sex of the baby at an early stage, generally before the mother has handled or often even seen the baby. By chance alone approximately half the mothers are going to be mildly to markedly disappointed in the sex of their offspring. In such cases there is a risk that the set of the mother towards her child may be detrimentally affected.

In the terminology of ethology the binding of the mother to her child is dependent on certain releaser stimuli. The clinical circumstances in which the first contact takes place between the mother and her newborn child can hardly be conducive to what is sometimes a precarious binding process.¹ However, it would be possible to avoid declaring the sex of a newborn baby and allowing the mother to discover this for herself. Satisfaction of her curiosity will necessitate both looking at and handling the baby to discover the sex. This exploration may provide important releaser stimuli²⁻⁶ uncontaminated by maternal disappointment. To avoid unnecessary anxiety the practice of not declaring the sex at birth could be explained beforehand.

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¹ Kennell, J. H., *et al.*, *Developmental Medicine and Child Neurology*, 1974, **16**, 172.

² Collias, N. E., *Ecology*, 1956, **37**, 228.

³ Hersher, L., Moore, A. U., and Richmond, J. B., *Science*, 1958, **128**, 1342.

⁴ Klaus, M. H., *et al.*, *New England Journal of Medicine*, 1972, **286**, 460.

⁵ Barnett, C. R., *et al.*, *Pediatrics*, 1970, **45**, 197.

⁶ Klaus, M. H., *et al.*, *Pediatrics*, 1970, **46**, 187.

Laparoscopy explosion hazards with nitrous oxide

SIR,—Readers of your correspondence columns may be confused as to the difference between hysteroscopy, the subject of our letter (1 March 1975, p 511) and laparoscopy (Professor J. S. Robinson and others, 27 December, p 760). During hysteroscopy the uterine cavity is insufflated with gas or liquid to allow telescopic inspection, whereas with laparoscopy it is the peritoneal cavity which is insufflated with gas. The changes we observed in PaCO_2 during hysteroscopy have indeed been further investigated, and our original thoughts (1 November, p 288) seem to have been confirmed.

Turning back to laparoscopy we find it hard to comprehend the statement by Professor Robinson and his colleagues concerning "initial opening of the bowel by diathermy" as this is not usually a part of laparoscopic sterilisation, although it may occur as a complication.¹ However, diathermy of the bowel may be part of an elective surgical procedure, and N_2O is almost universally used during anaesthesia. Explosions such as those described by the Birmingham workers (27 September, p 764 and 27 December, p 760), although unfortunate, do not seem to be very frequent. Nevertheless, an explosion in a closed cavity may be more hazardous than one in an open cavity.

Leaving the above points aside, we are in agreement with Professor Robinson and his colleagues that more work needs to be done in this area. Nevertheless, for those performing tubal diathermy the choice lies between using CO_2 , with some risk of cardiovascular collapse (on occasion fatal), and N_2O , with a theoretical risk of causing an intraperitoneal explosion. Regrettably the data currently available do

not permit, in our opinion, quantification of these two risks.

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¹ Rawlings, E. E., and Balgobin, B., *British Medical Journal*, 1975, **1**, 727.

"Whites only" at the University of Natal

SIR,—On 17 December the cabinet of the South African government sent instructions to the Medical Faculty of the University of Natal to stop admitting first-year African medical students. This was done without consulting and with the subsequent opposition of the Faculty.

The Medical Faculty of the University of Natal was established in 1951 to educate non-White medical students. It has developed over the past 25 years into a leading medical school and is the main place of medical education for non-Whites in South Africa. Limited numbers of non-White students are educated at Cape Town and Witwatersrand universities, while the White students have a choice of five medical schools.

Now that the Natal Medical Faculty is established as a leading centre the South African government has decided unilaterally to take it over for White students. First, African students are to be excluded and will have to go to an African medical school north of Pretoria in the intellectual wilderness of the "Homelands." This is not yet built and will not produce doctors before 1982. The Indian medical students will then be excluded and will also have to start up a new faculty in Durban in a run-down tuberculosis hospital—King George V Hospital.

