cies shown emphasize the complementary nature of each procedure. In the assessment of patients with lung disease for surgery, as opposed to physiological research, the bronchoscopic technique is the more useful because it is on an anatomical lobar or segmental basis, rather than being topographical like the xenon, and because it is possible not only to measure the distribution of the tidal volume into different lobes but to assess the ventilation and blood-flow balance in them, as well as getting information about the mechanical changes in the lung at a lobar level (Hugh-Jones and West, 1960 ; McGrath and Hugh-Jones, 1963). The radioactive-gas studies, along with whole-lung tomographs, afford a valuable screening procedure, for the detection of areas with a poor blood-flow, before bronchoscopy.

A major difficulty has been the selection of patients suitable for operation. The recognition of the degree to which chronic bronchitis contributes to a patient's disability has been of the utmost importance. Further, the contribution of this factor to post-operative mortality has been indicated. We would consider any form of untreated bronchitis a contraindication to assessment or recommendation for surgery. On the other hand, we have shown that, in spite of gross disability and generalized emphysema, dramatic subjective and functional improvement may occur.

A further difficulty has been the assessment of the surgical result due to the variation in the procedures performed. As the appearances at thoracotomy do not necessarily confirm the expected radiological or regional functional findings, decision regarding the most suitable procedure must be decided when the lung is exposed. For this reason no clear recommendation for the type of procedure can be given at present.

Further investigation of mechanical events in the lung before and after surgery is required, particularly their relation to the presence of tracheobronchial collapse as observed on the spirogram and at bronchoscopy. Some standardization of the type of operation is clearly necessary in addition to careful pathological study of resected material to correlate the type of emphysema with the functional result.

Summary

An investigation was begun to assess the possible surgical alleviation of "irreversible" airways obstruction due to emphysema. Fifty-two patients were studied by clinical and radiological examination, together with general and regional lung-function tests. Twenty-four patients were submitted to operation for resection or plication of bullae or localized transradiant areas. Nine patients showed clear evidence of functional improvement. Subjective improvement correlated most closely with the simple ventilatory tests, though isolated cases showed more improvement in other parameters. A notable finding was the improvement in five out of six patients who had basal lesions. The problems of selection of patients, the type of operation, and the significance of the functional results are discussed.

We wish to thank members of the Medical Research Council's Cyclotron Unit at Hammersmith Hospital for their expert help with the ^{133}Xe scans. Dr. John West was concerned with the examination of patients early in the series ; we are most grateful for his help and advice. Miss E. Douglas and Nurse N. Lamb gave invaluable assistance with lung-function tests and the bronchoscopic procedures, while Mr. Len Smith and Mr. John Holden were responsible for maintaining the complex equipment, including the mass spectrometer, which did not fail during any bronchoscopic procedure. We are grateful to the physicians and surgeons from different hospitals who referred patients under their care to us for assessment, especially those at the Brompton Hospital, St. George's Hospital, Midhurst Sanatorium, St. Helier Hospital, and the Milford Chest Clinic. We are indebted to our surgical colleague, Mr. A. M. Macarthur, for his interest and cooperation in performing the surgery on most of the patients more recently in the series.

