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"Twin" Intracranial Aneurysms

SIR,—We are prompted by Mr. B. Fairburn's memorandum (27 January, p. 210) on the occurrence of aneurysmal haemorrhage in monozygotic twin sisters to report two similar cases of our own.

Case 1.—A woman aged 42 years was referred to one of us (P.J.E.W.) after proved, coma-producing subarachnoid haemorrhage on 3 September 1969. The next day, though still drowsy, inert, and photophobic, she had no focal neurological deficits. Obesity and labile hypertension were noted. An electrocardiogram showed left bundle-branch block, sinus bradycardia, and T-wave depression in leads II, III, and aVF. Carotid angiography showed a small lobular aneurysm of the left middle cerebral artery (fig. 1) and an "infundibulum" (arguably

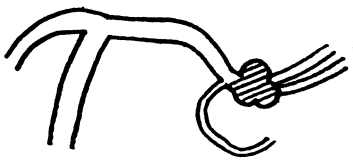

FIG. 1.—Case 1. Anteroposterior view, left carotid angiogram (tracing).

a second small aneurysm) at the origin of the right posterior communicating artery. There was no clot or vasospasm. Three days later, after direct-puncture vertebral angiography (with normal findings), she suddenly became aphasic with right hemiplegia, though retaining full consciousness. Recovery occurred during the next few days. An expectant regimen, with hypotensive medication, was followed and she has remained neurologically well.

Case 2.—This woman was referred to one of us (I.P.C.) at the age of 45 years after proved, non-coma-producing subarachnoid haemorrhage on 26 November 1972. She was the identical twin of the previous patient (but formal haematological substantiation of monozygosity was not made). She was a known hypertensive and had had toxaemia of pregnancy. Two days after her haemorrhage she was alert and free of focal neurological signs, though both plantar responses were extensor. Carotid angiography showed a small saccular aneurysm of the left middle cerebral artery (fig. 2) and an "infundibular"

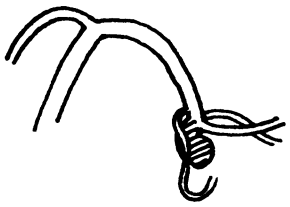

FIG. 2.—Case 2. Oblique view, left carotid angiogram (tracing).

origin of the right posterior communicating artery. Vertebral angiography and excretion pyelography were normal. Again an expectant regimen was advised. Her hypertension was somewhat resistant except to large doses of methyldopa and clonidine. On 23 December 1972 she had a second subarachnoid haemorrhage and four days later a further sudden, rapidly fatal haemorrhage. The diagnosis was confirmed at necropsy but detailed neuropathological studies are not yet complete.

The aneurysms were more closely congruous in our cases than in Mr Fairburn's and were at one of the commoner sites. Both our patients were hypertensive, as was their mother. Their father had died 15 years previously of "cerebral haemorrhage." Whether the hypertension was in any way linked with the haemorrhages, either as an immediate precipitant or as a factor in the development of the aneurysms, is conjectural. Whether, in the light of the fate of her sister, our first patient's expectant regimen should be abandoned for a more aggressive surgical approach now poses an unusual clinical dilemma.

The incidence of aneurysmal subarachnoid haemorrhage in a closed community of 400,000 was found by Pakarinen[1] to be 13 per 100,000 per annum. The incidence in the population at large cannot be computed with precision, but synthesis of the Registrar General's statistics[2] and published data of large series of proved cerebral aneurysms[3 4] enables certain broad predictions to be made, assuming chance to be the sole operating factor. Thus an angiographic search of the population of England and Wales over the age of 30 years could be expected to yield eight pairs of male and 37 pairs of female monozygotic twins with at least one cerebral aneurysm. In any pair of female monozygotic twins of whom one has an aneurysm the chance of the other having an aneurysm at any site would be 1 in 50 (that is, an order of risk comparable with that of the female population at large). The chances of each twin having an uncommon (for example, "carotid-ophthalmic") aneurysm[5], as in Mr. Fairburn's cases, would be of the order of 1 in 1,000; and of each twin having a common (for example, middle cerebral) aneurysm they would be of the order of 5 in 1,000. It can be predicted that in the U.K. as a whole, one case of twin aneurysmal subarachnoid haemorrhage in females should come to light every year, and one such case in males every four years (D. J. B. Ashley, 1973, personal communication).

