from the urethra grew no pathogen. He made a full recovery when treated with benzyl penicillin 1 mega unit intramuscularly six hourly.

His wife, who was without symptoms at this time, eventually agreed to be examined. She had cervicitis without erosion but no clinical evidence of urethritis. Cultures, however, grew the gonococcus from both sites.

This family demonstrates once again the well-known latency of gonorrhoea in women, and it also seems very likely that the condition had remained latent in the husband until septicaemia ensued though no proof of this can be obtained. The most unusual finding was the development of one of the rarest of the complications of gonorrhoea—namely, gonococcal septicaemia in both husband and wife. We have not been able to find a similar example recorded in the literature.—We are, etc.,

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V. H. PATTERSON

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¹ Oates, J. K., *British Medical Journal*, 1971, 3, 580.

How Infectious is Gonorrhoea?

SIR,—Dr. A. S. Wigfield (16 December, p. 672) provides some evidence that gonorrhoea is a highly infectious disease and that there is little difference in the infectivity rates for males and females. This is remarkable when one considers that gonococci implanted on the cervix are not exposed to the considerable fluid flows present in the male urethra during micturition. Further, the cases described by Dr. W. K. E. Bernfield (21 October, p. 173) show that within an hour after intercourse gonococci can be anchored to the urethral mucosa and are able to resist the flow of urine. In a recent electron-microscope study of human gonorrhoea¹ we have demonstrated the specific adherence of gonococci to cells from the urethral mucosa (see fig.). This attachment can explain the ability of the gonococcus to maintain itself in the urethra despite micturition.

Currently we are investigating the mechanism of this attachment. Freshly isolated

gonococci possess long hair-like filaments, pili, which can be seen radiating from the cells on electron microscopy.² On subculture in the laboratory these gonococci mutate to lose their pili. Our preliminary results show that gonococci with pili have a much greater ability to attach to human fibroblasts than the non-piliated mutants of the same strain. Furthermore, a scanning electron-microscope study of human Fallopian tubes maintained in an organ bath during perfusion with pilated gonococci has revealed organisms apparently anchored down to the epithelial surface by their pili. It would seem likely, then, that the critical factor in the high infectivity of gonorrhoea is the ability of pilated gonococci to attach to mucosal cells. Nevertheless, the non-pathogenic neisseria which grow on mucosal surfaces are also pilated,³ suggesting that other factors, such as resistance to host defence systems,⁴ must be important in the virulence of the gonococcus.—We are, etc.,

M. E. WARD

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- ¹ Ward, M. E., and Watt, P. J., *Journal of Infectious Diseases*, 1972, 126, 601.
- ² Swanson, J., Kraus, S. J., and Gotschlich, E. C., *Journal of Experimental Medicine*, 1971, 134, 886.
- ³ Wistreich, G. A., and Baker, R. F., *Journal of General Microbiology*, 1971, 65, 167.
- ⁴ Ward, M. E., Watt, P. J., and Glynn, A. A., *Nature*, 1970, 227, 382.

Glove-powder Peritonitis

SIR,—Following the publication of my letter on glove-powder peritonitis (17 July 1971, p. 183) and a scientific paper by Mr. Julian Neely and Mr. J. Douglas Davies (11 September 1971, p. 625) considerable correspondence has been received indicating that this type of peritonitis is much more common than was supposed. It seems that there is no substitute for the corn starch used, and the only way of avoiding this complication is to avoid the corn starch. One of the principal glove manufacturers kindly produced for a trial period gloves prepared

without the use of the corn-starch powder, which is normally applied as a slurry at the end of the manufacturing process. These gloves, gamma-ray sterilized, were a little more difficult to apply because it was necessary for the hands to be really dry. However, they not only eliminated completely the powder risk, but were a tremendous improvement in quality and did not become sticky in use; the grip was very much more satisfactory at the end of a long period of operating. These gloves were tried by various surgeons in different parts of the country and unfortunately the consensus of opinion was against their use on account of the difficulty of application. The manufacturers will therefore not proceed with this project as a commercial undertaking. For those who cannot dry their hands adequately, the difficulty of application could be overcome by the use of Bio-Sorb cream, a corn-starch preparation (which can be supplied in sachets). This avoids the use of scattered powder. It seems incredible that this risk to patients, acknowledged as very real, has got to continue because of the additional expense of manufacturing safe gloves.

