The alternative to opiates is Entonox, but in my experience this has never been as effective.

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Effect of beta-blockers on arrhythmias

SIR,—In their report Dr J M Roland and others (1 September, p 518) conclude that beta-blockers have no effect in preventing "serious" arrhythmias in the acute phase after myocardial infarction. It is possible that their results may have been affected by the following factors.

Firstly, the recordings were obtained quite late after chest pain (mean of 17 hours in placebo and atenolol-treated groups and 18 hours in the propranolol-treated group). The incidence and frequency of serious arrhythmias is considerably lower than in the earlier hours.¹ It is possible that in patients in whom recordings were obtained earlier (say under 12 hours after chest pain) there is a greater chance of showing a benefit. This may have been missed by the late onset of tape recording.

Secondly, the use of oral beta-blockers may have led to inadequate blood levels in the first 24 hours. This has been shown to occur with propranolol² and we have seen this with atenolol (unpublished data). Therefore the use of oral preparations would have further decreased the chances of showing a benefit. This is important, as tape recording may well have finished before effective beta-blockade was achieved in some patients. The delay in adequate beta-blockade would increase still further in the atenolol group, where a third of the patients would have received placebo as the first tablet.

The authors report a significant fall in maximum heart rate over the first 24 hours as evidence of adequate beta-blockade. This shows that beta-blockade was achieved some time over the first 24 hours. It would be more relevant to know the time of onset of a "significant bradycardia."

The authors state that serious arrhythmias occurred in similar numbers of patients in both groups. They do not state whether the frequency of these arrhythmias in each patient was less on treatment or on placebo. We agree that it may prove impracticable to analyse episodes, "as the results can be too heavily biased by individual patients" if standard *t* tests are used. However, this may be overcome by using simple non-parametric tests such as Wilcoxon's rank test or by using the log values prior to using standard *t* tests.⁴

A recent paper³ has demonstrated that earlier intravenous administration of acebutolol (6 hours after the first rise in creatinine phosphokinase) decreased ventricular arrhythmias and is at variance with the study of Dr Roland and his colleagues. Although this may be due to the different beta-blocker used, it is highly likely that the difference in methodology is responsible.

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¹ Adgey, A A J, *et al*, *Lancet*, 1971, 2, 501.

² Rutherford, J D, *et al*, *Clinical and Experimental Pharmacology and Physiology*, 1976, 3, 297.

³ Ahumada, G G, *et al*, *British Heart Journal*, 1979, 41, 654.

Large-bowel obstruction caused by cancer

SIR,—Mr L P Fielding and others (1 September, p 515) are right to keep alive the unresolved and controversial matter of the best treatment for large-bowel carcinoma presenting with obstruction. Their figures bear out what has always been suspected, that the skill and experience of the surgeon and the conditions under which he operates are of undoubted significance. Until a larger series has been collected and a more generally accepted definition of "acute obstruction" found results cannot be evaluated.

What is of special interest is that primary resection *without* primary anastomosis, a procedure advocated by me as long ago as 1960,¹ is now being practised. This small series of 24 cases so treated carried a 33% mortality. If this third option were more widely practised it is possible that the figure would improve.

In view of the great number of variables involved, a larger number of cases must be studied, thus enabling comparison to be made not only between the classical staged procedure and primary resection with immediate anastomosis but also primary resection with delayed anastomosis.

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¹ Sames, C P, *Lancet*, 1960, 2, 948.

SIR,—I read the article on the management of large-bowel obstruction caused by cancer by Mr L P Fielding and colleagues with interest (1 September, p 515). However, I do not feel that their conclusions are valid on the data presented in this article. Surely it is necessary to define the causes of mortality after surgery before concluding that the surgeon, trained or otherwise, is responsible for that mortality.

With respect to the arguments for and against primary tumour resection, I think most surgeons would agree that the tumour should be excised if this can be done without increasing the risk to the patient. This need not involve an immediate anastomosis, and I would be interested to know how many of the 19 deaths in the primary resection group were due to anastomotic leakage.

The 31 patients treated by staged resection fared well, with only two postoperative deaths. Of the remaining 16 patients in this group, 11 died and presumably several if not most of those died of causes unrelated to their surgical or anaesthetic management. It is my contention that such deaths would probably have occurred irrespective of the type of surgery undertaken.

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*. We sent a copy of this letter to Mr Fielding, whose reply is printed below.—ED, BMJ.

SIR,—Thank you for allowing us to comment on Mr C G Marks's letter; we have tried to answer the five points raised.

The problem of who is "responsible" for the death of any patient, particularly in a group of patients who are often seriously ill, is usually a difficult and sometimes a sensitive issue. The

suggestion that to identify a cause of death is to be able to apportion responsibility for that death may be attractive, but is too simplistic a viewpoint and is not supported by our experience of the outcome of mortality conferences, especially in the context of complex patient management. The causes of death in our study are set out in the accompanying table.

