very first year, and this could be the resultant of some real diminution in contraceptive efficacy in the third year combined with the group's lower fertility for the above reason. But, as Dr Sivin says, the third year rate should be obviously higher if there were serious interference by deposits with the contraceptive action.

Mr Newton states that there was no difference in age and parity between the two groups. But the difference in rates of removal for planned pregnancy (zero for the "replacement group," 10% for the "continuation group") could point to an important difference in attitudes to the risk of unplanned pregnancy and hence possibly in behaviour. When electing for replacement or continuation with the same IUD the women were presumably aware of the possibility of reduced contraceptive protection if they took the second course. The "replacement group" might therefore accumulate an excess of users of additional methods such as spermicides. They might also check more obsessively for lack of cervical displacement of their IUD (a potent cause of pregnancy with this particular design).

Pending more clinical data, what guidance can be offered to clinicians? A blanket policy of replacement at two years makes medicolegal sense but might be unnecessarily early for those whose IUDs are least affected by incrustation phenomena. Measuring copper levels in cervical mucus, as suggested by Dr Gosden and her colleagues, may one day be practicable. For the present, if use beyond two years is desired it would be wise to discuss the available facts carefully with the IUD user herself.

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¹ Loudon, Nancy. Personal communication.

² Johnson, A B, et al, *Contraception*, 1976, **14**, 507.

Maternal antibody against pertussis

SIR,—In your leading article on whooping-cough immunisation (2 July, p 5) in which you aptly support the advice of the Joint Committee on Vaccination and Immunisation¹ you state in previous studies "maternal antibody against pertussis was found (my italics) not to be transferred to the fetus across the placenta, so that there was no objection to early immunisation, as was the case in diphtheria prophylaxis." The joint committee was more cautious, saying that whooping cough "is different from other infectious diseases of childhood in that maternal protective substances are not generally believed (my italics) to be transferred to the newborn baby."

This belief is part of the pertussis folklore and is presumably based on the readiness with which infants can develop whooping cough in the first few months of life. It even leads some to the false deduction that antibodies play only a minor role in protection against whooping cough or that only IgM is of significance. Actually the levels of pertussis agglutinins in infants' sera are directly related to those in the mothers' sera.^{2,3} For example, mother's titre 128, infant's titre 16 at 3 months and 8 at 4 months; mother 64, infant 8 at 3 months and <4 at 4 months; mother 16, infant 4 at 3 months and <4 at 4 months; mother 8, infant <4 at 2 months.

However, the mothers' sera rarely contain

detectable amounts of antibody to pertussis factor 3 and so the infant is susceptible to type 1,3 infection, just as vaccinated children were before 1967 when many batches of those earlier vaccines lacked antigen 3.

If, as you suggest, the presence of maternal antibody against diphtheria is reason for avoiding early immunisation (before 3 months of age), then the same applies to pertussis. The joint committee's recommendation that vaccination should start at 3 months of age is therefore not the compromise that you imply but is consistent with the currently available evidence.

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¹ *Whooping Cough Vaccination. Review by Joint Committee on Vaccination and Immunisation*. London, HMSO, 1977.

² Abbott, J D, Preston, N W, and Mackay, R I, *British Medical Journal*, 1971, **1**, 86.

³ Preston, N W, et al, *Journal of Hygiene*, 1974, **73**, 119.

Carbon dioxide-dependent *Staphylococcus aureus*

SIR,—We read Dr M Rahman's letter (30 July, p 319) with interest, as we have recently isolated a similar strain of carbon dioxide-dependent *Staphylococcus aureus* from a clinically infected occipital wound in a child. These strains, though rare, are well documented as dwarf or G strains and usually produce tiny colonies in the absence of added carbon dioxide after prolonged incubation.^{1,2} Although they may be isolated from clinical lesions in man their virulence as judged by mouse inoculation has been reported as low.² Dwarf strains are described as being induced by antibiotic administration,³ and a previous isolation in this department of a carbon dioxide-dependent strain was from a patient with osteomyelitis of the radius who was receiving penicillin.

