to sedate children that have stood the test not only of scientific investigation but also of time. Morphine, barbiturates, and the more recent additions such as fentanyl and alfentanil have all been used in critically ill patients without major problems.[5] As propofol does not have a licence for use as an infusion in children and, at best, scientific investigation suggets that it may be detrimental in critically ill children, it seems mandatory that this drug is not used routinely for sedating children in intensive therapy units. Cook should realise that if we use drugs before scientific investigation has shown them to be safe we have only ourselves to blame if they are later shown to cause problems.

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EDITOR,—David O'Flaherty and Anthony P Adams speculate that the lipaemic serum observed in the five children who died after infusion of propofol might be explained by adrenocortical suppression resulting in diminished lipolysis and oxidation of fat.[1]

Lipaemia occurs when large triglyceride rich particles such as chylomicrons and very low density lipoprotein are increased in plasma since, because of their size, these particles reflect light, giving a turbid appearance. Propofol consists of an emulsion of such large triglyceride rich complexes. Lipolysis occurs during relative cortisol excess or insulin deficiency and is associated with increased secretion of very low density lipoprotein from the liver (producing lipaemia) as a secondary response to the increased mobilisation of free fatty acids from adipose tissue. Consequently, cortisol deficiency resulting in diminished lipolysis would not be expected to contribute to the development of lipaemia.

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1 O'Flaherty D, Adams AP. Propofol infusion in children. *BMJ* 1992;**305**:952-3. (17 October.)

EDITOR,—T J Parke and colleagues report on five children who died after receiving propofol infusion for sedation during ventilation.[1] We report a case in which the child recovered.

A 1 month old baby was admitted to the Royal Aberdeen Children's Hospital with an eight day history of paroxysmal cough, whoop, and vomiting. The clinical diagnosis of whooping cough was supported by a raised white cell count ($37{\cdot}1\times10^9$/l) and lymphocytosis ($22{\cdot}2\times10^9$/l). After admission to hospital she gradually deteriorated, with increasingly frequent paroxysms of coughing associated with cyanosis, and became increasingly tired. Intubation by experienced anaesthetists was achieved only after several attempts. During this time she had a short convulsion, which was treated with phenobarbitone. She was treated with mechanical ventilation and sedated with intravenous propofol at a rate of 10 mg/h. After four days her serum was severely lipaemic; the propofol infusion was immediately stopped. Despite this lipaemia there was no evidence of acidosis or clinical evidence of haemodynamic compromise.

The child required 16 days' ventilation and there was concern about her neurological status after extubation, but she made an excellent recovery; at review at 21 months of age she was developing appropriately for her age.

In many ways this case is similar to those reported by Parke and colleagues, but there was no acidosis before serum lipaemia developed. The recognition of lipaemia and early cessation of treatment with propofol may have contributed to this child's recovery.

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1 Parke TJ, Stevens JE, Rice ASC, Greenaway CL, Bray RJ, Smith PJ, *et al.* Metabolic acidosis and fatal myocardial failure after propofol infusion in children: five case reports. *BMJ* 1992;**305**:613-6. (12 September.)

Diagnosing maxillary sinusitis

EDITOR,—N P van Duijn and colleagues' paper on using ultrasound to diagnose maxillary sinusitis misses the point.[1] Focusing on the antrum as the source of nasal symptoms has never been reasonable, since Ewing and Sluder in 1900 pointed out that pain and headache due to nasal conditions may occur in the absence of purulent sinus infection.[2]

In the 1940s Wolff showed that the antrum itself was relatively insensitive and that the pain of sinusitis was more likely to be mediated by congestion in the middle meatus of the nose, which invariably accompanies maxillary sinusitis.[3] Recent advances in endoscopic diagnosis have confirmed this.[4] Using simple outpatient rigid nasal endoscopy, Levine found abnormalities in 58 of 150 patients with nasal and sinus symptoms; no abnormalities had been found on conventional ear, nose, and throat examination.[5] Many of these patients had seen several physicians and had frustrating, longstanding symptoms. Such abnormalities cannot be diagnosed by ultrasound examination but can be treated successfully. Ultrasound examination of the antrum is not popular in Britain since a comparative study showed that it failed to improve on radiology in predicting the presence of fluid in the antrum.[6] As van Duijn and colleagues state, the symptomatic borders between sinusitis and nasal pain are not clear; concentrating solely on confirming or excluding fluid in the antrum is not productive as absence of fluid is most probably a secondary phenomenon due to obstruction of mucociliary clearance pathways in the anterior ethmoid. What is important is to give patients with recurrent or chronic symptoms an endoscopic examination so as to diagnose the problem, be it maxillary sinusitis or not.