References

Bernstein, L., D'Silva, J. L., and Mendel, D. (1952). *Thorax*, **7**, 255.
Bower, G. (1961). *Amer. Rev. resp. Dis.*, **83**, 894.
Campbell, E. J. M. (1958). *Respiratory Muscles and the Mechanics of Breathing*. Lloyd-Luke, London.
Dollery, C. T., and Gillam, P. M. S. (1963). *Thorax*, **18**, 316.
—— and Hugh-Jones, P. (1963). *Brit. med. Bull.*, **19**, 59.
Fenn, W. O., Rahn, H., and Otis, A. B. (1946). *Amer J. Physiol.*, **146**, 637.
Fletcher, C. M., Hugh-Jones, P., McNicol., M. W., and Pride, N. B. (1963). *Quart. J. Med.*, **32**, 33.
—— and Tinker, C. M. (1961). *Brit. med. J.*, **1**, 1491.
Gaensler, E. A. (1962). *Ann. Rev. Med.*, **13**, 319.
Gandevia, B. (1963). *Quart. J. Med.*, **32**, 23.
Gilson, J. C., and Hugh-Jones, P. (1949). *Clin. Sci.*, **7**, 185.
Goldman, H. I., and Becklake, M. R. (1959). *Amer. Rev. Tuberc.*, **79**, 457.
Goodwin, J. F., and Abdin, Z. H. (1959). *Brit. Heart J.*, **21**, 523.
Hamer, N. A. J. (1962). *Clin. Sci.*, **23**, 85.
Hugh-Jones, P. (1963). In *Symposium Chronic Respiratory Disorders*, p. 30. R.C.P. Edinburgh.
—— and West, J. B. (1960). *Thorax*, **15**, 154.
Jones, N. L. (1964). M.D. Thesis, London University.
Jones, R. S., and Meade, F. (1960). *Lancet*, **1**, 94.
Kory, R. C., Callahan, R., Boren, H. G., and Syner, J. C. (1961). *Amer. J. Med.*, **30**, 243.
—— Smith, S., and Callahan, R. (1966). To be published.
Lloyd, B. B. (1958). *J. Physiol. (Lond.)*, **143**, 5p.
McGrath, M. W., and Hugh-Jones, P. (1963). *Clin. Sci.*, **24**, 209.
Medical Research Council (1960). *Brit. med. J.*, **2**, 1665.
Newman, F. (1962). Ph.D. Thesis, London University.
Ogilvie, C. M., Forster, R. E., Blakemore, W. S., and Morton, J. W. (1957). *J. clin. Invest.*, **36**, 1.
Severinghaus, J. W. (1958). *J. appl. Physiol.*, **12**, 485.
West, J. B. (1963). *Brit. med. Bull.*, **19**, 53
—— and Hugh-Jones, P. (1961). *J. appl. Physiol.*, **16**, 697.

Mechanism of Growth and Rupture in Cerebral Berry Aneurysms*

M. R. CROMPTON,† M.D., PH.D., M.C.PATH.

Brit. med. J., 1966, **1**, 1138–1142

It has long been accepted that arterial hypertension and its frequent concomitant, atheroma, are important factors in the initiation, growth, and rupture of cerebral berry aneurysms (Crawford, 1959). Certainly both clinical (McKissock *et al.*, 1959) and necropsy (Crompton, 1964) consecutive series have shown about 60% of patients with ruptured aneurysms to be hypertensive by comparable, if arbitrary, standards. This is higher than the prevalence of hypertension in a control group of similar age distribution. However, there has been little investigation of the actual mechanisms of growth and rupture, and it has been widely assumed that, like a blow-out in a bicycle inner tube, the aneurysm must rupture when the strength of its thinning wall can no longer withstand the tension in it. The wall of an aneurysm, unlike an inner tube, is a living and metabolizing structure, able to add to and reinforce itself. Enlargement does not necessarily imply thinning.

Crawford (1959) stated that most aneurysms ruptured at 6 to 15 mm. diameter, that 64% of ruptures were at the fundus

* This paper is a condensation of part of a Ph.D. thesis submitted to London University in 1965.

† Senior Lecturer and Honorary Consultant Neuropathologist, St. George's Hospital at Atkinson Morley's Hospital, London.

or apex of the sac, 10% lateral or in the middle third of the sac, and only 2% at the neck or proximal third of the sac. He described thin-walled "bubbles" on the surface of some aneurysms and thought that rupture often occurred through these bubbles.

Falconer (1954) said that the symptoms of massive rupture seldom accompany exertion. He suggested that exertion might initiate dissection of blood in the wall of the aneurysm which might later lead to seepage of blood from the aneurysm with recurrent headache, which could lead to terminal massive rupture. This theory is supported by Nyström (1963), who examined the walls of berry aneurysms under the electron microscope and saw red cells within them.

