Key messages

- The rate of suicide in patients discharged from psychiatric hospital has been reported to be highest in the first 28 days after discharge
- In Scotland during 1968 to 1992 the standardised rate of suicide in the first 28 days after discharge almost trebled in female patients and decreased by 40% in male patients
- These period trends have occurred against a background of important changes in mental health service provision, with a 60% reduction in the number of psychiatric beds for adults between 1976 and 1992 and a trend towards shorter stays
- The increase in the suicide rate soon after discharge in female patients highlights the increasing importance of targeting services at recently discharged patients

years at risk did not decline. Therefore, although we cannot dismiss this explanation, it is not well supported by the data.

Whatever the reason for the increase in the rates in female patients, the main finding is that their risk of suicide in the first 28 days after discharge is increasing. Some of these deaths might be avoidable if community services are targeted at this high risk period. In Scotland, although the number of beds has been reduced, no comprehensive and integrated community service is yet in place.[26] If the increase is due to earlier discharge with inadequate community support, community services and planning procedures for discharge need to be developed before numbers of beds are reduced further.

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1 Goldacre M, Seagroatt V, Hawton K. Suicide after discharge from psychiatric inpatient care. *Lancet* 1993;**342**:283-6.
2 King EA, Barraclough B. Suicide *Lancet* 1993;**342**:744-5.
3 Isometsä E, Henriksson M, Heikkinen M, Lönnqvist L. Suicide after discharge form psychiatric inpatient care. *Lancet* 1993;**342**:1055-6.
4 King E. Suicide in the mentally ill: an epidemiological sample and implications for clinicians. *Br J Psychiatry* 1994;**165**:658-63.
5 Secretary of State for Health. *The health of the nation: a stategy for health in England*. London: HMSO, 1992.
6 Gunnell D, Frankel S. Prevention of suicide: aspirations and evidence. *BMJ* 1994;**308**:1227-33.
7 Audit Commission. *Making a reality of community care*. London: HMSO, 1986.
8 Morgan HG. Suicide prevention. Hazards on the fast lane to community care. *Br J Psychiatry* 1992;**160**:149-53.
9 Appleby L. Suicide risk in psychiatric patients: risk and prevention. *Br J Psychiatry* 1992;**161**:749-58.
10 World Health Organisation. *Mental disorders: glossary and guide to their classification in accordance with the ninth revision of the International Classification of Diseases (ICD-9)*. Geneva: WHO, 1978.
11 Kendrick S, Clark J. The Scottish record linkage system. *Health Bulletin* 1993;**51**:72-9.
12 Kreitman N. Suicide in Scotland, with special reference to youth. *Health Bulletin* 1994;**52**:300-9.
13 Charlton J, Kelly S, Dunnell K, Evans B, Jenkins R. Trends in suicide deaths in England and Wales. *Population Trends* 1992;**69**:10-6.
14 Diekstra RFW. The epidemiology of suicide and parasuicide. *Acta Psychiatr Scand Suppl* 1993;**371**:9-20.
15 Clayton D, Hills M. *Statistical methods in epidemiology*. Oxford: Oxford University Press, 1993.
16 Coleman M, Douglas A, Hermon C, Peto J. Cohort study analysis with a FORTRAN computer program. *Int J Epidemiol* 1986;**15**:1347.
17 Breslow NE, Day NE. *Statistical methods in cancer research. Volume II: the design and analysis of cohort studies*. Lyons: International Agency for Research on Cancer, 1987.
18 Baker RJ, Nelder JA. *The GLIM system, release 3.77*. Oxford: Numerical Algorithms Group, 1978.
19 Statistics and Epidemiology Research Corporation. *EGRET: epidemiological graphics, estimation and testing*. Seattle, WA: SERC, 1991.
20 McGonigal G, McQuade C, Thomas B. Accuracy and completeness of Scottish mental hospital in-patient data. *Health Bulletin* 1992;**50**:309-14
21 Geddes JR, Black RJ, Whalley LJ, Eagles JM. Decline in the diagnosis of schizophrenia. *Br J Psychiatry* 1994;**164**:564-5.
22 Flood RA, Seager CP. A retrospective examination of psychiatric case records of patients who subsequently committed suicide. *Br J Psychiatry* 1968;**114**:443-50.
23 Allebeck P, Varla A, Kristjansson E, Wistedt B. Risk factors for suicide among patients with schizophrenia. *Acta Psychiatr Scand* 1987;**76**:414-9.
24 Engberg M. Mortality and suicide rates of involuntarily committed patients. *Acta Psychiatr Scand* 1994;**89**:35-40.
25 Vassilas CA, Morgan HG. General practitioners' contact with victims of suicide. *BMJ* 1993;**307**:300-1.
26 Pullen I. Hunting the gowk?—psychiatric community care in Scotland. *BMJ* 1993;**306**:710-2.