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1 Van Duijn NP, Brouwer HJ, Lamberts H. Use of symptoms and signs to diagnose maxillary sinusitis in general practice: comparison with ultrasonography. *BMJ* 1992;**305**:684-7. (19 September.)
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EDITOR,—As N P van Duijn and colleagues point out, the term maxillary sinusitis does not simply denote a cavity filled with pus.[1] It covers a range of disease from temporary obstruction of the sinus ostium by mucosal swelling of any cause to an acute exacerbation of a chronic pansinusitis. Ultrasonographic examination of the maxillary sinus, which van Duijn and colleagues used as "the gold standard," is non-invasive but is a crude means of defining pathology in the area of the maxillary ostium. It is maintenance of the patency of the osteomeatal complex (an area where the frontal, anterior ethmoidal, and maxillary sinuses have a common outlet) that is crucial in the health of all the sinuses. Obstruction can produce symptoms without any opacity or a fluid level. This may be why only 212 of the 441 episodes of sinusitis in the study were confirmed by ultrasonography. Michael Gleeson's point that facial pain attributed to sinusitis is often due to other causes is also pertinent.[2]

Van Duijn and colleagues draw attention to the 10% of patients whose symptoms persisted, half of whom were found to have persistent evidence of maxillary disease. Probably several of these patients had chronic rhinosinusitis, with affected ethmoidal sinuses, which had gone unrecognised. Allergic rhinitis as well as infection may contribute to the mucosal hypertrophy and sinus obstruction. If such patients do not respond to medical treatment, which may include measures to control their allergic rhinitis, antibiotics with a short course of topical decongestants, or a combination of these, then referral is warranted. Rigid nasal endoscopy allows outpatient inspection of the osteomeatal region and may indicate whether surgery is needed to clear the osteomeatal complex.[3] Treatment directed solely at the maxillary sinus will leave many patients with residual disease.

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1 Van Duijn NP, Brouwer HJ, Lamberts H. Use of symptoms and signs to diagnose maxillary sinusitis in general practice: comparison with ultrasonography. *BMJ* 1992;**305**:684-7. (19 September.)
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Bone mineral measurements

EDITOR,—Kay-Tee Khaw and colleagues reported bone measurements adjusted for body mass index (weight/height^2). This is an example of the growing use of the body mass index to normalise bone mineral estimates made by single photon absorptiometry, dual photon absorptiometry, and dual energy x ray absorptiometry.[2-4] The rationale for using the body mass index, which is often used as a measure of adiposity, is unclear but is presumably related to thinness being a risk factor for osteoporosis and adipose tissue having oestrogenic properties.

There are potentially serious statistical pitfalls, however, in using the bone mass index adjustment uncritically. Numerous studies have shown that measurements of bone mineral content (in g or g/cm) and areal bone density (in g/cm^2) are positively, and independently, correlated with body weight and height.[5-7] This shows that, in general, bone mineral content and areal bone density are

predicted by body size and not by adiposity. The essential difference is that an adjustment for body size involves weight and height acting in the same direction, but a correction for adiposity, in the form of body mass index, forces weight and height to act in opposite directions (a positive correlation with body mass index signifies an increase with weight but a simultaneous decrease with the square of the height).

The exact nature and significance of the relation with weight and height depend on the skeletal site measured and the population group studied. However, using body mass index when adjustment for body size is required may lead to spurious relations emerging or genuine relations being obscured.

The practical solution is always to adjust for both weight and height separately in multiple regression analysis. If their regression coefficients emerge with opposite signs (usually a negative height coefficient) then this shows that adiposity is a significant predictor of bone mineral content or areal bone density. Conversely, if the two coefficients have the same sign (usually positive) then it is the overall size of the body that is important.

A more informative procedure is to use logarithmic transformations of weight and height in the regression.[8,9] This allows the effect of adiposity to be readily examined because log BMI=log weight−2 log height. Thus if the log height coefficient is approximately twice the log weight coefficient and of opposite sign then log weight and log height can legitimately be replaced by log BMI. Unfortunately this is rarely the case.

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Euthanasia

EDITOR,—Euthanasia has been debated since before the time of Hippocrates and will probably be debated for the next 2000 years while we struggle with unanswerable questions.[1] Some disquieting questions arise. Two correspondents recommend that patients should be rendered comatose with drugs until death comes "naturally."[1] Surely this merely induces a permanent pharmacological coma, which can be regarded only as a living death, the very thing we are trying to avoid. It seems to be more an emollient to the consciences of the medical attendants than an attempt to deal with the problem, and it should be recognised for what it is—an ethical fraud.[2] To administer opiates or any other sedatives in large doses with the stated intention of treating pain but with the definite consideration that the treatment will also cause the death of the patient is euthanasia. It doesn't matter how you dress it up.

Royal commissions, as with any other committee, may be set up with the unstated intention of promoting or defeating an idea. Where attitudes to euthanasia are concerned, all the arguments seem to reduce to whether one considers the intentional removal of life or continued life in severe pain with loss of dignity to be the greater evil. In this respect the arguments become polarised and there is no way out of the dilemma. There is hardly any law that is not open to abuse of one sort or another, but surely it is time we heeded the words of Shaw in *Doctor's Dilemma*—"The theory that every person alive is of infinite value is legislatively impracticable"—and returned to the notion that murder is unlawful death with malice aforethought.