Krauland (1957) and Hassler (1961) found eosinophilic material resembling fibrin or fibrinoid in thin portions of the walls of some large and small aneurysms. This might occur together with inflammatory cells. They did not comment on the possible significance of this appearance, but Stehbens (1963) also saw it and suggested that it might indicate imminent rupture.

Wood (1964) stated that in over 80% of patients with multiple aneurysms it was the largest aneurysm on angiography that had ruptured. Jain (1963) made an interesting, and possibly important, observation in ipsilateral multiple aneurysms in which one lay distal to the other on the same artery or a branch. Studying 18 such cases, he found that the proximal aneurysms had ruptured in 12. Using rubber aneurysms in a fluid circuit with a pulsatile flow, he found that the pressure oscillation or variation distal to an aneurysm was less than it was proximal to the aneurysm. Possibly a proximal aneurysm acts as a damper or diminishes the pulse pressure, and therefore the distal aneurysm pulsates less and is less likely to enlarge or rupture. He found that his rubber aneurysms could stand much greater non-pulsatile rises of pressure than pulsatile rises, without rupturing. This is probably the reason for the protective value of carotid ligation, which reduces the pulsation distal to it, though the total distal blood-flow remains the same through the collateral supply.

Little evidence has been collected on the rate of enlargement of aneurysms. Repeated angiography is the only reliable method of assessing this, and it is not considered justified by many clinicians. Incidental evidence may be picked up at necropsy. Björkesten and Troupp (1962) reported that in 19 patients with berry aneurysms a second angiography was performed between two weeks and 10 years after the first. No surgery was performed in the interval. Ten aneurysms had increased in size, six of which had had a recurrent haemorrhage, and one had disappeared on the second angiogram. They concluded that aneurysms enlarged most rapidly after one or more haemorrhages from them.

Results

The brains of 289 patients dying consecutively from the rupture of a cerebral berry aneurysm were examined. One hundred and fifteen unruptured aneurysms of 2 mm. or more external diameter were also present in 90 of these patients. In other words, 31% of them had multiple aneurysms. Among these patients three had four aneurysms, 19 had three, and 68 had two.

Site of Rupture

In 271 of the 289 cases it was possible to determine on which portion of the aneurysm the rupture lay. The aneurysms were divided lengthwise from origin to fundus into three equal thirds. The proximal third adjacent to the parent artery was termed the *neck,* the middle third was the *body,* and the distal third was the *apex.*

The rupture had occurred through the apex in 227 cases, through the body in 38, and through the neck in only six. This confirmed Crawford's (1959) original findings, which are of surgical importance.

Multiloculation (or surface bubbles)

It was possible in 275 of the 289 cases to examine the ruptured aneurysms for the presence of more than one distinct loculus or secondary bubble on their surface. In 157 (57%) of these cases the aneurysms were multiloculated. Similarly in 112 of the unruptured aneurysms 18 (16%) were multiloculated. This might appear to suggest that multiloculation is associated with rupture; however, in the 50 unruptured aneurysms of 4 mm. or more external measurement 13 (26%) were multiloculated, suggesting that multiloculation is directly related to size. This is shown in Fig. 1 to be true also of ruptured aneurysms.

Size of Aneurysms at Time of Rupture

The maximum external diameters of the sacs of 273 ruptured and 115 unruptured aneurysms are shown in Fig. 2. It can be seen that the majority of ruptured aneurysms were about 5 mm. in their maximal external diameter. There is a steep rise up to this and a more gradual fall away to the larger