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¹ Wilson, G S, and Miles, A A, *Topley and Wilson's Principles of Bacteriology, Virology and Immunology*, 6th edn. London, Arnold, 1975.

² Slifkin, M, et al, *American Journal of Clinical Pathology*, 1971, **56**, 584.

³ Wise, R I, and Spink, W W, *Journal of Clinical Investigation*, 1954, **33**, 1611.

Genital yeast infection

SIR,—The question whether genital yeast infection should be considered a sexually transmitted disease (STD) (Dr R N Thin and others, 9 July, p 93) might benefit from a clarification of the terminology. Is STD defined as the presence of micro-organisms that can cause disease and are usually transmitted from one host to another by sexual contact? This is the concept that seems implicit in the routine STD statistics, and the problem is then to determine whether or not sexual contact is the usual mode of transmission of genital yeasts.

On the other hand it seems implicit in the name itself that a patient with STD should (a) have a disease, and (b) have acquired that disease by sexual contact. If the presence of genital yeasts in the absence of any clinical

problem is not a disease, then the fact that they might be "usually sexually transmitted" would not make their presence an STD; and similarly, if genital yeast infection can occur without the yeasts having been sexually transmitted, then any such infection would not be STD.

It would be of value statistically, and maybe also clinically, if we were to differentiate clearly between micro-organisms that are sexually transmissible and diseases that are sexually transmitted.

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Infectious mononucleosis complicated by hydrocele

SIR,—Infectious mononucleosis is now recognised as a systemic disease, but its association with hydrocele has not so far been described. The following case history may therefore be of interest.

A 4-year-old boy was admitted with a 36-h history of swelling of the right scrotum. A few days before the appearance of the swelling he had felt unwell and feverish and had noticed a swelling on the left side of his jaw. On examination there were large masses of firm, painless lymph nodes in both posterior triangles of the neck, the axillae, and the groins. His spleen was palpable to 3 cm below the costal margin, but the liver was not palpable. There was a right hydrocele with both testicles easily palpable. Investigations showed haemoglobin 13 g/dl, leucocytes $25.6 \times 10^9/l$ (25.600 mm^3) with 40% lymphocytes and numerous (47%) atypical mononuclear cells, and normal platelet count. The Paul-Bunnell test was strongly positive. Screening for cytomegalovirus, toxoplasmosis, and mumps was negative. He was discharged home and when seen later in the outpatient clinic the hydrocele had resolved and the spleen had also decreased in size.

The aetiology of the hydrocele was obscure, but it may be speculated that it was due to infiltration with large atypical cells, which has been described in other complications.^{1,2} Pleural effusion with infectious mononucleosis has been described and so has the urogenital complication of acute urinary retention.³ The patient reported presented with hydrocele and generalised lymphadenopathy, both conditions resolving during the same clinical course, and infectious mononucleosis was shown to be the primary diagnosis.

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¹ Michel, R G, et al, *Archives of Otolaryngology*, 1975, **101**, 486.

² Sarka, T K, *Diseases of the Chest*, 1969, **56**, 359.

³ Sperber, A, Tessler, A N, and Berczeller, P, *Urology*, 1973, **2**, 456.

Measuring airways obstruction in asthma

SIR,—Your recent leading article (13 August, p 414) is a timely reminder of the importance of frequent measurements of airways obstruction in the management of asthmatic patients.

The instruments you mention, however, the peak flow meter or gauge, are very useful but relatively expensive. For this reason I was associated with the development of a cheap and simple instrument¹ to monitor changes in airways obstruction and provide a warning of

impending danger. This monitor consists of a spring piston that slides along a rod within the body of the instrument and moves an indicator along an arbitrary scale. In order to keep the cost down and make it semi-disposable no calibration has been attempted, but clinical trials have shown the readings to be reproducible and may be related to spirometric measurements initially.¹

Readings are made several times a day by the patient and are a most useful adjunct to the control of therapy when seen by the doctor during consultation. Also it soon becomes possible to establish a danger point below which help should be sought. The monitors are manufactured by Vitalograph Limited.

