Editorial

MENINGEAL HÆMORRHAGE

In the nature of things it does not appear likely that meningeal hæmorrhage can ever alter its aspects and in some obscure way become different from what it always has been. As regards its analogue, cerebral hæmorrhage, with which indeed it is very often found in unison, no conspicuous change has come over our conceptions of its pathogenesis and types, although no doubt knowledge of its mechanism in some particular circumstances has of late been amplified. But in respect of the other an increasing number of case-reports are being published entitled 'Subarachnoid hæmorrhage,' with or without the epithet 'spontaneous' to qualify it. Older expressions such as 'meningeal apoplexy' are losing or have lost their vogue, and the idea would seem to be gaining ground that the subarachnoid variety is a disease-condition per se, with clinical features of its own, independent of other states, and recognisable at sight, so to speak, by the recovery of blood from the cerebrospinal fluid. No doubt the chief, possibly the sole, reason for this is the ease with which lumbar puncture is performed and with which effusion within the theca can be detected. Yet this facility is apt to prove misleading, for it imparts a spurious simplicity to the subject, one which in reality it is far from possessing.

Another possible explanation of the rise into prominence of this topic is related to the occurrence of so-called congenital aneurysms on cerebral vessels and to their disclosing themselves by rupture and extravasation of blood through the subarachnoid system. That these lesions appear to develop with greater frequency than was formerly supposed is not merely a plausible surmise, but receives support from actual examination. For instance, among 124 cases of subarachnoid hæmorrhage collated by Symonds in 1924 a ruptured aneurysm was discovered in only 11, but since that time the percentage has risen curiously; no less than 15 of 16 cases autopsied by Biemond and ter Braak (1933) were verified as having been thus produced. The natural consequence seems to be that a
stage has been reached where bleeding under the arachnoid, of sudden onset perhaps, though not invariably, and particularly when it occurs in persons under the age of 50, is assigned to congenital aneurysm without a thought of any other interpretation. But the facts militate strongly against so artless a view. Whereas no signs of hypertension or arteriosclerosis were observed by the Dutch neurologists mentioned above in their series of cases, exactly the opposite was noted by Strauss and his colleagues (1932), who in every one of 11 cases of subarachnoid hæmorrhage confirmed postmortem—though the average age of their patients was no higher, if not indeed actually lower, than that of the subjects examined by Biemond and ter Braak—found signs of either local or generalized arteriosclerosis, with aneurysmal formation in seven of the series. In other words, the lesion is attributed by the former to congenital aneurysm, by the latter to arteriopathic disease of a different kind altogether. Here is a clinical and pathological conflict which cannot easily be explained away.

Concentration of attention on the subarachnoid variety of meningeal apoplexy and on congenital aneurysm has been conducted at the expense of pathological principles and clinical observation. When blood is found in the spinal fluid the mere fact provides no clue to its provenance. Blood can come from arteries, veins, capillaries, and sinuses; from any of these it may leak, and in different directions. From without inward meningeal hæmorrhage might be schematically classified as extradural, dural, subdural, subarachnoid, pial, and subpial respectively; while not all are of equal importance or frequency, some are likely to be combined. To what extent any may occur strictly by itself is a point on which knowledge is vague, and sorely in need of statistical amplification. The subarachnoid variety, like all meningeal effusions, is merely a complication of some other lesion. Blood within the membranes is nothing else than an incident in a process whose seat may be remote and character diffuse. Subdural hæmorrhage often bursts through the fragile arachnoid, which may be torn, too, by blood originally extravasated underneath it. Further, bleeding may start under the pia and escape into the arachnoid system. To diagnose subarachnoid hæmorrhage, therefore, because of the presence of blood in puncture fluid, is in a superficial sense correct, but in no other; it is to mistake a sign for the lesion, a symptom for the process, a termination for a cause. And even when the condition is diagnosed by the spinal fluid, of itself it can furnish no information as to the underlying
morbid state. *Pace* the simplifiers of the day, it tells us exactly nothing in respect of the latter. The symptoms and signs of intracranial aneurysm are not those of its rupture, which may or may not take place. And numerous other lesions are liable to be overlooked when aneurysm is complacently regarded as the sole cause of the condition. For example, syphilis and a variety of other infections, different kinds of meningitis, alcoholism, heatstroke, sinus occlusion, and still other states are all likely enough to induce subarachnoid bleeding. The first sign of an otherwise symptomless cerebral tumour may be escape of blood. Of no little interest, too, is the apparent association of migrainous attacks with subarachnoid effusion. To ascribe these last-mentioned symptoms to congenital aneurysm, as is often done to-day, encounters many obstacles; it is hard to understand how the process should continue for years, sometimes many years, before culminating in hæmorrhage, and equally so to account for the migrainous type induced if, as is customary, the aneurysmal lesion is at the base. When not verified postmortem the inference that aneurysm has been the cause of both ‘migraine’ and subarachnoid effusion may be altogether mistaken.

Claims, however, have been advanced to exclude the foregoing causes of subarachnoid hæmorrhage by alleging that the type for which the term should be used and to which it should be restricted can be distinguished as being at once ‘spontaneous’ and ‘massive.’ Both considerations are erroneous. The former adjective is at times employed to-day as though it were identical with or equivalent to ‘causeless,’ which is absurd. Though extrinsic factors may seemingly be lacking, intrinsic factors can not; the condition of the vessel wall, which cannot be foretold, governs the situation; the coats give way not ‘spontaneously,’ but because they are compelled to do so. If synonymous with ‘non-traumatic’ the question of what constitutes ‘trauma’ has first to be answered. As for the other qualifying term, it cannot be defined; who shall say, and on what criterion shall he rely in asserting, that an effusion is ‘massive’? Aside from this crux, every degree and grade of effusion take place even in aneurysm, if we are to believe those who affirm that aneurysms leak and give rise to no more than headache and giddiness at one time, but inundate the arachnoid system at another.

The usefulness and clinical value of the subarachnoid conception, then, would seem to be seriously impaired if it is made too exclusive, and if it is regarded as standing for a
morbid entity. But if the form is put in its proper place, as a manifestation of a process of varying causation, and if it is realized to be often nothing more than a part of meningeal and cerebral haemorrhage in general, it becomes a matter of genuine significance, less a thing-in-itself than a clue to what has been going on within the skull.