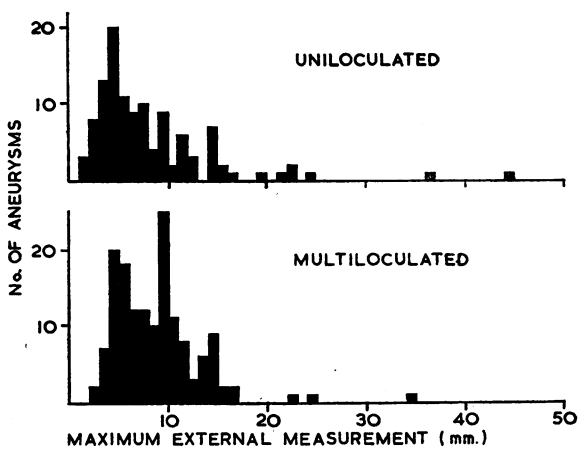

FIG. 1

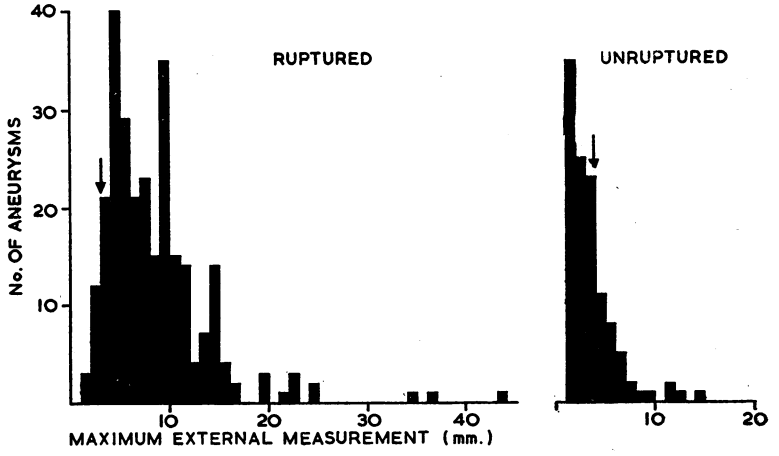

FIG. 2

FIG. 1.—Proportion of unilateral and multiloculated sacs among 275 ruptured aneurysms of varying maximum external fundal diameter. There were more multiloculated aneurysms among the larger ruptured aneurysms of 10 mm. maximal external diameter or over. FIG. 2.—Maximum external fundal diameter of 273 ruptured and 115 unruptured aneurysms.

measurements. Most of the unruptured aneurysms were the smallest measured, 2 mm., and there was a rapid fall off after this. This suggests that, as one might expect, aneurysms are most stable at the smallest external diameters and then become rapidly more unstable as they enlarge and are most likely to rupture at an external measurement of 5 mm. The *critical size* when an aneurysm would appear suddenly to become likely to rupture is between 2 and 5 mm., when about equal numbers of ruptured and unruptured ones are found. This is seen in Fig. 2 to be at the maximum external diameter of 4 mm. (arrowed).

Size of Aneurysms at Different Ages

Fig. 3 shows the maximum external diameter of 278 ruptured aneurysms plotted as points against the age of the patients in whom they occurred. It can be seen that most of the 10-mm. peak of ruptured aneurysms (see Fig. 2) occurred between 55 and 60 years, which is the peak age for ruptured aneurysms in general, and the other peak of 5 mm. was more spread out between the ages of 40 and 65 years.

Though no really large aneurysms of 25 mm. or more occurred under the age of 40 years, there was no convincing evidence of aneurysms tending to begin early in life and gradually enlarging so that the older patients ruptured the largest aneurysms. On the contrary, there was equally good evidence to suggest that aneurysms can begin at any age and enlarge rapidly.

Rupture of Multiple Aneurysms

It was found that in 79 (88%) of the 90 cases with multiple aneurysms the aneurysm of greatest maximum *external* diameter had ruptured. This supports Wood's (1964) angiographic findings.

In 36 cases there were two or more aneurysms on the same side and on the same artery or its branch. In 25 (70%) of these cases it was the proximal aneurysm which had ruptured. Of the 11 cases in which the distal aneurysm had ruptured seven were combinations of ruptured anterior communicating and unruptured internal carotid aneurysms. Thus, excluding anterior communicating aneurysms, there does appear to be a tendency in ipsilateral combinations of middle cerebral and internal carotid aneurysms for the proximal aneurysm to have ruptured first.