I grant that pain and all other reversible problems must be treated to the best of our ability and no person should be placed in the terrible predicament of feeling that he or she has a duty to die before the possibility of euthanasia is contemplated. But to continue with useless life, with the person reduced to nothing more than a wailing, pitiful shell begging for a release that is not forthcoming, is also cruel. A royal commission is hardly likely to answer the question to the satisfaction of all concerned, but, whether or not it were to recommend euthanasia, euthanasia incorrectly applied would still be murder and, for that matter, incorrectly applied abortion could be viewed in the same light.

Euthanasia is happening at the present time and Dr Nigel Cox has, through his actions, shown us this. To deny this is like saying that abortion never happened before it was legalised. It is hardly likely to stop merely because we refuse to change the law. Abuse under the law is always a possibility, even perhaps likely. But is it any worse than at present, when euthanasia is administered furtively and the alternative is sanctimoniously to deny a patient's wish of a peaceful release from pain? Perhaps it may also be said that we prefer to live with the uncomfortable feeling that the crime of caring for a patient is not in an act of compassion but in being caught.

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1 Correspondence. Euthanasia. *BMJ* 1992;**305**:951-2. (17 October.)
2 Goldenring J. Code or no code decisions. *N Engl J Med* 1979;**300**:1058.

EDITOR,—The extensive correspondence on euthanasia shows that the potential for abuse is a major element in many people's doubts about the wisdom of legalising euthanasia.[1] But the difficulties described in many of the letters can—and, as we have recently seen, do—happen whether or not euthanasia is legalised. Nor are these difficulties unique to euthanasia.

Doctors do many things that are both controversial and open to abuse. Problematic areas include prescribing heroin to addicts and giving psychiatric treatments of special concern—that is, psychosurgery and hormone implantation. These treatments are carefully controlled. In the first case only approved specialists are licensed. In the second case an independent doctor and two appointed non-doctors must certify in writing that the patient is capable of understanding the nature, purpose, and likely effects of the proposed treatment and has consented to it. Overprotecting the patient from what he or she chooses becomes a more likely problem than the reverse.

Do patients have less of a right to determine the time and manner of their deaths than their doctors? Few would claim so. Legislation that permitted assisted suicide, but only under carefully defined conditions, would not only serve those patients who want informed choice but also help to protect all of our patients. The advantage of appropriate legislation is that euthanasia would be controlled. This is the best way of all to avoid the feared "slippery slope."

Our disquiet about Dr Nigel Cox's action perhaps arises partly because we suspect that he is only one of many who have tried, in good conscience, to act on behalf of their patients. Imagine what might happen if he and like minded others acted within a law in which they were obliged to refer their patients to designated specialists to determine not only that they wanted to die but also—vitally—that they had received the most effective treatment. Their patients might be offered alternative and better hospice skills. They might then choose to live instead of to die.

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EDITOR,—Much of the discussion concerning euthanasia ends with an account of the high quality of care and freedom from pain that are now achieved in good hospitals and hospices.[1] Less attention seems to be given to the wishes of the patient. For instance, when the time comes that I can no longer care for myself or am in continuous pain I do not wish to go into a hospice. The quality of life available there, however caring, is not something I want for myself. What I shall want is to die with dignity and without pain, and I shall be grateful to any doctor who can help me to that end. If, when the time comes, I change my mind I have only to say so and this will immediately negate any advance directive I may have given.

Those who believe that life is sacrosanct are perfectly entitled to their hospice; I would not dream of denying it to them. I have different beliefs: why should those people be allowed to dictate to me?

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EDITOR,—Many people have seized the opportunity presented by the case of R *v* Dr Nigel Cox to assert their points of view regarding voluntary euthanasia.[1] Unfortunately, the special circumstances surrounding the death of Mrs Boyes—central to this case—have been insufficiently emphasised.

Five days before her death Mrs Boyes's was dying of her own volition, asking only that she be relieved of pain. Oral morphine had ceased to be effective. Intravenous heroin was also ineffective, and suitable points for continued intravenous administration were becoming increasingly difficult to find, so Dr Cox resorted to increased doses of heroin by subcutaneous infusion. Not only did the pain increase but an allodynia developed, which persisted for the remaining days of Mrs Boyes's life. She had been a very stoical patient, but now she screamed "like a wounded dog," and "yelped with pain if touched."

Such a combination of features led us to suspect that Mrs Boyes was suffering from a rare condition known as paradoxical pain. Subsequent research at the Pain Research Institute, Liverpool, supported this opinion,[2] and two conclusions: that continued use of heroin by Dr Cox—in even larger doses or by epidural or intrathecal infusion—would have further compounded Mrs Boyes's problems and that there was then no rationale for using other drugs.