side of the bed, insertion of a percutaneous endoscopic gastrostomy tube will not necessarily prevent this as these tubes may themselves be pulled out by a determined patient—indeed, some authorities believe that psychosis and dementia are relative contraindications to insertion.[8]

Thus percutaneous endoscopic gastrostomy is not the end of the line for nasogastric feeding but an example of one of several useful techniques that may be considered for long term enteral feeding. Nasoenteral tubes will continue to provide the main route of access for most patients requiring enteral nutrition.

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AUTHOR'S REPLY,—Jason Payne-James may have read our editorial rather too closely. It is, of course, important to distinguish the functions of an editorial from those of a detailed review. The purpose of our piece, to which Payne-James takes such exception, was to introduce percutaneous endoscopic gastrostomy and not to review the whole topic of enteral nutrition in hospital practice.

As our article was written to accompany the study by Park and colleagues it seems curious that it could be interpreted as recommending that endoscopic gastrostomy feeding should replace nasogastric feeding for short term nutritional support over just a few days. Even the most ardent gastrostomist might baulk at such a suggestion.

It is correct to suggest that the main hazards of operative gastrostomy are related to the anaesthetic. But it has always seemed a little unfair to blame the anaesthetist for a postoperative death when he or she was called into action only because a surgeon decided to perform an operation. Despite Payne-James's comments we continue to recommend endoscopic rather than operative gastrostomy for most of our patients.

A comprehensive review of enteral nutrition would surely have emphasised the radiologist's potential in siting gastrostomy tubes. Many endoscopists faced with ever increasing workloads would welcome the help of colleagues from the *x* ray department in lightening their burden.

The image of a patient with hands bandaged to the sides of the bed was intended to be emotive but does illustrate the point about the continuing discomfort that patients can experience with nasogastric tubes. Our clinical experience of nutrition in hospital practice, like that of the patients in Park and colleagues' study, is that a gastrostomy tube is not only more successful but is better tolerated over the long term than a nasogastric tube. As with any treatment, the patient's mental state must be taken into consideration when the route for nutritional support is being planned.

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Psychosocial problems in epilepsy

EDITOR,—J E Chaplin and colleagues' paper provides interesting evidence on the psychosocial problems of people in whom epilepsy is newly diagnosed.[1] Similar evidence of good adjustment is available for another group of people with epilepsy—those in whom it is in remission—from the Medical Research Council's antiepileptic drug withdrawal study.[2] As part of this study, information was collected from over 600 patients about employment, social relationships, leisure activities, feelings of stigma, and general wellbeing. The study was a randomised clinical trial, and the patients in it were a selected group, but comparison of their clinical and demographic characteristics, as well as their responses to the psychosocial questionnaire, with those of patients who were not randomised indicated that the results can be generalised to less selected populations.

Overall, in this group of patients levels of distress over epilepsy seemed to be low.[3] Respondents emerged as being well adjusted to their epilepsy and experiencing few problems because of it. Rates of employment were comparable with those in the general population, and few patients reported any instances of discrimination at work attributable to epilepsy. Although the rate of marriage and cohabitation was lower than in the general population, it was higher than that reported for people with chronic epilepsy, and the proportion of people living alone did not differ from that in the population as a whole. Few patients reported any restrictions in their social life because of their epilepsy, and few reported feeling stigmatised because of it. Like Chaplin and colleagues, however, the Medical Research Council's study found that the extent of reported problems was related to the recency of the last seizure.

Similar findings of good adjustment have been reported by Trostle *et al*, who also found little evidence of psychosocial impairment among the population they studied.[4] These findings of good adjustment are important and contrast greatly with the view of epilepsy as universally stigmatising. The process of adjusting to epilepsy is one about which we know relatively little, and further research is needed. We await with interest the results of the five year follow up of the patients in the national general practice study.

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Treating obesity in children

EDITOR,—We are concerned about the treatment of obesity in children. A patient of ours, a 13 year old girl, was referred by her general practitioner for assessment of right sided abdominal pain. In the referral letter the general practitioner stated that she was not taking any medication. She was admitted for observation, and the pain settled spontaneously.

On close questioning for clerking on admission she admitted that she took tablets because her "metabolism was slow," resulting in her being overweight. Her mother told us that she had always been fat as a baby, and had first been treated for obesity at the age of 4. She was currently being treated at a private slimming clinic and had been prescribed a 12 month course of "thyroid tablets," but she did not know the dosage. Her father brought the drugs in: thyroxine sodium 100 μg daily, phentermine 15 mg daily, and "Armour thyroid" two tablets daily. On repeat questioning the patient did not have any thyroid, cardiac, gastrointestinal, or menstrual irregularities. She had not had any blood tests at the private clinic. Her height was on the 10th centile for her age and her weight just below the 50th centile.

