considered in the differential diagnosis of acute severe chest pain in young fit adults.

DYNESH RITTOO

Department of Surgery,
Wythenshawe Hospital,
Manchester M23 9LT

D B RITTOO

Department of Anaesthesia,
Hope Hospital,
Manchester University,
Salford M6 8HD

DYLMITR RITTOO

Department of Cardiology,
Western General Hospital,
Edinburgh EX4 2XU

1 Henry JA. Ecstasy and the dance of death. *BMJ* 1992;305:5-6. (4 July.)

EDITOR,—John A Henry's editorial¹ and Ibrahim H Fahal and colleagues' drug point² highlight a growing medical problem arising from the recreational misuse of ecstasy (3,4-methylenedioxymethamphetamine or MDMA). For each relatively rare and major complication of ecstasy, however, we suspect that many lesser events will go unrecorded. These will take their silent toll of the people concerned and consume valuable resources in medical institutions.

Two young men were brought to the accident and emergency department at this hospital by ambulance. The first was aged 18 and had suffered a fit after taking ecstasy. He admitted to having had a fit one month earlier, also after misuse of ecstasy. Examination showed a sinus tachycardia but no other abnormality. He discharged himself from casualty. The second patient collapsed after complaining of a headache and was carried in on a stretcher. We suspected that he had had a fit, but no eye witnesses accompanied him. He was drowsy but scored 15 on the Glasgow coma scale. He had a tachycardia and both biochemical and electrocardiographic evidence of hyperkalaemia. He was given activated charcoal and admitted for overnight observation. On discharge the next morning he stated that his experience would not stop him using ecstasy again.

We suspect that many acute hospitals throughout the country could relate similar stories. Although reports of chronic psychosis and acute rhabdomyolysis associated with misuse of ecstasy give cause for concern, we believe that the main burden for hospital and ambulance services will be the more mundane and often unreported effects of acute intoxication.

J SAWYER
W P STEPHENS

Trafford General Hospital,
Manchester M13 3SL

1 Henry JA. Ecstasy and the dance of death. *BMJ* 1992;305:5-6. (4 July.)
2 Fahal IF, Sallomi DF, Yaqoob M, Bell GM. Acute renal failure after ecstasy. *BMJ* 1992;305:29. (4 July.)

EDITOR,—In his otherwise comprehensive editorial on the medical complications of using ecstasy, or 3,4-methylenedioxymethamphetamine (MDMA), John A Henry omits cerebrovascular disease.¹ Sympathomimetics are widely recognised causes of both cerebral infarction and haemorrhage.² We have reported four cases of spontaneous intracerebral haemorrhage related to ingestion of ecstasy or amphetamine.³ The patients were aged 16-30, and two had consumed ecstasy inadvertently in spiked drinks. Three patients presented with hemiparesis and one in coma. Cerebral angiography was performed in all four. Three had normal intracranial vessels and made good functional recoveries. The other, whose computed tomogram showed a considerably larger

haematoma, had an arteriovenous malformation and died.

Drug misuse should be considered in young patients presenting with stroke, and serum and urine should be screened for ecstasy and amphetamine if indicated. We agree wholeheartedly that the popular belief that ecstasy is a safe recreational drug is erroneous.

R N DE SILVA
D P HARRIES

Institute of Neurological Sciences,
Southern General Hospital,
Glasgow G51 4TF

1 Henry JA. Ecstasy and the dance of death. *BMJ* 1992;305:5-6. (4 July.)
2 Kaku DA, Lowenstein DH. Emergence of drug abuse as a major risk factor for shakes in young adults. *Ann Intern Med* 1990;113:821-7.
3 Harries DP, de Silva RN. Ecstasy and intracerebral haemorrhage. *Scott Med J* (in press).

Service increment for teaching and research

EDITOR,—The editorial and two papers examining the allocation of the service increment for teaching and research were not sufficiently critical of what was a pragmatic solution to an unrelated problem.¹⁻³ The introduction of the Resource Allocation Working Party formula in the mid-1970s, which allocated on the basis of need rather than historic costs, threatened the privileged position of the teaching hospitals, which had previously received 14% of revenue with only 6% of beds.⁴ Legitimised by some questionable statistical work,^{5,6} the service increment for teaching helped to paper over the fact that the large hospitals, especially those based in London, were too expensive by attributing much of their excess costs to teaching.

The situation of the expensive teaching hospitals was further exposed by the NHS reforms, which link hospitals' revenue much more closely with services delivered and average prices. This prompted the inclusion of a research component in the service increment for teaching and a flurry of work on ways to allocate it, sometimes resulting in perverse incentives.