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A survey of acute pain services in the United Kingdom

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The report of a joint working party of the Royal College of Surgeons of England and the College of Anaesthetists to consider pain after surgery called for the development of an acute pain service in every hospital performing surgery.[1] Despite this firm recommendation, it is perceived that such services are not as prevalent as might be expected. We undertook a survey of the current status of acute pain management in the United Kingdom.

Subjects, methods, and results

We sent a questionnaire to each of the tutors of the Royal College of Anaesthetists. The questionnaire sought details of the existence, staffing, and funding of any form of acute pain service in each hospital. An outline of the hospital's current methods of pain management after major surgery was also requested.

A total of 281 questionnaires was distributed and 221 replies (79%) were received. Of the responding hospitals, 97 (44%) reported having some form of acute pain service. Of these, 62 (28%) employed a specific pain nurse and 36 (16%) had specific "fixed" consultant sessions allocated to acute pain. Only 18 hospitals reported that additional funds had been provided for the purpose. In units where there was no acute pain service, the main reason (66/124; 53%) was financial. There was a regional variation in the availability of acute pain services (table).

Virtually every responding hospital used the newer analgesic techniques such as patient controlled analgesia or epidural opioid infusion to some extent, but in 42% of units (93) these techniques were used in less than 10% of patients after major surgery. Intermittent intramuscular injection of opioid remains the most commonly used technique in most hospitals, with 47% of units (104) using it in more than 50% of their patients.

Comment

More than four years after the publication of the report of the joint working party of the Royal College of Surgeons of England and the College of Anaesthetists on pain after surgery[1] and despite reports of acute pain services in the United Kingdom,[2,3] it is disappointing that less than half of the hospitals that responded in this survey had an acute pain service. However, the authors are aware that several hospitals have since developed such a service. Specific funding allocated to enable the establishment of an acute pain service was reported by

Hospitals responding to questionnaire that had an acute pain service by region

Region	No (%) of hospitals
East Anglia	5 (62)
Mersey	3 (37)
North East Thames	9 (45)
North West Thames	8 (44)
North West	7 (50)
Northern	5 (35)
Oxford	5 (62)
South East Thames	5 (33)
South West Thames	6 (50)
South West	7 (58)
Trent	4 (28)
West Midlands	5 (28)
Wessex	4 (44)
Yorkshire	7 (50)
Wales	6 (43)
Scotland	7 (41)
Northern Ireland	4 (67)

only 18 hospitals and the relative rarity of specific "pain nurses" and dedicated medical sessions gives concern as to the permanence of these services. *Pain, Discomfort and Palliative Care*, a Welsh Health Planning Forum document, gives clear advice and targets for both the purchaser and the provider—including the establishment of an acute pain service in all hospitals in Wales by 1995.[4] However, at the time of this survey, fewer than 50% of responding units in Wales had achieved this.

Although patient controlled analgesia and epidural opioid administration were available in most units, in many units such techniques were seldom used. Intermittent intramuscular injection of opioid is still widely used in a large number of hospitals. This may not necessarily be detrimental; good levels of analgesia can be achieved, as has been shown by Gould *et al.*[5]

It is to be hoped that an increasing number of hospitals will establish acute pain services. To this end, continued pressure should be placed on the purchaser to demand optimal acute pain management from their provider units—and be prepared to pay for it.