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¹ Haydu, S P, Chapman, T T, and Hughes, D T D, *Lancet*, 1976, 2, 1225.

Aerosol corticosteroids and deaths from asthma

SIR,—We consider Dr H Morrow Brown's letter (28 May, p 1408) to be timely and we agree with him that the opinion expressed in your leading article (30 April p 1117) could certainly cause confusion, doubt, and anxiety in those who care for asthmatics.

We have been concerned about treatment regimens with corticosteroids for asthma since 1954. About that time a number of trials in Britain with cortisone acetate were inconclusive,^{1,2} but other trials demonstrated its efficacy in improving the ventilatory capacity in asthmatic adults³ and children.^{4,5} There followed an era when opinions were sharply divided concerning the administration of steroids in acute and chronic asthma, and a number of tragedies occurred as a result of patients being switched from the care of protagonists to antagonists of corticosteroid therapy. Happily there is now more agreement concerning the use of steroids and it would be rare indeed for corticosteroids to be withheld in a case of status asthmaticus.

Originally we tried local aerosol corticosteroids for the treatment of asthma in 1959.⁶ At that time we were unable to demonstrate any advantage of hydrocortisone acetate over placebo and concluded that to be effective it would be necessary to give aerosol steroids in such a high dosage that a systemic effect would occur from local absorption. However, the preliminary trials with beclomethasone dipropionate of Dr Morrow Brown and his colleagues in 1971⁷ and their later results⁸ were so convincing that we instituted trials in 1972. We have been using beclomethasone aerosols extensively since then and our initial⁹ and long-term experience¹⁰ has been almost identical with that of Dr Morrow Brown and his colleagues in Derby. We are in full agreement with their practice of issuing emergency supplies of oral steroids to severe asthmatics who have had systemic steroids in the past. We agree that patients who are to be treated with steroid aerosols and who have been on either oral steroids or corticotrophin (ACTH) should be gradually weaned and closely monitored during this period. When they are successfully weaned then an addendum is attached to their blue steroid card. This addendum states how long the patient has been on steroids, the date when he or she was

weaned, and a note to the effect that an emergency supply of steroids has been issued to the patient for use in the event of severe asthma, injury, or infection, together with the dose of steroids that should be taken. We have found that such an issue of emergency steroids has only rarely been abused and we are convinced that this procedure has saved lives.

We also agree with the practice of the Derby clinic in issuing bronchodilators to patients subject to acute sudden attacks. Here such patients are issued with a bronchodilator in the form of salbutamol aerosol (Ventolin), which can be used with benefit before inhaling their aerosol corticosteroid and also when acute attacks occur.

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- ¹ Savidge, R S, and Brockbank, W, *Lancet*, 1954, 2, 889.
- ² Medical Research Council, *Lancet*, 1956, 2, 798.
- ³ Davies, B M, and Williams, D A, *British Medical Journal*, 1955, 2, 293.
- ⁴ Kennedy, M C S, and Thursby-Pelham, D C, *British Medical Journal*, 1956, 1, 1511.
- ⁵ Kennedy, M C S, and Thursby-Pelham, D C, *British Medical Journal*, 1958, 1, 243.
- ⁶ Kennedy, M C S, and Thursby-Pelham, D C, *Lancet*, 1960, 2, 816.
- ⁷ Brown, H M, and Storey, G, VIIIth European Congress of Allergy, 1971, Marseilles.
- ⁸ Brown, H M, Storey, G, and George, W H S, *British Medical Journal*, 1972, 1, 585.
- ⁹ Kennedy, M C S, and Posner, E, *Bulletin de Physiopathologie Respiratoire*, 1973, 9, 816.
- ¹⁰ Kennedy, M C S, Posner, E, and Thursby-Pelham, D C, *Postgraduate Medical Journal*, 1975, 51, suppl 4, p 84.