Thus once again the South African government is taking the best for the White community and the disfranchised non-Whites become the victims of a political ideology that must be condemned by all people of the democratic Western world.

SOUTH AFRICAN DOCTOR

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Steatorrhoea complicating therapy with mefenamic acid

SIR,—We were interested to read the report by Drs J. S. Marks and M. H. Gleeson (22 November, p 442) describing mefenamate-induced steatorrhoea.

We have seen two such cases in women aged 44 and 69 years respectively, who had profuse steatorrhoea, abdominal distension, and weight loss during mefenamic acid therapy. One became asymptomatic when ibuprofen was temporarily substituted for mefenamic acid, but the diarrhoea recurred on restarting mefenamic acid. Faecal fat output was increased (14.2 and 12.5 g/24 h respectively), with normal serum vitamin B_{12} and folate levels. However, jejunal biopsy appearances were abnormal. The villi were irregular and many leaf-forms were present. There was plasma-cell, macrophage, and neutrophil-cell infiltration. Goblet cells were increased in numbers, both in the crypts, which were hyperplastic, and in the surface epithelium. These appearances reverted to normal on stopping therapy.

Experiments in animals have shown that mefenamic acid and related compounds cause small-bowel mucosal damage and ulceration.¹ This is also seen in indomethacin therapy. It is worthy of note that both drugs are recirculated enterohepatically.^{2,3} Our results suggest a direct toxic action on the small-bowel mucosa.

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¹ Kaump, D. H., *Annals of Physical Medicine*, 1967-8, **9**, suppl, p 16.

² Brodie, D. A., *et al.*, *Toxicology and Applied Pharmacology*, 1970, **17**, 615.

³ Glazko, A. J., *Annals of Physical Medicine*, 1967-8, **9**, suppl, p 23.

Laboratory proficiency

SIR,—Your leading article (3 January, p 5) refers to the objective assessment of histological opinions. Semantics apart, a consultant histopathologist is appointed after a long training and must defend his opinions daily with clinicians who together quickly establish the measure of his professional competence. Finally, difficult and questionable necropsy findings are commonly debated at clinicopathological conferences, which in our view provide the best form of medical audit that we can devise. Medicine, including pathology, is still a long way from being an exact science.

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Hazards of cephalosporins in penicillin-allergic patients with meningitis

SIR,—In discussing the treatment of meningitis and encephalitis Dr C. C. Smith (8 November, p 335) states a number of times that when the patient is allergic to penicillin cephaloridine should be used intravenously and perhaps intrathecally.

We suggest that this is not the best alternative therapy. Firstly, if cephaloridine is to be used then intrathecal therapy is mandatory. None of the cephalosporins efficiently penetrate the blood-brain barrier.¹ A recent review of 106 cases of bacterial meningitis treated with cephalosporins² indicated that response to intravenous therapy was poor unless accompanied by simultaneous intrathecal administration of the drug. Cephaloridine, the cephalosporin which crosses the inflamed meninges with the least inefficiency, did not achieve therapeutic levels in the cerebrospinal fluid despite large intravenous doses.

Secondly, there is evidence that patients who are allergic to penicillin have an increased risk of allergy to cephalosporins as well. In a recent review of nearly 10 000 patients receiving treatment with cephalosporins Petz³ showed that the overall incidence of allergic reactions was 1.2% in those with no history of allergy to penicillin. However, this was increased nearly five-fold to 8.7% in those who did have a positive history of penicillin allergy.

Chloramphenicol has neither of these disadvantages. It crosses the blood-brain barrier

extremely well, obviating the need for intrathecal administration. Also we are not aware of any evidence to suggest that penicillin allergy increases the risk of adverse reactions to chloramphenicol. Consequently it would appear that when the organism is sensitive to chloramphenicol this drug is the treatment of choice for bacterial meningitis in the penicillin-allergic patient.

