There are, however, additional factors to be considered in interpreting the findings of Dr Hillbom and coworkers.

The table indicates that 13 patients had "fatty liver" and that another 13 patients had "normal liver." Only 16 patients, however, had biopsy specimens taken (three normal and 13 showing fatty liver). Therefore the definition for 10 of the 13 patients with "normal liver" was not histologically proved but seemed to be based on serum transaminase activities less than four times higher than an unknown normal value. This classification has disadvantages as most patients with fatty liver have serum transaminase activities well below that limit.[3] In a study carried out in this department serum asparatate transaminase activities (mean (SD) upper limit of reference range: 25 U/l) were: 28 (6); 31 (14); and 35 (7) U/l in patients with normal liver histology (n=7), mild to moderate fatty change (n=21), and severe fatty change (n=5) respectively (S B Rosalki, personal communication). Clearly, the considerable overlap indicates that histology remains the only acceptable form of classification. The only conclusion entirely based on histology (correlation between bleeding time and fatty infiltration) was of borderline significance and included only three patients with normal histological findings.

Abnormal platelet function in alcoholics tends to correct itself after one to two weeks' abstinence,[1] yet it is unlikely that histological appearances of the liver change considerably during this time. Therefore, a similar degree of fatty infiltration is associated with abnormal or "normal" platelet function, depending on sampling time.

Hillbom *et al* report only platelet aggregation induced by adenosine diphosphate, and others, as well as ourselves, have shown (in in vitro and ex vivo studies with patients or volunteers) that this aggregation is fairly insensitive to the effects of ethanol.[1 4-6]

Non-parametric statistical analysis of the results seems indicated as the standard deviations are often considerable (one actually exceeds the mean). It is also interesting that in primary biliary cirrhosis impaired platelet function was mainly related to thrombocytopenia and not to diminished aggregability.[7] Others have gone as far as to state that "significant platelet dysfunction is generally not observed in patients with stable chronic liver disease."[8]

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Effect of growth hormone on short normal children

SIR,—The study of Drs P C Hindmarsh and C G D Brook (5 September, p 573) provides food for thought but begs a few questions. How do they define "short normal"? Have they included children who were small for gestational age or children of small parents, or children suffering from socioeconomic deprivation? Some further clinical information would provide useful rumination. Further, what are their ultimate objectives (other than a wish to study the effects of biosynthetic human growth hormone)? What was the cost in £/cm height gain? Could, for example, dietary supplementation have produced a similar 2 cm growth increment?

Which of us, by taking thought, would not wish to add a few centimetres to children's height? But can we afford the clamour, the dolour (300 subcutaneous injections a year), and cost of such a campaign if applied to some 3% of the child population, who, by definition are "short normal"?

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SIR,—The very careful study by Drs P C Hindmarsh and C D G Brook on the response of normal short children to growth hormone made fascinating reading, although the effects of therapeutically administered growth hormone on normal children might perhaps have been predicted from the rare experiments of nature—gigantism—where supraphysiological amounts of growth hormone are present before puberty.

Of possibly more fundamental interest, however, is the philosophy of using an excess of a systemically active product to increase the height of normal children and the direction that such research might take. While Drs Hindmarsh's and Brook's interest may be confined to the academic investigation of growth hormone's actions in normal children, the interest of the general public, when it learns of their work, will almost certainly extend to demands for treatment on behalf of their short children. The public will already have read a full page article on the authors' work ("Getting High on Hormones"[1]), which seriously confuses the issue of normal and abnormal smallness. Short children undoubtedly suffer socially and scholastically, but shortness is relative and, however great the demand for growth hormone, a third centile will never disappear and the children below it will continue to regard themselves, and be regarded medically, as short.

Because the response to growth hormone does not influence the genes for height the implications for the child of normal small parents whose heights were "normalised" by the use of growth hormone in their own childhood are doubly serious, as they will not only be genetically small but will be subject to an artificial correction factor for parental height. It will be interesting to see data of similarly high quality on the response of the sizable group of short children with reduced growth velocity for ill defined reasons—that is, data on children who are failing to achieve their genetic potential rather than those who may effectively be seeking to exceed it.

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AUTHORS' REPLY,—The comments of your correspondents were predictable. We can assure Professor Gill that, as far as possible, we excluded children who were small for gestational age or children who suffered socioeconomic deprivation. We of course included children with small parents. The object of the study was to anticipate questions which parents will ask of their doctors. The profession must be in a position to provide an answer to a parent who asks whether growth hormone will make their child taller. We submit that the science of medicine demands studies of the calibre of ours. We can assure Dr Wilkin that we are pursuing the question of the wider uses of growth hormone in the groups of children he outlines.