Causes of death after primary and staged resections for large-bowel cancer

Cause of death	Primary resection group (n = 90)	Staged resection group (n = 47)
Sepsis	8	5
Cardiorespiratory .. .	3	3
Bronchopneumonia .. .	2	2
Pulmonary embolism .. .	1	2
Carcinomatosis .. .	2	1
Cerebrovascular accident .. .	2	0
Burst abdomen .. .	1	0
Total	19	13

Mr Marks's suggestion that most surgeons agree that immediate resection for obstruction is the treatment of choice is, sadly, not our conclusion. On the contrary, it is clear that primary resection for obstructing left-sided tumours remains controversial that colostomy being said to be simple and safe in sick patients even for the inexperienced surgeon. Our data suggest—only suggest—that this is not the case.

Anastomotic leakage is often difficult to define, especially when postoperative radiological investigation is omitted. In the primary resection group one-third of the deaths were probably attributable to anastomotic leakage.

Of the 16 patients in the staged resection group who in fact had only a stoma, 11 died; only three of these deaths (two pulmonary emboli, one bronchopneumonia) could be said not to have been a direct consequence of the surgical or anaesthetic management. Furthermore, one could argue that all 18 (38%) of these patients (13 deaths and five no resection) had "failed treatment": a statistically higher rate than in the primary resection group (19/90, 21%; $P < 0.05$).

Although it is Mr Marks's contention that these deaths would have occurred anyway, it seems to us common sense that the greater experience of a senior surgeon would be of benefit both to these patients, who are frequently ill, and (dare we say it?) to the instruction of the surgeon in training. If this is not the case what is the meaning or utility of "clinical experience"?

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Fetal malnutrition—the price of upright posture?

SIR,—I was very interested in the hypothesis advanced by Dr André Briand (4 August, p 317) and my recent experience in a developing country is relevant.

I am currently employed by a mining company in Zaire as a general practitioner with the responsibility for health and welfare of the whole local population of approximately 60 000. The local community consists largely of subsistence farmers growing mainly rice, cassava, and groundnuts. There is virtually no

modern means of production and women of childbearing age make a major contribution to the agricultural labour force.

Early this century a system of a "lying-in village" was introduced, where women live with a female relative during the last weeks of pregnancy. Here they have no agricultural duties and only the lightest of domestic chores. Even firewood is provided. Food is brought mainly by the family and a small supplement of rice with palm oil is given out by the medical service. This system is well accepted and about 30% of pregnant women, particularly those from far-away villages, come to the lying-in village without any encouragement.

Birth records (June 1978 to May 1979) show a strikingly lower stillbirth rate in women who used the lying-in village (1.9%) than in those who did not (4.2%)—a significant difference ($\chi^2=5.8$, $P<0.05$). The standard of antenatal care for both groups was similar. Antimalarial prophylaxis (25 mg pyrimethamine every fortnight) was administered to women in both groups who attended antenatal clinics. Iron and folic acid tablets were given in cases of clinical anaemia.

We appreciate that these results do not fully substantiate Dr Briend's assertion that rest in itself improves nutrition during fetal life. The sampling in our study may have been biased by some unknown factors and further observations may be needed. Factors other than improved placental perfusion may have contributed towards the difference in the stillbirth rate. For example, the nutritional state of the women using the lying-in village may have been improved by the reduction in their energy expenditure. We do not know if reduced sexual contacts during the last few weeks of pregnancy might have an effect on the risk of infection of the amniotic fluid.

However, our data support Dr Briend's suggestion that rest at the end of pregnancy could be an important measure in reducing perinatal mortality in developing countries and merits further investigation. If villagers themselves were to adopt this arrangement for women during the last weeks of pregnancy, it could constitute a cheap and beneficial public health measure.

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Nutritional deficiencies in schoolchildren

SIR,—Recent surveys in the West Country confirm the deficiencies recorded by Professor A E Bender (22 September, p 732), but in them I have found other important deficiencies. Modern nutrition knowledge has as yet not influenced the food provided for schoolchildren.

Inspecting school meals with catering officers, I found that dietary fibre is obviously deficient, as white flour, white bread, excessive sugar, and "sloppy" sweet puddings are almost universally provided and eaten.

There is little folic acid or vitamin C in most school meals. In a school visited last week, potatoes were available only as chips, brought into the school in plastic bags, prepared and "blanched" a day or more previously by a "wholesaler." In another school cabbages were four days old, cooked for half an hour, and eaten two hours later. With 500 meals, where a

choice was given, 12 salads and eight oranges were available. Vitamins of the B group were also obviously low or absent.

In a recent publication¹ evidence is presented of widespread nutritional deficiencies, especially in hospital patients and in institutions. Surveys at St Thomas's Hospital² show that the million or so children receiving free school meals, mainly in classes IV and V, are significantly shorter than other children and "of lower nutritional status," and that withdrawal of school meals "might well prejudice their future development."