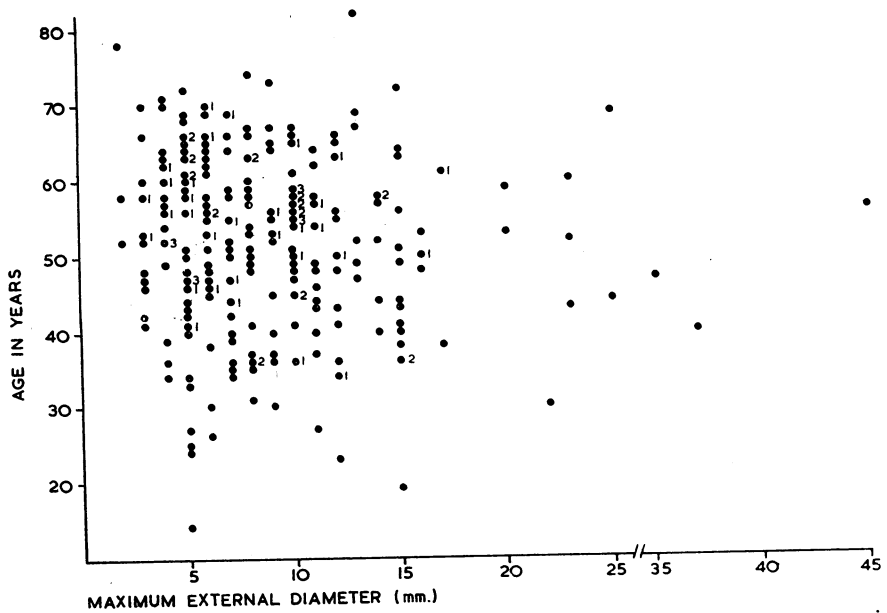

FIG. 3.—Distribution of maximum external fundal diameter of 278 ruptured aneurysms with age. The number against a dot indicates the number of identical measurements at that age.

Mechanism of Rupture.—It was noted in the histological sections of ruptured aneurysms stained by the picro-Mallory method that the wall of the aneurysm at the margin of the rupture always stained bright red as for fibrin. Further from the rupture only the inner part of the wall adjacent to the lumen was stained in this manner. Closer examination showed that in some instances the material staining red lay on the lumen side or internal to the endothelium, suggesting recent mural deposition, whereas in other cases it was covered by or external to the endothelium, suggesting subendothelial permeation with fibrin or endothelization of an original mural deposition. Further away still from the rupture the red staining ceased but numerous polymorphonuclear leucocytes, plasma cells, and small round cells lay beneath or external to the endothelium and also permeated deeply into the wall of the aneurysm (Fig. 4).

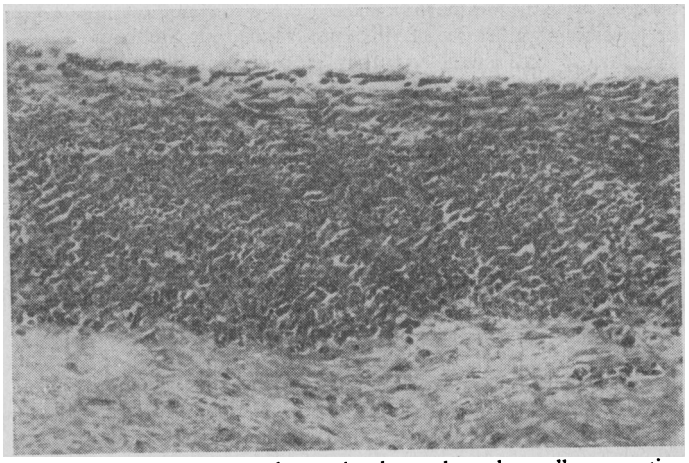

FIG. 4.—Abundant mononuclear and polymorphonuclear cells permeating the wall of an aneurysm adjacent to an apical rupture. The endothelium is above. (Picro-Mallory. ×110.)