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¹ Moellering, R C, jun, and Swartz, M N, *New England Journal of Medicine*, 1976, **294**, 24.

² Fisher, L S, et al, *Annals of Internal Medicine*, 1975, **82**, 689.

³ Petz, L D, *Postgraduate Medical Journal*, 1971, **47**, suppl, p 64.

Polyuria in paroxysms of tachycardia

SIR,—Your expert's explanation of the polyuria in paroxysms of tachycardia (8 November, p 339) is incomplete and may be incorrect. Wood¹ was attracted by the hypothesis that polyuria is the result of a transient diabetes insipidus. Thus the diuresis may be related to left atrial distention, non-osmotic stimulation of the baroreceptors, and subsequent anti-diuretic hormone inhibition.^{2,3} This does not exclude other possible mechanisms, but it bears no direct relationship to cardiac output and renal perfusion if the attack is not a prolonged one. The polyuria is immediate but bladder emptying may be delayed.

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¹ Wood, P, *British Heart Journal*, 1963, **25**, 273.

² Luria, M H, Adelson, E I, and Lochaya, S, *Annals of Internal Medicine*, 1966, **65**, 461.

³ Maxwell, M H, and Kleeman, C R, *Clinical Disorders of Fluid and Electrolyte Metabolism*, 2nd edn, p 241. New York, McGraw-Hill, 1972.

Segregated smokers

SIR,—Your call for a greater protection of the majority of the public who do not smoke from aggravation by those who do is most welcome. It would, however, carry greater weight if, as a profession, we gave a better example in our hospitals.

We could do much more to protect non-smokers in our wards. The *Which?* survey¹ showed that 68% of smokers and 77% of non-smokers thought that smoking should not be allowed on hospital wards. For many years my own have been non-smoking wards and I have found that most smokers are grateful for the help they get to stop smoking while in hospital, and very few have complained. Those patients suffering from terminal disease or who are unwilling to stop can usually be moved to side wards.

I recently inquired into the sale of cigarettes in 83 large hospitals, including the 12 London teaching hospitals, the 33 teaching hospitals outside London, 21 district hospitals in the NW Thames Area, and the 16 London postgraduate teaching hospitals. All replied and the results were as follows: of 68 hospital shops 49 (72%) sold cigarettes, while of 81 ward trolley services 38 (47%) sold cigarettes. It is a disgrace that nearly three-quarters of our hospitals still sell cigarettes and surely makes a mockery of preventive medicine. How can we expect patients to take notice of our

advice on smoking if we continue to sell cigarettes in our hospitals and often make ill-gotten profits thereby for staff amenities? I recently visited a well known chest hospital where many patients are admitted for the treatment of lung cancer, chronic bronchitis, and coronary heart disease and I was amazed to find a prominent display of cigarettes in their hospital shop. It is surely the responsibility of medical committees to advise area health authorities on this matter. The National Association of Leagues of Hospital Friends and the Women's Royal Voluntary Service, who provide us with such an excellent service, tell me that they are very ready to stop selling cigarettes in hospital shops that they run if asked by their hospital authorities to do so.

May I suggest a few principles which could be adopted on smoking in hospitals? (1) Cigarettes should not be sold in any acute general hospital. (2) Non-smokers should have the right to be nursed in smoke-free wards. (3) Non-smoking should be the norm in all acute medical and surgical wards. (4) Hospitals should provide segregated areas for smokers. An approach to the problem on these lines by all hospitals would do much to demonstrate that we actually do consider the prevention of 50 000 premature deaths from cigarette smoking each year a matter of priority.

