paper focused on discrimination in its widest sense and the hypothesis that this might explain differences in outcome. Thus we looked at two groups that had different risks of discrimination: people of Caribbean origin and white British people. Evidence suggests that Irish people are discriminated against in the United Kingdom,[3] so we excluded them. We were interested in culture only as it might affect discrimination, which is consistent with our statement that we aimed to demarcate a white group that was "as culturally homogeneous...as possible to decrease difficulties in interpreting results."

Azuonye highlights the problem of how to use data on the minority of patients of Caribbean origin who are not of African ancestry. Because we did not know what level of discrimination they would suffer and no systematic evidence exists, we set our criteria to include such patients. We were unable to assess this question as our sample contained only people of African ancestry.

The paper in the *Psychiatric Bulletin* to which Azuonye refers relates to culture, not discrimination.[4] It was aimed at helping researchers to choose the most appropriate variables when comparing ethnic groups. We believe that by using populations at differing levels of risk we chose the most appropriate variables.

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Performance of neonatal screening programme must be monitored

EDITOR,—We agree with Francesco P Cappuccio and colleagues that monitoring the performance of the neonatal screening programme for phenylketonuria and congenital hypothyroidism is difficult at district level,[1] but our experience in the Northern region shows that it is not impossible. While neonatal screening is complex and involves many health professionals—midwives, health visitors, laboratory staff, consultants with responsibility for neonatal screening, child health officers, and the directors of the neonatal screening service—close communication between these groups can overcome difficulties.

We audited the neonatal screening programme in the Northern region in 1993.[2] We found initially that only six of the 16 districts had a timely, failsafe mechanism in place for ensuring that all babies were screened. Five other districts had mechanisms to ensure that babies were screened, but this was not always timely. After the audit all districts except two had a timely, failsafe mechanism for identifying babies who had not been screened. Those districts where the system worked well were those in which a consultant (usually a community paediatrician) took a lead in coordinating and monitoring it and in which a working group of all the professionals involved met regularly to discuss problems. In a circular on screening to detect phenylketonuria issued in 1969 responsibility for the screening programme in each district was clearly stated to lie with the medical officer of health[3]; the current equivalent is the director of public health. Directors of public health have an important role in ensuring the provision of a high quality screening programme that includes monitoring arrangements, either through direct involvement or by clearly delegating responsibility to another consultant.

We agree that the neonatal screening service must continue to be provided regionally and not be fragmented. In addition, the overview of the service must be maintained centrally by the Department of Health so that any further screening programmes—for example, for cystic fibrosis—can be coordinated throughout Britain.

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Postoperative deep vein thrombosis and surgery for varicose veins

EDITOR,—In his critical editorial comment Bruce Campbell discusses the possible links between thrombosis, phlebitis, and varicose veins.[1] While pointing out the evidence for an association between varicose veins and deep vein thrombosis in patients undergoing abdominal or pelvic surgery, he questions the need for a thromboembolic prophylaxis in patients undergoing surgery on their varicose veins.

Admittedly, reliable data on the incidence of deep vein thrombosis after varicose vein surgery are not available from controlled, randomised studies. However, we reported a few years ago on the incidence of pulmonary embolism in a large retrospective series of 19 161 patients who were operated on over a 10 year period (1980-9) in our clinic of digestive surgery.[2] Among the 1063 patients who underwent stripping of varicose veins in that population, four presented with clinically manifest and objectively confirmed pulmonary embolism during hospital stay, with two additional events occurring during the four weeks after discharge from hospital, giving an overall incidence of postoperative pulmonary embolism after this type of surgery of 0.56% (95% confidence interval 0.21 to 1.23), which was similar to the incidences of 0.40% (0.22 to 0.66)that was observed after biliary surgery and 0.60% (0.22 to 1.30) after laparotomy.

On the basis of these data and the established increased risk of deep vein thrombosis at the time of abdominal or pelvic surgery in patients with varicose veins, we believe that patients undergoing surgery for varicose veins are likely to be at special risk of developing postoperative venous thromboembolism and should thus receive prophylaxis until especially designed trials will confirm or negate this need.