It seems reasonable to postulate a transmissible genetic factor in some cases of familial subarachnoid haemorrhage, especially when kinship is close[6] and the aneurysms congruous. Not only are the aetiological implications of twin aneurysms of obvious importance, but the possibility, in sibs "at risk," of *elective* investigation and surgical prophylaxis[7 8] cannot be overlooked. These are further reasons for neurosurgeons to pool their case material in this field.—We are, etc.,

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SIR,—Mr. B. Fairburn (27 January, p. 210), reporting "twin" intracranial aneurysms causing subarachnoid haemorrhage in a pair of monozygous "twins," concludes that some common genetic factor was involved. I would suggest that this is a good example of "mirror-imaging," common in identical twins (in about 30%), which results from the early twinning-division of the zygote (fertilized ovum).—I am, etc.,

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Gonococcaemia in the Family

SIR,—The presentation of gonorrhoea in a family has previously been reported[1] and since then a more unusual family has come our way.

A healthy, 23-year-old West Indian woman gave birth to a baby boy. Four days later she developed a maculopapular eruption on the legs, arms, and buttocks; arthritis of the left knee and ankle; and a pyrexia of up to 102°F (38·9°C). Gram-negative intracellular diplococci were found in a smear taken from the urethra though they were not grown in culture. A clinical diagnosis of gonococcal septicaemia was made and she was treated with procaine penicillin intramuscularly and made a rapid recovery. At the same time her baby developed ophthalmia, the gonococcus being found in both smear and culture.

She denied intercourse with anyone but her husband, who, as it happened, was being investigated in the same hospital for hypertension. He had had no urinary symptoms or urethral discharge and swabs from the urethra were negative for the gonococcus. He was given no treatment. Three months later the husband was admitted to hospital with a three-day history of rash, joint pains, and shivering attacks. His temperature was 103°F (39·4°C), and there was a scanty erythematous and vesicular rash on his trunk, arms, and legs. The right knee joint and right wrist were hot, swollen, and very tender, the former containing a sizeable effusion. He denied any previous genitourinary symptoms and there was no clinical evidence of urethral discharge. He did not admit to any extramarital relationship. *Neisseria gonorrhoeae* were isolated from a blood culture though not from a specimen of the effusion in the knee joint. A swab

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 urthritis. 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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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 litle 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 gonorrhoea[1] 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-pilated 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.,

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1 Ward, M. E., and Watt, P. J., *Journal of Infectious Diseases*, 1972, **126**, 601.
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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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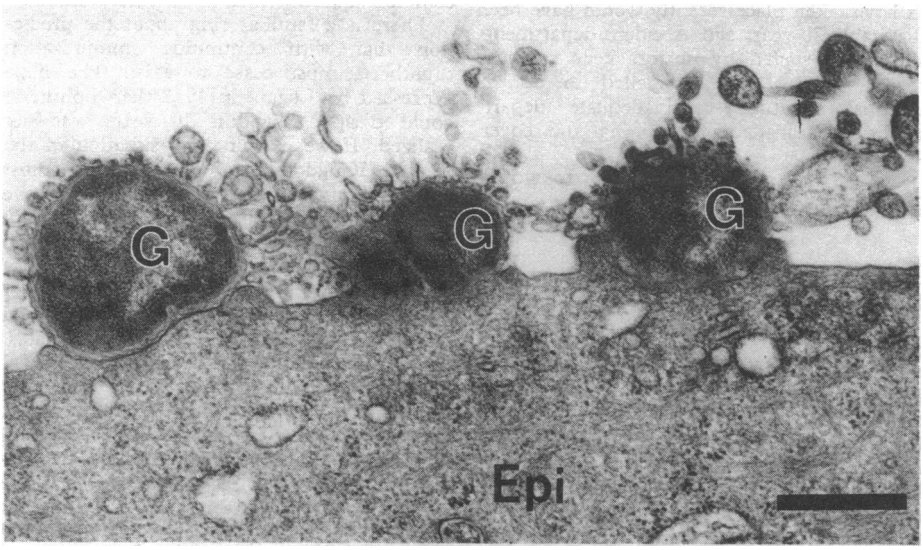

FIG. 1.—Electron micrograph showing gonococci (G) closely adherent to the surface of a urethral epithelial cell (Epi) from a male patient with early symptomatic gonorrhoea. The bar represents 500 nm (× 23,620). (Reproduced from the *Journal of Infectious Diseases*[1] by permission of the University of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

Pulmonary Oedema in Pulmonary Thromboembolism

SIR,—One of the reasons for the poor diagnostic rate[1] in the very common[2] 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 1965[4] 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