KEITH BALL

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Hon Secretary,
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¹ *Which?*, February 1975, p 56

SIR,—I am, of course, well aware of your establishment's views on smoking. Whatever my own views on smoking, however, I feel I must take issue over this article (31 January, p 244) as I feel that the style and the composition failed to attain a standard worthy of the importance of the subject, nor indeed of a leading article in the *BMJ*. I had the sensation when reading it that not only had the writer's anti-smoking militancy been over-stressed but that there was perhaps a smack of the chauvinism and casuistry so familiar in one who has recently stopped smoking. Was there possibly even a suggestion among the subject matter of withdrawal symptoms? Those trite clichés, "this obnoxious practice," "inflict their offence on people who resent it," "the non-smokers, as well as being right . . .", and "there are already too many people saying No," might look in place coming from the mass media or from a politician, but do little to grace a learned journal.

To "puff carcinogens" is, to my mind, unbridled exaggeration. Surely the minute traces of tobacco tars joining those of many other "indictable substances" in the atmosphere can be of little significance. The theme of segregation of social undesirables, either as therapy or punishment, is to my mind archaic. In practice it could be said with justification, I think, that segregation has proved of very doubtful value in either context and in many instances has proved actually harmful. As to the benison of "pure air," one imagines that—if I myself may resort to cliché—this would be more likely to win the plaudits of the militants than those of ordinary folk. Again, if we are to have stipulated "smoking areas" may this not lead to demands for other areas earmarked for further doubtful attributes and activities? On the one hand should we segregate those with halitosis, or bromidrosis, or those who use the "wrong" suntan ointment? On the other hand should we have areas for teenage

sex play and perhaps for mugging and even less acceptable activities too?

Finally, Sir, is it no longer customary to accord capital letters to the West End of London? And, since smokers are stated to be brainwashed, should we assume that non-smokers' brains are unwashed?

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Propanidid in dysrhythmias

SIR,—Dr R J Vecht and his colleagues (18 October, p 143), report a case (No 1) in which ventricular tachycardia reverted to sinus rhythm during induction of anaesthesia with propanidid.

I would, if I may, question the diagnosis of ventricular tachycardia as it would appear that the most important ECG leads for the interpretation of the condition have been omitted—namely, the standard and unipolar leads. It may be argued that the differential diagnosis in this case includes atrial tachycardia and junctional tachycardia, both with aberrant ventricular conduction. In the absence of standard and unipolar leads, particularly leads 2, 3, AVF, and AVR, differentiation between these two is difficult.

In my opinion no ventricular tachycardia is evident in the ECG tracings reproduced with the article and I am convinced that they are more suggestive of a junctional tachycardia with aberrant ventricular conduction. Lead V₁ favours an aberration. In general, approximately 85% of aberrantly conducted supraventricular dysrhythmias show right bundle-branch block form and a triphasic ("M" shaped and upright) pattern, as shown in lead V₁. Rapid junctional tachycardia at a ventricular rate of close to 200/min usually shows aberrant conduction. A regular baseline is frequent in junctional tachycardia and unusual in ventricular or atrial tachycardia. In nearly all of the ECG leads presented the baseline is regular.

Lead V₁ appears to show P waves at P-P intervals of 0.3 s and they precede each QRS complex, supporting my view that this was a junctional tachycardia of "upper junctional type." Certainly P waves are not visible in leads V₂-V₆ owing to the high ventricular rate, but the R-R interval in all the leads presented is also 0.3 s, which confirms the origin of this arrhythmia.

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Lesser curve necrosis after proximal vagotomy

SIR,—Your recent leading article (29 November, p 487) and the paper by Professor David Johnston (6 December, p 545) on highly selective vagotomy have again drawn favourable attention to this operation for duodenal ulcer. I feel qualified to comment as I have over the past five years collected a personal series of 260 highly selective vagotomies for duodenal ulcer and for its complications. The results have in general been very satisfactory and I shall be reporting them in full in due course. However, to date there has been no operative mortality, minimal operative morbidity, and only four patients have had recurrence of their duodenal ulcer.