The King's study, for example, weights the number of student full time equivalents per specialty by the costs of that specialty over and above costs in non-teaching hospitals.⁷ This makes the untenable assumption that all excess costs are directly proportional to teaching. It is then tautological to show an association between excess prices and the service increment for teaching offset so calculated. More worrying is that the same number of students will attract different amounts of money depending on the excess costs of the specialty. This underwrites inefficiency and, because it ignores the actual teaching input, creates no incentive to teach the students allocated, let alone teach them well.

The Leicester study, which used students' diaries, found considerable variation in teaching input.⁸ It is being repeated with quality measures added. Universities should take advantage of this opportunity to exert more control over hospital based teaching, ensuring the quality of the teaching and learning environment rather than spending time developing sophisticated techniques for allocating a crudely calculated sum.

As health care in London is rationalised and more students are taught in "non-teaching" hospitals, where they can get a broader and more humane experience, so the current arrangements will come under greater critical scrutiny. A more efficient approach is needed whereby students are taught in the cheaper centres (while quality is ensured) rather than money being allocated to the more expensive ones.⁹ Accredited hospitals and

health centres should be able to bid for the opportunity to teach students as part of a clear syllabus, with a realistically calculated service increment for teaching as an explicit payment.

Surely the time has come to recognise that the service increment for teaching was a post hoc rationalisation for expensive service delivery generally unrelated to teaching. Where this expense is justified ways to fund it explicitly should be sought; where it is not justified this hidden subsidy should be replaced by improvements in efficiency.

TREVOR A SHELDON

Academic Unit of Public Health Medicine,
Leeds University,
Leeds LS2 9LN

1 Chantler C. Service increment for teaching and research. *BMJ* 1992;305:71-2. (11 July.)
2 Black GB, Bevan G, Peters TJ, Eddleston ALWF. King's model for allocating service increment for teaching and research (SIFTR). *BMJ* 1992;305:95-6. (11 July.)
3 Smith CL. Service increment for teaching and research (SIFTR): the Southampton experience. *BMJ* 1992;305:97-8. (11 July.)
4 Forsyth G. *Doctors and state medicine*. London: Pitman, 1973.
5 Culyer J, Wiseman J, Drummond M, West P. What accounts for the higher costs of teaching hospitals? *Social and Economic Administration* 1978;12:20-30.
6 Straf M. Revenue allocation by regression: NHS appropriations for teaching hospitals. *Journal of the Royal Statistical Society Series A* 1981;144:80-4.
7 Sheldon TA, Clarke M, Woods J. The student diary survey: a method of monitoring hospital-based medical education. *Med Educ* 1991;25:213-23.
8 Sheldon TA. *The Leicester University study of undergraduate clinical teaching*. Leicester: Trent Regional Health Authority and Department of Public Health Medicine, Leicester University, 1990.
9 Sheldon TA. The NHS review and the funding of teaching hospitals. *Management in Medicine* 1991;5(2):6-17.

EDITOR,—C L Smith states that "sufficient detail of the attributable costs [under the service increment for teaching and research] can be derived to allow for accountability and justification of use."¹ The figures given in the paper do not seem to support this. Costs derived from the survey that Smith quotes, and about which we are given no details, account for only 35% of the service increment for teaching and research allocated. Virtually no evidence is offered in support of the contention that costs related to infrastructure account for 70% of the total allocation.

There is a basic difficulty in that we know what the difference in cost between teaching and non-teaching hospitals is but not what it should be. Drawing conclusions about revenue costs from differences in use of space is problematic: buildings represent only a small proportion of the cost of health services, and space is unlikely to be a good indicator of the costs of activity. It may well be true, as Smith argues, that the true costs are greater than the funding, but on the evidence offered this remains unproved.

CHARLES NORMAND
MARTIN MCKEE

Department of Public Health and Policy,
Health Services Research Unit,
London School of Hygiene and Tropical Medicine,
London WC1E 7HT

1 Smith CL. Service increment for teaching and research (SIFTR): the Southampton experience. *BMJ* 1992;305:97-8. (11 July.)

Coronary vasospasm and sumatriptan

EDITOR,—B H C Stricker presents data on 12 Dutch patients who experienced chest symptoms, variously described as anginal pain, substernal pressure and discomfort, heaviness, and tightness.¹ It seems that electrocardiograms were obtained in only three patients and were normal.

We do not believe that these cases can legitimately be described as similar to the case reported

by F Willett and colleagues, in which chest pain was accompanied by electrocardiographic changes.² As W M Castle and V E Simmons discuss, this patient had a retrospective diagnosis of Prinzmetal's angina,³ which the datasheet states is a contraindication to sumatriptan.

