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 PaCO₂ 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 N₂O 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 CO₂, with some risk of cardiovascular collapse (on occasion fatal), and N₂O, 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.

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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 B₁₂ 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