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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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