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1 Working Party of the Commission on the Provision of Surgical Services. *Pain after surgery*. London: Royal College of Surgeons of England, College of Anaesthetists, 1990.
2 Wheatley RG, Madej TH, Jackson IJB, Hunter D. The first year's experience of an acute pain service. *Br J Anaesth* 1991;67:353-9.
3 Cartwright PD, Helfinger RG, Howell JJ, Siepmann MK. Introducing an acute pain service. *Anaesthesia* 1991;46:188-91.
4 Protocol for Investment in Health Gain. *Pain, discomfort and palliative care*. Cardiff: Welsh Health Planning Forum, 1992.
5 Gould TH, Crosby DL, Harmer M, Lloyd SM, Lunn JN, Rees GAD, *et al.* Policy for controlling pain after surgery: effect of sequential changes in management. *BMJ* 1992;305:1187-93.

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Pulmonary oedema and haemoptysis induced by strenuous swimming

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Pulmonary haemorrhage induced by exercise is well known in racehorses.[1] Recent studies attribute the phenomenon to rupture of pulmonary capillaries because of a large increase in pulmonary blood flow and pressure.[2] Pulmonary oedema has previously been described in scuba divers and swimmers who have been immersed in very cold water, albeit without excessive exertion.[3] We report on a group of highly trained swimmers in whom severe dyspnoea and haemoptysis developed during the first 45 minutes of strenuous swimming in temperate Mediterranean waters.

Patients, methods, and results

Thirty young men on a military fitness training programme were engaged in a swimming time trial over 2·4 km in the open sea. The sea was calm and the measured water temperature 23°C. They swam in the supine position wearing only a bathing suit and using fins. Because of the high heat load expected, the swimmers had been instructed to drink large quantities of water before the swim, to avoid becoming dehydrated. They each drank about five litres of water during the two hours preceding the exercise.

Pronounced shortness of breath developed within 45 minutes in eight of the subjects, forcing five of them to terminate the swim prematurely. Since dyspnoea persisted, they were examined in the clinic at the training facility 30-60 minutes after the drill, and oxygen saturation was measured using a pulse oximeter. Those in more severe distress were given 20 mg intravenous frusemide and all were transferred, receiving oxygen by mask, to a local hospital. The patients' findings and the results of the laboratory investigation carried out in the emergency department are shown in the table. None had abnormal cardiovascular signs or hypertension. Their symptoms and signs resolved spontaneously during an overnight stay in hospital. All eight returned to similar activities and completed the training programme, apart from two (cases 5 and 7) who had recurrent episodes of pulmonary oedema or haemoptysis, or both, when they swam.

Summary of patients' characteristics and laboratory results

	Case No							
	1	2	3	4	5	6	7	8
Age (years)	18	18	19	18	18	19	18	18
In clinic at training facility:								
Cough	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Haemoptysis:								
Complained of	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observed	No	No	No	Yes	Yes	No	No	No
Oxygen saturation (%)	95	90	98	84	81	88	87	96
In emergency department:								
Partial pressure (mm Hg)								
Oxygen	84	82	85	66	62	77	82	85
Carbon dioxide	38	36	39	39	37	40	37	38
pH	7·42	7·38	7·45	7·38	7·41	7·39	7·41	7·37
Infiltrates in chest x ray film	Yes	No	No	No	No	No	Yes	No
Recurrent episodes	No	No	No	No	Yes	No	Yes	No

Comment

We surmise that three precipitating factors combined to produce a transient increase in the pulmonary capillary pressure: maximal exertion, immersion, and overhydration. Exercise causes an increase in cardiac output to meet the tissues' increased demand for oxygen. However, the increase in cardiac output will by itself rarely if ever raise pulmonary capillary pressure to the point of rupture in humans; pulmonary oedema and haemoptysis have never been reported even in top Olympic athletes with above normal cardiac performance. Immersion causes central blood pooling, thus increasing cardiac preload.[4] Water overload, the result of well intentioned but overzealous concern about dehydration, probably further increased pulmonary vascular pressure.

Haemoptysis and pulmonary oedema recurred in two of the patients without an increased fluid intake; recurrences gradually became less severe, gradually proceeding to complete spontaneous resolution. They may have been the result of residual damage to the alveolar-capillary unit, which finally healed.

Pulmonary oedema associated with immersion has previously been described in 11 divers and swimmers exposed to cold water.[3] Although the subjects did not engage in heavy exercise and their water balance was