Thyroxine sodium is licensed for use in hypothyroidism.[1] Phentermine is structurally and pharmacologically related to the amphetamines. It is "an anorectic agent intended for short term use as an adjunct to the treatment of patients with moderate to severe obesity for whom close support and supervision are also to be provided."[1] It is not recommended for use in children. We could find no reference to using these drugs to treat obesity in children.

In addition to the obvious dangers of these drugs being prescribed to such a patient it is of concern that the general practitioner was not informed that his patient was being prescribed these drugs and that neither the patient nor her mother knew the exact nature or dosage of the drugs. The matter is being taken further by one of the consultant paediatricians, and we hope that such practices may be stamped out.

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Coronary vasospasm and sumatriptan

EDITOR,—F Willett and colleagues describe an episode of coronary vasospasm after subcutaneous administration of sumatriptan.[1] The details of this case are well known to Glaxo.

The patient under discussion had complained of intermittent retrosternal chest pain for 11 months before admission in December 1991, not only in relation to methysergide and sumatriptan but also on waking. In August 1991 atypical chest pain was noted. As a consequence of the challenge with sumatriptan that the authors describe the chest pain was confirmed as Prinzmetal's angina, a point omitted in the published drug point. It is clear in retrospect, however, that the underlying problem predated treatment with sumatriptan by several months.

Although chest symptoms have been noted with sumatriptan, extensive clinical investigations have not shown ischaemic electrocardiographic changes in otherwise healthy patients after subcutaneous or oral administration. Postmarketing experience has likewise failed to show any evidence of myocardial ischaemia in the absence of symptomatic cardiac disease. Furthermore, although Willett and colleagues refer to a second report of coronary vasospasm, that report was of a patient with a history of angina and heavy smoking who was treated on several occasions with sumatriptan without chest pain but experienced a myocardial infarction two weeks after the last dose. A causal relation is therefore considered to be unlikely.

Underlying ischaemic heart disease and Prinz-

metal's angina are both given as contraindications in the datasheet for sumatriptan; the datasheet also includes advice against the concomitant use of ergotamine. Willett and colleagues' report therefore clearly highlights the need for these warnings and emphasises that sumatriptan should be avoided if there is a history of ischaemic heart disease or undiagnosed chest pain.

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1 Willett F, Curzen N, Adams J, Armitage M. Coronary vasospasm induced by subcutaneous sumatriptan. *BMJ* 1992;**304**:1415. (30 May.)

AUTHORS' REPLY,—Though we agree that the case that we reported highlights the need for warnings against using sumatriptan in ischaemic heart disease and known cases of coronary vasospasm, we believe that we clearly implied that the injection of sumatriptan given in this case did cause coronary vasospasm, which is commonly thought to be the underlying mechanism of so called Prinzmetal's angina. We therefore believe that we expressed this diagnosis clearly.

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EDITOR,—In their report of a case of coronary vasospasm after subcutaneous administration of sumatriptan F Willett and colleagues state that the Committee on Safety of Medicines has received only one similar report.[1] The Netherlands Centre for Monitoring of Adverse Reactions to Drugs has received reports of 12 similar cases, mostly after oral intake (table).

All patients experienced chest symptoms, almost invariably within one hour after administration of sumatriptan. Symptoms varied from substernal tightness to severe cramping angina-like pain radiating to the left arm and hand. In one patient a transient increase in blood pressure to 200/120 mm Hg was noted, which later fell to 160/90 mm Hg. Four reporting medical practitioners classified the symptoms as anginal, and two of them notified these as "classical" or "real" angina pectoris. In all patients symptoms resolved without further treatment.

Electrocardiograms were normal in cases 3, 5, and 8 but were obtained after the chest symptoms had resolved. An echocardiogram and results of an exercise test performed after the first episode of chest pain in case 8 were normal. Except for one patient, who was said to have had a similar reaction to ergotamine in the past, none of the patients had had similar episodes before using sumatriptan and none developed such symptoms after stopping it.

Early studies suggested that serotonin-1 ($5\text{-}HT_1$) receptors are largely confined to the cranial circulation. Serotonin induces contraction of isolated epicardial coronary arteries,[2] which seems to be unopposed.[3] As serotonin-2 receptors are more common than serotonin-1 receptors in coronary arteries the effect of sumatriptan on coronary vasculature seems to be relatively mild.[4] Even so, sumatriptan elicited a vasoconstrictive response 30% of that to serotonin.[3]

Although this may not be clinically relevant in most patients, Chester *et al* suggested that when atherosclerotic changes decrease the luminal cross sectional area of the artery, problems may arise as the response to serotonin-1 is maintained in the area distal to an atherosclerotic occlusion.[3] This may be important, as the enhanced vasoconstrictive response of atherosclerotic isolated epicardial coronary arteries to histamine[2] is also seen to serotonin.[5] A study by the manufacturer of sumatriptan in 10 patients with existing or suspected coronary artery disease showed an average constriction of coronary arteries of 13·9% 10 minutes after subcutaneous administration of 6 mg.[5] Although aortic and pulmonary artery pressures were raised, cardiac output did not change. Although no electrocardiographic abnormalities were noted, only one patient experienced chest tightness (Glaxo, unpublished data).