At first it was thought that these changes were probably secondary to and occurred after rupture of the aneurysm. However, two cases were seen in which death occurred 9 and 12 hours after confirmed aneurysmal rupture, and the above-described changes were extremely prominent, rendering the apical one-third of both the aneurysms apparently necrotic. It was thought that these changes were too pronounced to have occurred in 9 and 12 hours and that they must have been present before the rupture of the aneurysm. It was therefore decided to examine a number of unruptured aneurysms of 2 mm. maximum external diameter or more to see if comparable changes occurred without or prior to the rupture of cerebral aneurysms.

Seventy-nine unruptured aneurysms were sectioned at several levels and examined. They varied in maximum external diameter from 2 to 12 mm.

Permeation of the aneurysm wall by polymophonuclear leucocytes and other cells (Fig. 5) occurred in 23 cases. In 18 of these it was at the apex. Fibrin stained red by the picro-Mallory method was present in 12 of the 23 cases with cellular infiltration of the wall. It did not occur alone, suggesting that the cellular infiltration was an earlier stage in the process. In some instances the fibrin was covered by endothelium, and in others not. In a few it was deep in the wall of the aneurysm together with foamy cells. The fibrin was at the apex in 10 of the cases, in one it was in the body and in one at the neck

of the aneurysm. The average maximum external diameter of 21 of the aneurysms with these changes was 4.1 mm. The same average diameter of 35 of the aneurysms without these changes was 3.8 mm. This was not significant, but suggested that these changes tended to occur in the larger unruptured aneurysms, especially in those which had just passed the *critical size* of 4 mm. described above.

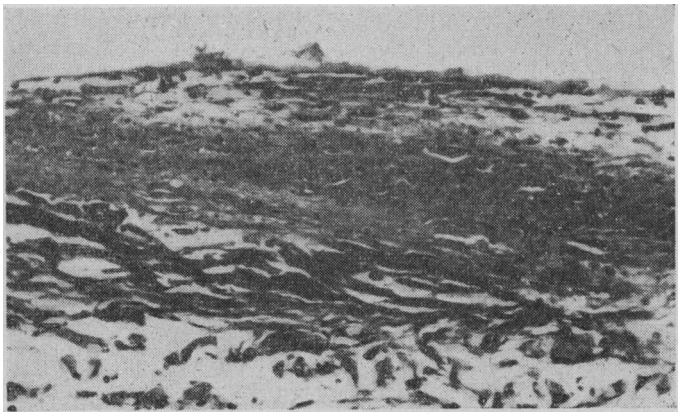

FIG. 5.—Mononuclear and polymorphonuclear cells permeating the apex of an unruptured aneurysm. The endothelium is above. (Picro-Mallory. ×110.)

In one aneurysm no blood was visible macroscopically around it, and it was apparently unruptured, but histology showed its apex to give the staining reactions of fibrin, with red cells, platelets, and white cells permeating and dissecting right through its wall at one point to form a small coagulum on its outer surface. This aneurysm was obviously oozing or weeping blood constitutents into the cerebrospinal fluid. Whether the process would have been checked or have proceeded to frank rupture it is impossible to say.

Discussion

The larger multiloculated aneurysm is more likely to rupture than the smaller uniloculated one. The important factor here is probably the size rather than the multiloculation. Multiloculation appears to indicate something of an aneurysm's past but little of its future.

There was no evidence for the traditional impression that very large aneurysms are unlikely to rupture. This has probably arisen from cases in which a large aneurysm has produced symptoms and signs by compression of cranial nerves and brain before it could rupture.

The evidence suggests that aneurysms can arise at any time in life, especially between the ages of 30 and 70, and increase rapidly in size, though they may do so slowly. This makes the onset of hypertension at any age a possible factor in the initiation as well as the enlargement of cerebral aneurysms and supports the view that an aneurysm arose *de novo* in an adult case reported by Graf and Hamby (1964).