Stricker refers to recurrence of symptoms after rechallenge. This is of considerable interest, especially in case 2, for despite chest symptoms the patient apparently used the drug more than 10 times. In our clinical trials programme 15 patients who had experienced at least one episode of chest discomfort, pain, pressure, or tightness were formally rechallenged under intensive electrocardiographic monitoring. Five experienced the same symptoms, and two of these five also experienced the symptoms after placebo. In none of these patients was any electrocardiographic change detected.

During the clinical development programme for sumatriptan almost 6000 electrocardiograms were recorded. They were obtained during and after the administration of subcutaneous sumatriptan in more than 1200 patients and after oral sumatriptan in more than 400 patients and volunteers. There is no evidence of a link between the chest symptoms which occur in 3-5% of patients and electrocardiographic change or other evidence of myocardial ischaemia except in extremely rare cases such as that reported by Willett and colleagues.

Sumatriptan (both oral and subcutaneous forms) has been available in the Netherlands for over a year and is also registered in 21 countries worldwide. The 12 cases of chest pain that Stricker reports are not surprising in the context of extensive patient exposure.

Stricker also refers to an article by Chester *et al*, who concluded that the minor vasoconstriction after sumatriptan seen in vitro in human coronary arteries might be important in the presence of atheroma.⁴ Their data, however, do not support this conclusion. They in fact show that in atheromatous sections of artery the constriction was even less than that in "normal" segments.

From our extensive study of the cardiovascular safety of sumatriptan in large numbers of patients we can find no evidence that the chest pain is due to myocardial ischaemia except in very rare cases, as noted above. Nevertheless, we recognise that the cause of these chest symptoms must be established and we are undertaking a programme of studies to this end in both patients and volunteers.

D K LLOYD
A J PILGRIM
V E SIMMONS

Glaxo Group Research,
Greenford,
Middlesex UB6 0HE

- 1 Stricker BHC. Coronary vasospasm and sumatriptan. *BMJ* 1992;305:118. (11 July.)
- 2 Willett F, Curzen N, Adams J, Armitage M. Coronary vasospasm induced by subcutaneous sumatriptan. *BMJ* 1992;304:1415. (30 May.)
- 3 Castle WM, Simmons VE. Coronary vasospasm and sumatriptan. *BMJ* 1992;305:117-8. (11 July.)
- 4 Chester AH, Martin GR, Bodelsson M, Arneko-Nobin B, Tadjkarini S, Tornebrandt K, *et al*. 5 Hydroxytryptamine receptor profile in healthy and diseased human epicardial coronary arteries. *Cardiovasc Res* 1990;24:932-7.

Treatment of natal cleft sinus

EDITOR,—H T Khawaja and colleagues conclude that widespread use of primary closure would improve the management of patients with uncomplicated natal cleft sinus.¹ Although trials such as this are useful in showing the superiority of one treatment over another, results are influenced by patient selection in a condition in which successful outcome owes much to clinical enthusiasm.² Such results must therefore be interpreted with caution before their general application is assumed. We

recently reviewed the case records of 229 patients undergoing both emergency and elective operations for pilonidal sinus of the natal cleft at Selly Oak Hospital, Birmingham, from 1986 to 1991. Our findings confirm that this condition is an important cause of morbidity in young people, for whom treatment is often unsatisfactory.³

Of 121 patients undergoing elective procedures, 20 were treated by simple excision and primary closure (by different consultants and senior registrars) and primary healing was obtained in only seven of these 20. Complete healing before discharge was documented in only nine cases, and nine patients defaulted from follow up. Better results were obtained in 29 patients whose defect was repaired with a transposition rhomboid flap (by one consultant with a special interest in this procedure): 20 of these wounds showed primary healing. Encouraging results have been reported with this technique,⁴ and a rotation flap also has the advantage of flattening the natal cleft and covering the area with good quality skin, which may decrease the likelihood of future recurrence.⁵

Of 108 emergency operations for pilonidal abscesses and discharging sinuses, 85 were undertaken by senior house officers, usually out of normal working hours (72 cases). Although outpatient follow up of these patients was often arranged, review was often unsatisfactory, with 30 patients defaulting and 30 being discharged before complete healing. This may be why 45 of those who had emergency operations for a discharging sinus had had at least one previous operation.

We believe that our findings are typical of the difficulties in treating natal cleft problems in a busy district general hospital. Considerable improvements in treating this common and often neglected condition could probably be achieved by simple measures, including a defined plan of management, better supervision of junior staff, and improved education of patients.

