1 Griffith, C. A., and Harkins, H. N., *Gastroenterology*, 1957, **32**, 96.
2 Amdrup, B. M., and Griffith, C. A., *Annals of Surgery*, 1969, **170**, 207.
3 Interone, C. V., Del Finado, J. E., Miller, B., Bombeck, C. T., and Nyhus, L. M., *Archives of Surgery*, 1971, **102**, 43.
4 Klempa, I., Holle, F., Brückner, W., Welsch, K. H., Handle, H., and Von Wolff, A., *Archives of Surgery*, 1971, **103**, 713.
5 Amdrup, B., Steinoe, K., and Bille-Brahe, N., *Acta Chirurgica Scandinavica*, 1971, **137**, 794.
6 Humphrey, C. S., and Wilkinson, A. R., *British Journal of Surgery*, 1972, **59**, 779.
7 Clarke, R. J., and Williams, J. A., 1972, *British Journal of Surgery*, **59**, 903.
8 Pedersen, G., and Amdrup, E., In press.
9 Wilkinson, A. R., and Johnston, D., *Annals of Surgery*. In press.
10 Gunn, I. F., and Williams, J. A. 1973, paper to Surgical Research Society.
11 Johnston, D., and Wilkinson, A. R., *British Journal of Surgery*, 1970, **57**, 289.
12 Everett, M. T., and Griffith, C. A., *Annals of Surgery*, 1970, **171**, 31.
13 Shiina, E., and Griffith, C. A., *Annals of Surgery*, 1969, **169**, 326.
14 Korman, M. G., Hansky, J., and Scott, P. R., *Gut*, 1972, **13**, 39.
15 Korman, M. G., Hansky, J., Coupland, G. A. E., and Cumberland, V. H., *Gut*, 1972, **13**, 163.
16 Wyllie, J. H., Stagg, B. H., Lewin, M. R., and Clark, C. G., 1973. paper to Surgical Research Society.
17 Hayes, J. R., Kennedy, T. L., Ardill, J., and Buchanan, K. D., 1973, paper to Surgical Research Society.
18 Johnston, D., Humphrey, C. S., Smith, R. B., and Wilkinson, A. R., *British Journal of Surgery*, 1971, **58**, 725.
19 Humphrey, C. S., Smith, R. B., Lyndon, P. J., and Johnston, D., *British Journal of Surgery*, 1972, **59**, 906.
20 Hall, R., Humphrey, C. S., Wilkinson, A. R., and Johnston, D., *British Journal of Surgery*, 1971, **58**, 273.
21 Fawcet, A. N., Johnston, D., and Duthie, H. L., *British Journal of Surgery*, 1969, **56**, 111.

"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

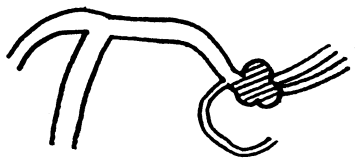

FIG. 1.—Case 1. Anteroposterior view, left carotid angiogram (tracing).

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"

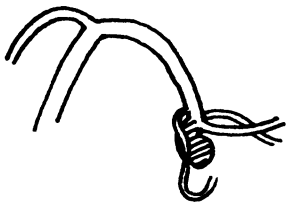

FIG. 2.—Case 2. Oblique view, left carotid angiogram (tracing).

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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1 Pakarinen, S., *Acta Neurologica Scandinavica*, 1967, **43**, suppl. 29.
2 Registrar General, *Statistical Review of England and Wales for the Year 1968; part I; Tables, medical*. London, H.M.S.O., 1970.
3 Locksley, H. B., *Journal of Neurosurgery*, 1966, **25**, 219.
4 Richardson, A. E., *British Medical Journal*, 1969, **4**, 89.
5 Kothandaram, P., Dawson, B. H., and Kruyt, R. C., *Journal of Neurosurgery*, 1971, **34**, 544.
6 Chakravorty, B. G., and Gleadhill, C. A., *British Medical Journal*, 1966, **1**, 147.
7 Mount, L. A., and Brisman, R., *Journal of Neurosurgery*, 1971, **35**, 728.
8 Moyes, P. D., *Journal of Neurosurgery*, 1971, **35**, 291.

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