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Ruptured abdominal aortic aneurysm presenting as ureteric colic

SIR,—We have been interested by the continuing correspondence relating to the presentation of leaking abdominal aortic aneurysm and, in particular, the confusion between abdominal aortic aneurysm and renal colic (Mr C G Moran and colleagues, 16 May, p 1279; Dr J B Roussak and Mr E R C T Owen, 25 July, p 267; Mr M J Stower and colleagues, 12 September, p 670).

Records available to us in Leeds, including those of the World Organisation of Gastroenterology's acute abdominal pain international survey,[1] show 32 cases in which leaking abdominal aortic aneurysm caused the patients to present to hospital with undiagnosed acute abdominal pain.

The table shows the initial diagnosis made in each of the 32 patients. Our data partly support the comments of others, in that there were three cases in which renal colic was the initial (erroneous) diagnosis. This, however, is not the whole study as only two fifths of patients had correct diagnoses made when first seen. A considerable variety of incorrect diagnoses were made, and in seven cases out of 32 no diagnosis at all was made by the doctor who saw the patient.

Your correspondents have thus performed a valuable service in alerting the clinician to the possibility of leaking aortic aneurysm in patients over the age of 50 with acute abdominal pain. The problem is, however, more widespread than might be imagined. In 2406 cases of acute abdominal pain in patients over the age of 50 the proportion with vascular problems (myocardial infarct, mesenteric occlusion, or leaking aneurysm) was 2%, and among those over 70 it was almost 10%. Furthermore, and no less importantly, a similar proportion had occult cancer.[1 2]

Initial clinical diagnoses made in 32 patients presenting to hospital with acute undiagnosed abdominal pain (excluding patients presenting with vascular catastrophe)

Initial diagnosis	No (%) patients
Aneurysm	13 (41)
Renal colic	3 (9)
Perforated peptic ulcer	2 (6)
Infarct, obstruction, cancer cholecystitis, pancreatitis, NSAP,* diverticular disease	1 (3) each
No diagnosis made	7 (22)
Total	32 (100)

*NSAP is non-specific (non-surgical) abdominal pain, where symptoms rapidly resolve and no specific cause (particularly no cause warranting surgery) is found.

The key to diagnosis in such circumstances seems to be radiological as 90% of this series of patients had the diagnosis correctly established after investigation. In this respect, the lateral decubitus film is particularly helpful.

The risk to life in acute abdominal pain is related to age, rising from well under 1% in the under 50s to 2% in patients in their 60s, 5% in those in their 70s, and 7% in the over 80s. We thus support the comments of previous correspondents but suggest a wider conclusion. All elderly patients with acute abdominal pain must be assessed with particular care (both neoplastic and vascular causes being routinely considered). Otherwise, even the alert may be misled.

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Bone turnover and trabecular plate survival after artificial menopause

SIR,—The measurement of bone matrix proteins in plasma or urine may answer Dr J Reeve's call for simple ways to identify perimenopausal women at risk of osteoporosis (26 September, p 757).

The report that combinations of serum alkaline phosphatase, urinary hydroxyproline, and urinary calcium have been shown to differentiate between fast and slow bone losers is exciting, but these measurements are not specific for bone turnover.[1] Recent advances in bone matrix protein biochemistry may, however, offer ways of improving specificity and sensitivity.[2]

Osteocalcin (bone Gla-protein) is released from activated osteoblasts, and serum concentrations predict the histological bone profile in postmenopausal women.[3] As yet there is not a corresponding marker of bone destruction, but estimations of urinary deoxypyridinoline may measure bone specific collagen breakdown.[4] Preliminary work has shown that postmenopausal women may be classified according to the degree of bone formation measured by serum osteocalcin, suggesting fundamental differences in bone turnover between patients.[3]

Prospective clinical studies of these new measures are needed to determine whether identification of patients with high or low bone turnovers before the menopause will help to predict increased risk of developing osteoporosis.

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Is birth weight determined genetically?

SIR,—We agree with Dr Roy Carr-Hill and colleagues (19 September, p 687) that elucidating the role of genetics in determining birth weight poses considerable conceptual and methodological problems. We are puzzled, however, by certain aspects of their study.

Their basic hypothesis, which is not clearly stated, is presumably that if genetic factors are important then a close correlation will be seen between the birth weights of mother-child pairs (who share 50% of their genetic material) once confounding environmental factors are taken into account. The confounders considered by Dr Carr-Hill and coworkers were maternal height, gestational age, and proteinuric pre-eclampsia, with fetal sex, maternal age, and parity controlled for essentially by selection.