J HOLLINGWORTH

Department of Surgery,
Leicester General Hospital,
Leicester LE5 4PW

Queen Elizabeth Hospital,
Birmingham B15 2TH

Selly Oak Hospital,
Birmingham B29 6JD

D M HEGARTY

H D KAUFMAN

- 1 Khawaja HT, Bryan S, Weaver PC. Treatment of natal cleft sinus: a prospective clinical and economic evaluation. *BMJ* 1992;304:1282-3. (16 May.)
- 2 Postnatal sinus [editorial]. *BMJ* 1980;281:959.
- 3 Fishbein RH, Handelsman JC. A method for primary reconstruction following radical excision of sacrococcygeal pilonidal disease. *Ann Surg* 1979;190:231-5.
- 4 Azab ASG, Kamal MS, Saad RA, Abou AL, Atta KA, Ali NA. Radical cure of pilonidal sinus by a transposition rhomboid flap. *Br J Surg* 1984;71:154-5.
- 5 Monro RS, McDermott FD. The elimination of causal factors in pilonidal sinus treated by Z-plasty. *Br J Surg* 1965;52:177.

EDITOR,—H T Khawaja and colleagues' short report suggesting that excision and primary closure of pilonidal sinus is cheaper and more effective than secondary healing is contentious.¹ Though we commend the authors for having successfully run a prospective trial in this infuriating condition, the numbers in each group (23 patients) are small and cannot support definitive statements on treatment policy.

Whether the wounds in the primary closure group were sutured in the midline or were offset is not stated. Why the wounds left to heal by secondary intention were packed for a week after surgery is unclear. Early use (after 72 hours) of Silastic foam dressing would have reduced or obviated the need for visits by a district nurse and so reduced costs.²

Details of cost analysis are not divulged. Pre-

vious large studies show healing by secondary intention to be 40-50% cheaper than primary closure,^{3,4} which is in direct contrast to the authors' findings.

No comment is made on the 26% of patients who underwent primary closure and subsequently experienced complications, other than that "healing of unsuccessful cases was not significantly different from that of the secondary healing group." No details are given about morbidity associated with breakdown of the wound, and we question whether a method with a complication rate of this magnitude is effective or even acceptable.

Follow up was limited to one year, but recurrence rates increase with length of follow up; most recurrences occur within three years.⁵

In conclusion, therefore, this trial, though having the advantage of being prospective, suffers from including few patients and fails to address the controversial points it raises; it cannot justify definitive statements concerning surgical management.

D P BERRY
K G HARDING

Wound Healing Research Unit,
University Department of Surgery,
University of Wales College of Medicine,
Cardiff CF4 4XN

- 1 Khawaja HT, Bryan S, Weaver PC. Treatment of natal cleft sinus: a prospective clinical and economic evaluation. *BMJ* 1992;304:1282-3. (16 May.)
- 2 Wood RAB, Hughes LE. Silicone foam sponge for pilonidal sinus: a new technique for dressing open granulating wounds. *BMJ* 1975;iv:131-3.
- 3 Allen-Marsh TG. Pilonidal sinus: finding the right track. *Br J Surg* 1990;77:123-32.
- 4 Bissett IP, Isbister WH. The management of patients with pilonidal disease—a comparative study. *Aust NZ J Surg* 1987;57:939-42.
- 5 Notaras MJ. A review of 3 popular methods of treatment of postnatal (pilonidal) sinus disease. *Br J Surg* 1970;57:886-90.

AUTHORS' REPLY.—In the patients in our primary closure group the wounds were sutured in the midline. The wound cavity changes rapidly in shape over the first few days, which is why we packed the wound for a week after surgery before using Silastic foam dressing.

Use of resources was measured by patient days in hospital, visits to the outpatient department, visits by a district nurse, and days off work. The main measures of effectiveness were days to complete healing and recurrence at six and 12 months.

The previous study showing that secondary healing is cheaper than primary closure was a retrospective analysis, with follow up by postal questionnaire or telephone (77% response rate), of patients treated for either natal cleft sinus or abscess five to nine years previously.¹ Information regarding time to healing, time off work, and recurrence was not available from all responders. Those who had primary closure were kept in hospital for a mean of 8.4 days. In his review Allen-Mersh pointed out that primary healing was achieved within two weeks in over 90% of the patients included in 17 published studies.² He assumed, however, that such patients stayed in hospital for seven nights. The main cost differential between the two treatment groups was the length of stay in hospital. If the costs were recalculated with the patients who had primary closure being treated as day cases (as in our study) primary closure would be much cheaper than secondary healing.

Our paper clearly states the morbidity in the six patients in the primary closure group whose wounds failed to heal. The wound broke open after removal of sutures in four cases; two patients required early removal of sutures owing to wound haematoma and wound infection.

D P Berry and K G Harding fail to understand the essential message of our paper. We compared two commonly used methods of treating pilonidal