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Postoperative pulmonary complications

Pain relief improves respiratory function

EDITOR,—John C Hall and colleagues addressed the important issue of postoperative complications after abdominal surgery.[1] However, their evidence does not support their conclusion; in the light of relevant recent literature, we would like to offer some explanation.

Major changes in respiratory function occur in all patients after anaesthesia and surgical incisions, especially on the thorax and upper abdomen, because of a decrease in the functional residual capacity with minimal change in the closing volume leading to airway closure during tidal breathing.[2] These changes are most exaggerated in elderly patients, obese patients, smokers, and those with pre-existing cardiopulmonary disease. These changes in pulmonary mechanics are not as great after surgery of the lower abdominal cavity.[2] Several randomised studies have shown that postoperative pulmonary complications correlate positively with the decrease in ventilatory efficiency.[2 3] Hall and colleagues, by combining upper and lower abdominal surgical patients, have diluted the impact of physiotherapy and the use of incentive spirometer on the prevention of postoperative complications.

The authors also suggest that the postoperative decline in respiratory functions is obligatory and is not reversible by effective analgesia. The most important cause of regional impairment of ventilation, ineffective cough, and impaired ability to sigh and to breathe deeply is incisional pain.[2 3] Considerable evidence has accumulated since the early 1980s confirming that these changes can be minimised and even prevented by effective analgesic techniques, especially if analgesia is started preemptively and continued postoperatively until wound healing has taken place.[3 4] Overreliance on systemic opiates rather than on regional analgesic techniques (only 31% in this study) contributed to the study's less than optimal result.

We are intrigued by the term "floppy" diaphragm. The pathophysiology of postoperative diaphragmatic dysfunction after abdominal surgery is complex and multifactorial. A reduction of intrinsic diaphragmatic contractile properties is not the predominant factor.[5] Reflex inhibition of phrenic nerve activity, particularly after upper abdominal surgery, is the most attractive explanation.[5] These changes might be expected to be considerable after upper abdominal surgery, but they are probably of little consequence after appendicectomy. Combining all surgical groups is therefore unhelpful.

We contend that high quality pain relief, centred around optimal afferent blockade,

greatly improves pulmonary function; subsequently, effective physiotherapy and incentive spirometry have their parts to play.

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Obesity, pain, and sedation are important

EDITOR,—John C Hall and colleagues stratify their patients as high risk only on the basis of American Society of Anesthesia grade (>1) and advanced age (>60 years) and then imply that other putative risk factors are similar in the two (high and low risk) groups. They seem to ignore one major risk factor for the development of postoperative respiratory complication—namely, obesity. Although the two groups are apparently well matched with respect to most criteria of comorbidity, no mention is made of the two groups' body mass indices. If populations are not weight matched it is impossible to make a valid comparison.[2]

In addition, Hall and colleagues' treatment of the role of postoperative analgesia in the development of pulmonary sequelae is superficial. Simply to classify the mode of pain relief as epidural or narcotic dosage is inadequate: what is more important is the quality of the analgesia delivered.[3] In the context of postoperative pain relief, the narcotic dosage alone is a meaningless concept.[4] Visual analogue scores are the optimal technique for assessing pain and can readily be used at the bedside. The quality of analgesia is of the utmost importance in this study. If patients' pain was inadequately relieved it is difficult to see how they could comply fully with physiotherapy, deep breathing, or incentive spirometry.

Similarly, although the authors refer to the importance of postoperative somnolence in the development of basal atelectasis and subsequent infection, this does not seem to have been assessed. This is a pity, since simple and reliable sedation scoring systems are available. It is of concern that sedation was not recorded in patients receiving epidural or narcotic infusions.[5]

In conclusion, we do not know whether the methods of prophylaxis against respiratory complications are equivalent. The results may be similar because one group was too fat, too sedated, or in too much pain to breathe deeply or comply with physiotherapy and so clear the secretions whose retention predisposes to chest infection.