Isolation of campylobacters

SIR,—Following the publication of Dr M B Skirrow's paper on campylobacter enteritis (2 July, p 9) several laboratories which have previously not isolated the causative organisms are attempting to do so. Campylobacters have a growth requirement of an atmosphere in which the tension of oxygen is reduced and that of carbon dioxide increased and this has proved to be an inconvenience to those laboratories in which cylinders of hydrogen or nitrogen were not previously kept. I have found that it is possible to produce the appropriate atmospheric conditions with gas generating envelopes.

Initially in our laboratory we simply incubated the inoculated agar plates in a closed 2.5-l jar containing an activated BBL GasPak hydrogen-generating envelope but no catalyst. The organisms grew well, but the system was abandoned because it was potentially dangerous since it entailed maintaining hydrogen under pressure at 43°C in an incubator which was not spark-proof. The difficulties and dangers can be overcome by using two vented jars, one simply to generate a safe and suitable atmosphere which can be transferred to the other in which the agar plates are incubated.

The generating jar contains a catalyst and in it one places either an Oxoid Gas-Kit (H₂ and CO₂) or two BBL GasPak gas-generating envelopes, one producing CO₂ alone and the other CO₂ and H₂; both BBL envelopes may be necessary to produce adequate CO₂. After activation of the envelopes the generating jar is closed and left for a sufficient time for all of the gases to be released, 30 min with the Oxoid Gas-Kit, 150 min with the BBL GasPaks. The jar should then contain a mixture primarily of N₂ and CO₂, with some H₂. At this time the inoculated agar plates are placed in the second

incubating jar, which contains no catalyst. It is closed, the air evacuated, and 80 ml of air is reintroduced with a syringe. The generating and incubating jars are then connected to each other; the gases flow from the former to the latter and equilibrium is reached in a few seconds. All of the taps are closed, the jars are separated, and the one containing the plates incubated. The incubating jar should be safe, as it contains only a small amount of hydrogen and is under a slightly negative pressure. I have tested the system with eight strains of *Campylobacter jejuni* and it consistently produces satisfactory results.

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Activity and recurrent hernia

SIR,—Your leading article (2 July, p 3) is entirely correct in stressing the lack of correlation between early ambulation and recurrent hernia. It is, however, the long-term follow-up studies that are meaningless in evaluating the relative merits of the various techniques of herniorrhaphy. So many conditions enter such reports that the method of operation is but another variable.¹ Anson and McVay demonstrated many years ago that there are inherent defects in the various layers of the inguinal area that are not anatomically apparent at operation.² It has also been noted that "recurrent" hernias often occur in these tissues adjacent to the primary repair.

The problem is not that traditions die hard, but rather the fact that under the National Health Service surgery in British hospitals has become to an extent emergency-directed. Patients for elective operations (such as herniorrhaphy) are relegated to less important positions. The economic loss to the country of prolonged postoperative hospitalisation and disability could be prevented, but it would cost money to do so.

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Salen,
Isle of Mull

- ¹ Jones, T I, *American Surgeon*, 1975, 41, 20.
- ² Anson, B J, McVay, C B, and Morgan, E H, *Surgery, Gynecology and Obstetrics*, 1938, 66, 186.

More hydatidiform moles?

SIR,—It was with great interest that I read the letter from Dr John P Calvert (27 August, p 578) on the increased incidence of hydatidiform mole that he had noticed from November 1976 to June 1977.

In Walton Hospital, Liverpool, the average occurrence of hydatidiform mole is about two or three a year, but when the "Asian" influenza epidemic of 1957 occurred, in the three months January-March 13 cases were admitted. All of the patients had had influenza at or around the time of conception and in six cases the husband was also ill with the disease. Four of the patients had spontaneous rupture of the uterus, the mole eroding the uterine wall completely, which necessitated removal of the uterus. In another patient multiple secondary growths were found in the lungs; as she was then 40 years old the uterus was removed and subsequently these secondaries completely disappeared. All the patients were followed up clinically and there was no occurrence of any malignancy.