The correlation between generations observed for birth weight was 0·215, which reduces to 0·154 after adjustment. Similar reductions are observed for both the slightly higher mother-daughter correlations and the slightly lower mother-son values. From the low adjusted correlations Dr Carr-Hill and colleagues conclude that genetic factors have only a minor influence on birth weight.

We have two main concerns about this study. Firstly, it is not clear that the factors taken into account are solely environmental. Specifically, it is inappropriate to control for maternal height in this context as it must at least in part reflect fetal genetic composition. Secondly, notwithstanding this potential overcontrolling, the overall effect of the adjustment for confounders is very small, particularly in comparison with sampling error. The emphasis in the paper's abstract on the adjusted correlations therefore masks the fact that the raw correlations are much lower than those found in other studies, the reasons for which are not at all clear from the paper.

In conclusion, though we agree with Dr Carr-Hill and coworkers about the need for due consideration to be given to confounding effects, their efforts have been severely compromised by overcontrol, a recurrent problem in epidemiological research.

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Commercialisation of medical education

SIR,—Professor M D Vickers rightly raises an important issue (26 September, p 785), which had also occurred to me.

I attended the recent *British Journal of Hospital Medicine*'s conference on intensive care with mixed feelings. On the one hand, it was an opportunity to participate, free and with little effort, in a very attractive, though exceedingly expensive, international scientific event and meet some of my distinguished colleagues and friends from abroad. On the other hand, as Professor Vickers points out, the meeting had a distasteful commercial basis, which I personally found especially disturbing as the Federation of Societies of Intensive and Critical Care Medicine, of which I am president, is desperately short of funds. The organisers also made certain unjustified claims and unacceptable announcements. In general, at least part of the profit from scientific meetings is returned to the relevant scientific body and is a valuable source of funds. I was informed that the primary purpose of this meeting was not direct profit but the promotion of the journal itself, indirect profit; certainly an unusual and doubly rewarding way of enhancing the subscription list of what is claimed to be an ethical scientific journal.

While I enjoyed the symposium I would, on reflection, not attend such a meeting again. Incidentally, I can assure Professor Vickers that I personally received no fee and claimed no expenses. One of my colleagues believed that the register of participants showed that about half were nurses, an interesting observation. Can British nurses get funds more easily than doctors?

The commercial exploitation of communication in medicine and science, indeed of learning in general, is not new. But while few contributors become rich through books and journals literature is indispensable, and publishers provide a service we cannot in a relationship normally as close to symbiosis as we can hope for. Unfortunately there are already too many interested commercial parties in the massive and highly lucrative international business of conference organisation, and this event has shown we can lose our legitimate share of such profit. The United States has a well established, and invaluable, system of credits, which, inter alia, demands the fulfilment of certain criteria for official recognition of scientific meetings. Should we, and other developed countries, consider such a system for our own protection? It could prove a powerful defence weapon in the face of a perceived threat to our legitimate professional interests. What about an ad hoc committee from the specialty of anaesthesia to begin with?

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SIR,—I would like to rise to the bait put out by my friend Professor M D Vickers in his letter on the commercialisation of medical education (26 September, p 985).

Parenthetically, readers may need to be reminded that Professor Vickers is a coauthor of more than one educational textbook, written no doubt with the highest ideals, but with commercial gain as a secondary and unfortunate byproduct.

With respect to the main points in his letter, it might be considered unfortunate that revenue escapes from the National Health Service. On the other hand, as a consumer of the product on three occasions, and not as a speaker, I would say that I buy a product only at a price that I think desirable. This must go on for the several hundred other people who have attended these meetings. Perhaps what the association and faculty might learn from the situation is that a good programme, with appropriate speakers on a subject of wide interest, is more saleable than some (but not all) of the stodgy programmes put out by the said organisations. Perhaps they might also consider whether the "purchasers" of postgraduate education always want to hear the same people (often academics and non-clinical) talking incessantly, often on clinical but disparate subjects, when they lack clinical credibility?

In a country where free market forces operate a product that offers the best value for money will always be purchased by the consumer, whether that product is a meeting or textbook. If any profit may be considered to be commercial is there any difference between that profit going to a publisher or to an individual author?

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Reducing late abortions

SIR,—The Birth Control Trust held a conference recently, which was reported on in Medical News (26 September, p 787). Unfortunately, the information in the first paragraph is seriously in error. Only 0·8% of all abortions performed for women