Before any decision is taken on school meals, I suggest that a detailed analysis should be made of the nutrient content of a random sample of school meals, with blood analyses of the levels of vitamin C and the B group vitamins in children eating the meals.

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¹ Taylor, T G (editor), *The Importance of Vitamins to Human Health*. Lancaster, MTP Press, 1979.

² Rona, R J, Chinn, S, and Smith, A M, *Lancet*, 1979, 2, 534.

Aetiology of appendicitis

SIR,—I am a very busy doctor working (until recently single-handed) in a 120-bed general hospital. It is situated in one of the most rural areas of Nigeria, and I have only just seen the reply from Drs A R P Walker and I Segal (19 May, p 1352) to the letter by Mr F T de Dombal and Dr A H Hedley (24 March, p 820).

Having been working for 29 years in the area I find Mr D P Burkitt's assertion and the comments on it very interesting (3 March, p 620). In my first 10 years here any appendicectomy I performed was because I found the appendix in a sliding hernia, which is common in the area. In the same period I had two cases of appendix abscess. But in the last five years appendicitis and ruptured appendix have become the most common cause of emergency operation in this hospital, which serves an area with a population of 98 000. I see an average of 2000-3000 children under 18 years a month—last month 3815. Schoolchildren, college students, and student nurses form the bulk of the patients seen. Some for various reasons refuse operations or promise to go for their parents, disappear into the villages, and are lost to me. But the figures for those coming to surgery in the last five years are: 1975-5; 1976-9; 1977-11; 1978-13; 1979 (eight months)-7.

Thirty years ago the population consisted of simple natives on subsistence agriculture. Unable to afford protein, their food consisted of carbohydrates—yams, cassava, and cocoyam, all making heavy meals rich in roughage. Years back, all a schoolchild had for breakfast before going to school was a roasted yam but today as he runs along he is tearing at a refined loaf of bread. For student nurses Pepsodent and Maclean toothpaste have replaced the ever-ready gum and tooth-hardening chewing sticks just as Quaker Oats and a glass of milk have replaced the roasted yam at breakfast. In increasing incidence dental caries, dyschezia, haemorrhoids, and many colonic disorders of the colon are running neck and neck with appendicitis.

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Is appendicitis familial?

SIR,—Dr N Andersson and his colleagues (22 September, p 697) report a strong family history of appendicectomy in the relatives of children undergoing this operation. As part of a larger study of psychosocial variables and appendicectomy in adults aged between 17 and 30 years, I have asked the same question.

A preliminary analysis reveals that, of 55 subjects with histologically proved appendicitis, 28 had a history of appendicectomy in a first-degree relative. This compares with 13 out of a control group of 50 randomly selected from the community (after exclusion of four people who had themselves had appendicitis). In the case of affected siblings, 15 of the 55 in the appendicectomy group and three of the 50 in the control group had such a history. Both differences are unlikely to have occurred by chance ($P<0.01$). On two occasions I have discovered at a follow-up interview that a brother of my subject was undergoing appendicectomy almost exactly one year after the index operation. I have also found a strong family history in those whose appendix was found to be normal.

Further analysis, when my data are complete, will search for any relationship between histological diagnosis, family history, and any other variables (such as age and sex) that will help in the interpretation of these findings.

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Treatment of adder bites

SIR,—A small number of cases of adder bite occur every year in this country. The mortality is low but the morbidity quite considerable.^{1,2} Despite the availability of highly refined Zagreb antivenom and an excellent review article,² standard guidelines in many casualty departments and poison centres and in the current *British National Formulary* repeat the misleading statement "the bite is less dangerous than the antiserum." The following recent case illustrates the danger of continuing this policy.

On 29 July a 14-year-old boy picked up an adder, mistaking it for a grass snake, and was bitten on the right wrist. His sister immediately applied a tourniquet and he was brought to the accident and emergency department of this hospital, arriving about 45 minutes after the bite. On arrival he appeared generally well, though he had vomited once before arrival. His right forearm and hand were swollen, blue, and cold. Because of the standard instructions he was not given antivenom but was treated with tetanus toxoid, hydrocortisone, and chlorpheniramide and was admitted for observation.

The boy remained well for the next 36 hours, although the oedema extended to involve his upper arm. During the early hours of 31 July he vomited twice, developed a tachycardia, and became hypotensive. Investigations showed a blood urea concentration of 19.5 mmol/l (117.5 mg/100 ml), creatinine phosphokinase 930 IU/l, white blood cells $17.4 \times 10^9/l$. Although his oral fluid intake had been satisfactory until this time it was felt that he had lost a considerable amount of fluid into the tissues with consequent dehydration, and intravenous saline was started—with some improvement, though he remained ill. At this stage I consulted Dr H A Reid of the Liverpool School of Tropical Medicine, who advised giving antivenom, the rationale being that venom can be released gradually into the systemic circulation and produce relatively late toxic effects. The antivenom was certainly well tolerated and the patient's