The case reported by Willett and colleagues shows that ST elevation may occur after subcutaneous sumatriptan,[1] and this Dutch series shows that oral intake may also be followed by anginal pain.

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Harm minimisation for drug misusers

EDITOR,—John Strang and Michael Farrell suggest that maintenance programmes with oral methadone may reduce the harm that drug misusers do to themselves.[1] Colin Brewer and colleagues chastise the authors for being too timid and criticise the many British clinicians who are reluctant to prescribe long term maintenance treatment with generous dosages of methadone.[2] Unfortunately, in their contributions none of these workers consider the harm that long term maintenance policies can do to those other than the drug misusers accepted for treatment.

I work as a general practitioner in a part of the country where drug misusers are commonly treated with long term maintenance programmes. Methadone is most commonly prescribed, but dihydrocodeine, diazepam, and temazepam are also often used. A greater emphasis is placed on achieving a stable lifestyle than on working towards a life without drugs of misuse.

One result of this policy has been a flood of drugs on to the black market as misusers sell them, either to gain money to buy the drugs they really want or to convert them into a regular weekly income. Drugs, originally prescribed legally, are now readily available in shopping centres, school playgrounds, and pubs; adults actively look for new children to supply so that the market is continually expanding. The money to pay for these drugs is nearly always raised by crime.

Whether or not long term maintenance policies can be justified, those who operate them have a strong obligation to see that as few drugs as possible leak into the rest of the community. Work showing that maintenance policies benefit the recipients is of little value if many more people are drawn into drug misuse as a result.

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Farmer's hip

EDITOR,—I was interested in Peter Croft and colleagues' finding that farmers are at increased risk of osteoarthritis of the hip[1] because, like others,[2-5] I have observed an increased incidence of hip replacement among my patients who are or have been farmworkers (table).

The controls were men of the same age (to the nearest five years) as the farmers who were on my practice list. Their occupation varied but was not necessarily sedentary: they were local government employees, teachers, shop assistants, police, gamekeepers, and builders. Those aged over 65 were retired businessmen and had previously lived outside Ryedale. Most had not engaged in manual jobs requiring the lifting of heavy loads.

The prevalence of hip replacement among men who had farmed for more than 10 years was 14 times and eight times greater than that in controls for those aged over 50 and over 60 respectively. These figures are similar to those found in moorland Staffordshire and lowland Cheshire.[1]

Croft and colleagues say that a question as yet unanswered is whether risks relate particularly to

Details of 12 cases of chest symptoms after administration of sumatriptan reported to Netherlands Centre for Monitoring of Adverse Reactions to Drugs

Case No	Age and sex	Dose and route	Latent period (min)*	Symptoms notified by reporting doctor	Recurrence of symptoms after rechallenge†	Other drugs
1	36, F	100 mg orally	30-45	Substernal pressure and discomfort, drowsiness, "shaky"	ND	Terfenadine 60 mg
2	38, F	100 mg orally	30	Substernal pressure and pressure in shoulders and neck	>10 times	Carbamazepine 600 mg, lactulose, hydroquinone hydrobromide dihydrate 100 mg
3	61, F	100 mg orally	About 30	Anginal pain radiating to left arm	2 times	Isosorbide dinitrate 5 mg
4	46, F	100 mg orally	15	Substernal pressure and chest pain, sweating	3 times	Oral contraceptive
5	53, M	100 mg orally	About 30	Anginal pain	ND	None
6	44, M	100 mg orally	30	Substernal chest pain, palpitations, pain in throat	ND	None
7	27, F	100 mg orally	30	Substernal chest pain, malaise, paraesthesia, heaviness of arms	ND	None
8	33, F	6 mg subcutaneously	1-5	Angina pectoris radiating to left arm and hand, dyspnoea	2 times	Propranalol 60 mg
9	45, F	100 mg orally	Same day	Substernal pressure, muscle stiffness	ND	None
10	19, F	6 mg subcutaneously	30-60	Chest pain, dyspnoea, nausea	ND	Oral contraceptive
11	50, F	6 mg subcutaneously	1-5	Anginal pain radiating to the jaw, hypertension	ND	Clonidine 100 μg, aspirin 600 mg
12	36, F	100 mg orally	30	Substernal chest tightness	ND	None

*Between first intake and onset of symptoms as notified by reporting doctor. †ND=Rechallenge not done.