There is little doubt that now we are aware of the risk of corn-starch peritonitis legal liability, in the event of this developing, must fall fairly and squarely on the surgeon and the manufacturers. Rubbing the gloves with a swab wetted with cetrimide removes much of the powder, but even after such a vigorous application and rinsing there is still a considerable amount remaining ingrained in the surface of the glove, as shown by a subsequent rinsing. A quick rinse alone is utterly insufficient. The only way of avoiding the risk is the elimination of powder in manufacture. Surely commercial considerations must be overcome.—I am, etc.,

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Pulmonary Oedema in Pulmonary Thromboembolism

SIR,—One of the reasons for the poor diagnostic rate¹ in the very common² condition of pulmonary thromboembolism is the variety of ways in which it may be manifest. Your report (30 December, p. 773) of an excellent clinicopathological conference on heart failure in a middle-aged woman contains a statement by Dr. Celia Oakley that "pulmonary embolism . . . does not cause pulmonary oedema." Since the appearance unchallenged of this statement in your columns suggests that this is a commonly accepted view, we wish to draw attention to the considerable contrary experimental and clinical evidence.

In 1942 Megibow *et al.*³ demonstrated that pulmonary oedema frequently complicated experimental pulmonary embolization in dogs. Dexter in 1965⁴ observed that in patients with preexisting heart disease the only manifestation of pulmonary embolism may be a subtle worsening of cardiac function, often presenting itself as pulmonary congestion or occasionally as florid pulmonary oedema. During the past few years we have had the opportunity to study 11 patients who presented with pulmonary oedema that was shown after full investigation, including haemodynamic measurements and pulmonary angiography, to be due to

FIG. 1.—Electron micrograph showing gonococci (G) closely adherent to the surface of a urethral epithelial cell (Epi) from a male patient with early symptomatic gonorrhoea. The bar represents 500 nm ($\times 23,620$). (Reproduced from the *Journal of Infectious Diseases* by permission of the University of Chicago Press.)

thromboembolism. Four of these patients had normal pulmonary "wedged" pressures (indirect left atrial pressures) and in one there was a slight increase. In the remaining six patients the pulmonary "wedged" pressure record could not be analysed because of difficulty in identifying an appropriate wave form. This could have been due to technical imperfection, but an alternative explanation might be pulmonary venous hypertension without left atrial hypertension. The occurrence of pulmonary venous constriction following experimental pulmonary embolism was demonstrated by Daicoff *et al.*⁵ This mechanism could account for pulmonary oedema in the absence of left heart failure and might also account for the difficulty in obtaining an adequate pulmonary "wedged" record.

Although in man the mechanism is obscure, there is no doubt that pulmonary oedema can occur as a result of pulmonary thromboembolism.—We are, etc.,

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- 1 Sasahara, A. A., Cannilla, J. E., Morse, R. L., Sidd, J. J., and Tremblay, G. M., *American Journal of Cardiology*, 1967, 20, 10.
- 2 Smith, G. T., Dexter, L., and Dammin, G. J., in *Pulmonary Embolic Disease*, ed. A. A. Sasahara and M. Stein, p. 120. New York, Grune and Statton, 1965.
- 3 Megibow, R. S., Katz, L. N., and Steinitz, F. S., *Surgery*, 1942, 11, 19.
- 4 Dexter, L., *Bulletin of the New York Academy of Medicine*, 1965, 41, 981.
- 5 Daicoff, G. R., Ranniger, K., and Moulder, P. V., *Surgical Clinics of North America*, 1968, 48, 71.

Pathology of Malignant Hyperpyrexia

SIR,—There are two minor inaccuracies which should be corrected in your otherwise excellent leading article (3 February, p. 249).

Firstly, malignant hyperpyrexia was first recognized as a complication of general anaesthesia in the 1930s.¹ At that time ether was probably the agent most frequently responsible. Secondly, Bradley and Murchison² studied muscle biopsy specimens from six patients at risk in two families, not four as stated. At a recent meeting of the Royal Society of Medicine³ the pathological and biochemical studies performed on malignant hyperpyrexia were discussed in greater detail.—I am, etc.,

W. G. BRADLEY

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Newcastle upon Tyne

- 1 Guedel, A. E., *Inhalation Anaesthesia; a Fundamental Guide*, p. 133. New York, MacMillan, 1937.
- 2 Bradley, W. G., and Murchison, D., *British Medical Journal*, 1972, 4, 108.
- 3 *Proceedings of the Royal Society of Medicine*, 1973, 66, 63.

Thiocyanate Metabolism in Human Vitamin B₁₂ Deficiency

SIR,—The letter from Dr. A. G. Freeman (27 January, p. 231) has reminded me of an article entitled "Optic Atrophy and Pernicious Anaemia; with Special Reference to Sex Distribution and Aetiology," published in 1951.¹ Two cases were described and the sex, age of onset, and smoking habits in 21 others reported in the literature were analysed. Of the total, 22 patients were males; in the only female the diagnosis was

in doubt. A positive history of smoking was obtained from 11 males; in the remaining cases no details were given. In the paper attention was drawn to the fact that this rare complication of pernicious anaemia predominantly affects males, and the aetiology of this chronic form of retrobulbar neuritis was discussed with special reference to the role of tobacco.