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Adequate pain relief is also necessary

EDITOR,—John C Hall and colleagues draw our attention to some of the ways of preventing postoperative pulmonary complications.[1] Pain and trauma of surgical procedures, particularly upper abdominal operations, leads to splinting of the ribs and diaphragm, which in turn leads to further collapse of basal lung units. It has generally been realised that effective analgesia improves patient cooperation during physiotherapy, enabling deeper breathing and better coughing, thus minimising the sputum retention.[2] In Hall and colleagues' study, only 20% of the high risk patients received epidural analgesia, and another 40% received opioid infusion during the perioperative period. Only 80% of the patients had documentation of pain relief after laparotomy, and little reference was made to the quality of postoperative analgesia.

Thoracic epidural analgesia is known to reduce the respiratory complications after repair of abdominal aortic aneurysm. Its routine use has been shown to reduce the incidence of respiratory complications from 30% to 13% during oesophagegastrectomy.[3] Intravenous opioid infusion also provides good pain relief but sometimes is ineffective during movement and physiotherapy. Effective epidural analgesia has the advantage of enabling easier ambulation and avoiding the depressive effects of analgesics and sedatives. Early ambulation also encourages better distribution of air in the lungs. In comparing the efficacy of incentive spirometry with deep breathing exercises, the authors could have adopted a policy of providing uniform analgesia. It would have been useful to have analysed the incidence of respiratory complications in the patients receiving epidural analgesia in comparison to those receiving opioid infusions, and to have measured the quality of pain relief by a pain score. Perhaps the benefits seen by incentive spirometry may be even greater when analgesia is optimal.

No significant reduction in postoperative pulmonary complications was reported previously with several prophylactic measures—incentive spirometry, intermittent positive pressure breathing, or deep breathing exercises.[4] Hall and colleagues also failed to show a reduction in pulmonary complications with incentive spirometry compared with conventional chest physiotherapy in their previous study of a much larger group of patients.[5] We therefore believe that other factors are involved in the development of postoperative respiratory failure which must be addressed before the effectiveness of incentive spirometry is concluded.

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Laparoscopic surgery leads to better postoperative pulmonary function

EDITOR,—We applaud the efforts of John C Hall and colleagues in drawing attention to the importance of postoperative chest physiotherapy,[1] but would point out that prevention is better than cure.

Upper abdominal wounds typically cause a reduction by 50% or more of preoperative forced expiratory volume at one second and forced vital capacity, despite adequate analgesia.[2] These changes are caused by a decrease in lung volume due to basal atelectasis and an alteration of chest and abdominal wall mechanics, respectively.

Although laparoscopic surgery is currently the bête noire of surgery, chiefly as a result of the inadequate training and arrogance of a minority, there is now good evidence that when it is both possible and sensible to use this approach, it leads to better postoperative pulmonary function than open surgery.[2-5] The inescapable conclusion is that although all patients stand to benefit from minimal access abdominal surgery (where applicable), those who stand to benefit the most are those in whom lung function is already compromised and in whom a reduction of lung function variables of 50% might be disastrous.

The counter argument of the theoretically increased risk of hypercarbia in these patients remains only a theoretical argument if a competent anaesthetist and a modern ventilator are used.

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Authors' reply

EDITOR,—It is interesting that a clinical trial evaluating postoperative respiratory complications has drawn comment mainly from anaesthetists. I can reassure them that the patients included in our study at Royal Perth Hospital were treated by a pain control team staffed by anaesthetists.

Our study was pragmatic in nature and did not evaluate the pathophysiology of atelectasis. Some time ago, Schwartz and Lellouch pointed out that there are essentially only two types of clinical studies, explanatory and pragmatic.[1] Explanatory studies look at underlying processes, while pragmatic studies provide management recommendations that are relevant to clinical practice.

Few readers would be surprised at the fact that anaesthetists and surgeons tend to concentrate on different aspects of a problem. In my mind, there is overwhelming evidence that good pain control, including pre-emptive therapy, improves postoperative respiratory function. However, there is a