4 µg/kg/minute) for eight days, and dobutamine (4 µg/kg/minute) for nine days. Echocardiography after four days showed improved left ventricular function. She was discharged from intensive care after 14 days and to rehabilitation three weeks later. At three month follow up she was able to take care of herself, without help, but complained of slight clumsiness. She had no clinical evidence of cardiac failure.

Case 5—A 45 year old woman was admitted to a local hospital after crashing her car at low speed. At the scene she was drowsy, but orientated, and noted to be twitching and hypertensive. On arrival at hospital her Glasgow coma scale was 15, her systolic blood pressure was 90 mm Hg, and she complained of neck and back pain. There was no evidence of external injury, but a chest radiograph showed pulmonary oedema and a computed tomogram showed subarachnoid haemorrhage. In the receiving hospital emergency department her level of consciousness deteriorated and she became hypoxic (arterial oxygen pressure (Pao₂) 5.8 kPa). Mechanical ventilation was begun, and she was transferred to our hospital. On arrival in intensive care she was profoundly hypoxic (Pao₂ 7.9 kPa, fractional inspired oxygen (Fio₂) 1.0). A pulmonary artery catheter was inserted and confirmed cardiogenic shock (table 1). She was treated with intravenous dobutamine (up to 37 µg/kg/minute) and noradrenaline (up to 0.6 µg/kg/

Fig 1—Initial cranial computed tomogram in case 1, showing right subdural haematoma and subarachnoid blood

minute). Cerebral angiography showed right posterior, middle, and anterior cerebral artery aneurysms. Echocardiography showed global left ventricular dysfunction (ejection fraction 20-25%). She continued treatment with dobutamine; her ejection fraction at 13 days was 35%. This improved to 50-55% by day 21. On day 22 she underwent clipping of all three aneurysms. She required inotropic support for a further seven days. She was discharged 20 days after surgery to a rehabilitation centre, and at three month follow up she was neurologically normal with no clinical evidence of cardiac failure.

Comment

The incidence of subarachnoid haemorrhage is estimated at 12 in 100 000, and in Australia it affects about 2000 people every year. Cardiac arrhythmias and pulmonary oedema are common complications, occurring in 35% and 22% of patients, respectively.¹ Abnormal ST segments or T waves are present on echocardiography in 25% of patients on admission.¹ Profound myocardial dysfunction is less common. It occurred in five of the 168 patients with aneurysmal subarachnoid haemorrhage admitted to the intensive care unit at this hospital between February 1992 and August 1995, an incidence of 3%. In all five cases it was present within a few hours of the initial haemorrhage. This early myocardial dysfunction is most likely the result of a "sympathetic storm" occurring in response to a rapid increase in intracranial pressure at the time of the initial haemorrhage. Focal myocardial damage is found in 62% of patients dying of acute intracranial events,² and similar lesions are seen in animal models of brainstem coning, when an abrupt increase in intracranial pressure produces sympathetic storm with damage to the myocardium and pulmonary vasculature.^{3,4} The resulting left ventricular dysfunction, combined with increased pulmonary capillary permeability, produces severe pulmonary oedema.

Our experience shows that patients who develop profound myocardial dysfunction after sustaining a subarachnoid haemorrhage merit active and aggressive treatment as a good outcome is likely.

Management is complicated and best conducted in an intensive care unit offering invasive haemodynamic monitoring and support. The combination of electrocardiographic changes and cardiogenic shock may be misdiagnosed as acute myocardial infarction, but echocardiography shows global myocardial injury rather than segmental injury in the distribution of a particular coronary artery. Hypotension and hypoxaemia may aggravate the neurological injury and must be rapidly corrected.

Pulmonary oedema may be due to increased pulmonary capillary permeability with normal pulmonary artery occlusion pressure or to left ventricular dysfunction with increased pulmonary artery occlusion pressure, or a mixture of the two. Measurement of pulmonary artery occlusion pressure and cardiac output

permits logical selection and titration of inotropic treatment and may prevent inappropriate fluid restriction or treatment with diuretics, both of which may further reduce cardiac output. The careful titration of fluid and inotrope treatment is important in the days after subarachnoid haemorrhage as delayed ischaemic deficits due to cerebral arterial vasospasm are the major cause of death and disability in patients who survive the initial haemorrhage. Angiographic vasospasm occurs in 67% of patients and is symptomatic in half those cases. Prophylaxis and treatment of vasospasm include the use of calcium antagonists (nimodipine in Australia), expanding the intravascular volume, increasing the arterial blood pressure to supranormal levels, and accepting a relatively low haemoglobin concentration (hypertensive, hypervolaemic, haemodilution treatment). This combination treatment has been associated with a reduction in mortality from vasospasm and cerebral ischaemia, but is in many ways the opposite of the normal treatment for someone with severe myocardial dysfunction. In our patients careful monitoring of central haemodynamic variables, including pulmonary artery occlusion pressure and cardiac index, allowed us to pursue this course while monitoring myocardial function. Dobutamine and adrenaline were used to increase myocardial contractility when measured cardiac index was low, with noradrenaline where necessary to counteract the peripheral vasodilatation that both dobutamine and nimodipine can cause. Invasive monitoring of this treatment is essential as patient response varies enormously and alters as myocardial function changes.

The role of intra-aortic balloon counterpulsation in patients with acute brain injury is unclear. In case 2 it seemed to stabilise and then reverse a rapid deterioration in cardiac function. Its use carries considerable risk of complications, particularly intravascular thrombosis as the anticoagulants that are normally used are contraindicated after subarachnoid haemorrhage.

The duration of myocardial dysfunction in our patients varied enormously, with inotropic support being required for between three and 29 days. Nevertheless, good functional recovery occurred in the patients requiring prolonged support. Our experience shows that such patients should be offered prolonged intensive care treatment because good cardiovascular and neurological recovery is likely.

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ONE HUNDRED YEARS AGO

NURSES' HOURS OF WORK.

IT is hardly possible to take up a daily paper without seeing some remarks more or less to the purpose on the above subject. The number of nurses employed in our hospitals resolves itself into a question of money, and we feel sure that, given more liberal support by the public, the managers would be prepared to act more liberally by the nursing staff. It appears to us that an increase in the number of supernumeraries would be a step in the right direction: an available supernumerary might, by the

timely relief of an overtaxed sister or nurse prevent a breakdown; and the supernumerary also, by taking the whole day's duty, would enable the one whose "day off" she was taking to be entirely free from work or responsibility. We should like to add by way of explanation that a nurse's hours "on duty" do not in all hospitals represent hours of incessant hard work; though "on duty," she is not of necessity at work.

(*BMJ* 1896;ii:777.)