Its publication in an obscure and short-lived journal (under my editorship) provides ample reason why this article has passed unnoticed.—I am, etc.,

DOUGLAS MCALPINE

Marnhull, Dorset

- 1 McAlpine, D., and Goldsmith, A. J. B., *Archives of the Middlesex Hospital*, 1951, 1, 109.

Medical Aspects of Ambulance Design

SIR,—I was very interested in the article on ambulance design by Dr. Roger Snook (2 September, p. 574). Recent experiences have underlined what he says about the motion of the vehicle affecting the condition of some patients and the inadequacies of many vehicles in respect of patients requiring life-support treatment. It would seem that giving a shocked patient a ride in a modern ambulance may well finish him off. How often do we read in the paper "The patient died on the way to hospital"? I know there are many problems attached, and money is not the least of them, but in this age of science could we not design an ambulance?—I am, etc.,

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Staffing of Accident and Emergency Departments

SIR,—I was interested by the suggestion of Mr. J. C. Scott (3 February, p. 292) that casualty work should be an essential prerequisite for entry into general practice. Had such service not been a requirement for the F.R.C.S., then the staffing crisis of casualty departments would have occurred many years sooner, and such improvements in the conditions of work of casualty officers as have taken place recently would have been inevitable 20 years ago. Accident departments should be staffed by doctors keen to work there, not by men compelled to do six months' servitude in inadequate departments with inadequate training and cover.—I am, etc.,

R. K. HOLDSWORTH

Rugby

SIR,—My friend Mr. P. A. M. Weston (13 January, p. 114) must have been living in Tannochbrae if he believes that the typical family doctor's work consists in caring for the patient as a whole and in continuity. The stresses put upon the general practitioner by the open-ended commitment of the N.H.S. have tended to have quite the opposite effect. Many doctors will reluctantly agree that they have time only to treat the presenting symptom. With regard to continuity, witness the large group practice where, out of office hours, the patient must be content with the service of any doctor. Witness the town doctor system where on a Wednesday after-

noon, one doctor is on call for the community. And witness the lock-up surgeries where the agency doctor has no access to the patient's records.

Mr. Weston agrees that G.P.s are suitable to staff small hospital accident units. This is a move in the right direction but, as I argued in my letter (9 December, p. 607), I believe that the only solution to the staffing problem is the universal use of doctors of this genre.

Mr. Weston's main point is that senior staff (and I presume he means casualty consultants) should be in the bigger accident departments to train the G.P.s. I believe that all departments need doctors with the G.P.-type of training who will be sensible enough to call upon the skills of the consultant staff to deal with the seriously ill or injured. No special training is necessary beyond that learned as an undergraduate and as a house surgeon, except perhaps some advanced first-aid techniques such as the confident passage of an endotracheal tube.—I am, etc.,

J. P. TURNEY

Whitehaven, Cumberland

Disappearance of Diphtheria

SIR,—We appreciated your leading article on "Infectious Disease" (13 January, p. 63) and welcome the series it introduces. In the light of our experience in a laboratory with a special interest in diphtheria we feel that the observations on that disease merit some further comment. It is a common assumption that the disappearance of diphtheria was due to immunization, but we do not know why diphtheria declined so rapidly, nor do we know what part was played by changes in the prevalent types of strain.

The isolation of atypical and non-toxicogenic strains first became fairly common about 1935, before either extensive immunization or chemotherapy could have played a major role. Two changes are noteworthy. There has been a steady fall in the incidence of virulent strains of each type isolated¹ and a change in the incidence of the biotypes, so that today endemic *mitis* strains, the majority of which are non-toxicogenic, are commonly found, but epidemic *gravis* strains are uncommon and *intermedius* is rarely seen.

There is a familiar ring about the prediction that with continuing immunization diphtheria must cease to exist. The hope expressed by Logan² in 1952 that diphtheria could disappear within 10 years was not realized. If we examine the situation in the United Kingdom, we find that the most vulnerable group, the underprivileged, have the poorest acceptance rate of immunization.

Experience in areas such as Alberta, Canada, drew attention to the presence of diphtheria bacilli in the skin of a section of the population, with occasional cases of fatal or classical diphtheria in their associates. Moreover, some of the patients with typical toxic diphtheria have apparently been fully immunized.^{3,4} Although the declining world incidence of classical diphtheria is a hopeful sign, we must remember that as the incidence of the disease has fallen, the mortality rate has risen steadily from 5% in the 1930s to 20% in 1970 in the United Kingdom. Since many laboratories became aware of the importance of looking for the organism, it appears that the diphtheria