There was a tendency in ipsilateral combinations of middle cerebral and internal carotid aneurysms for the proximal aneurysm to have ruptured. This supported the hypothesis put forward by Jain (1963). This did not apply to combinations of internal carotid and anterior communicating aneurysms, in which the collateral flow and pulsation from the opposite anterior cerebral stem appeared adequate to nullify any dampening of pulsation by the internal carotid aneurysm.

It is suggested that the cellular and fibrinous infiltrate found in the walls of unruptured aneurysms in the present investigation results in weakening of the aneurysm wall. This may then give way and enlarge the aneurysm and then organize, perhaps forming a bubble or secondary loculus, in which mural thrombus might organize and acquire an atheromatous appearance. Alternatively, blood constituents may seep or dissect through the affected aneurysm wall into the cerebrospinal fluid, as was seen to occur in the present series. This may proceed to frank rupture or it may organize and heal, with possible enlargement of the aneurysm and thickening of the adventitial component of the aneurysm wall, and, if mural thrombus forms, thickening of the intima and perhaps atheroma-like changes within it (Fig. 6).

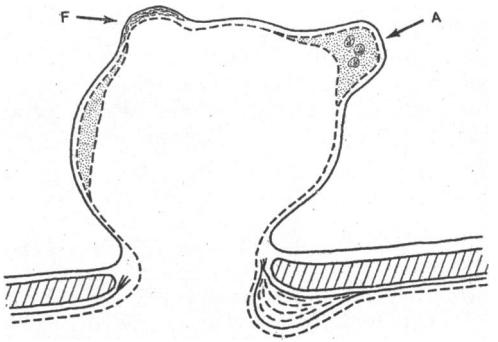

FIG. 6.—Diagrammatic representation of an aneurysm with an apical patch of fibrinoid necrosis (F) giving way to form a "bubble" which may rupture or thrombose and organize, with atheroma-like features, such as foam cells, within it (A).

These changes in the walls of aneurysms resembled the subendothelial polymorph permeation of cerebral arteries which accompanies stasis due to vascular spasm and proximal occlusion due to emboli and other causes (Crompton, 1964). They also resembled the basophil intimitis of Sheehan and Moore (1953), which those authors attributed to stasis in the small renal arteries and which was confirmed experimentally in rabbits by Sheehan and Davis (1958). Thus they appear to accompany intimal damage. This damage may be ischaemic, as in these arteries, or mechanical, due to pulsatile pressure-changes in a relatively inelastic sac or turbulent flow in an aneurysm (German and Black, 1954). In aneurysms an ischaemic factor may be added when plasma gets through a breech in the endothelium and permeates the wall, or mural thrombus impairs the diffusion of oxygen and metabolites into the aneurysm wall. Wood (1964) produced angiographic evidence of the retention of injected contrast material at the periphery of aneurysms, especially large ones. This suggests turbulent flow with friction between wall and blood, such as does not occur with axial flow. The resultant stasis and endothelial trauma might well lead to the formation of mural thrombus. Unless there was atheroma leading to the formation of vasa vasorum, no vessels were seen in the walls of aneurysms or of the thin-walled cerebral arteries from which they arise. Thus all their nutrition must be derived by diffusion from the blood in the lumen.

These changes in the walls of aneurysms appear to represent the result of damage to the walls, and they may lead on to distension, rupture, or thickening of the aneurysm wall, with possible atheroma-like changes ensuing. The sequence of events is probably very similar to the formation of some types of atheromatous plaques elsewhere in the vascular system.

Summary

Examination of 289 consecutive necropsies on patients dying after the rupture of a cerebral berry aneurysm led to the following conclusions:

Multiple cerebral aneurysms occurred in 31% of these patients.

The rupture occurred through the apex or dome of the aneurysm in most cases.

Multiloculation or the presence of secondary loculi or "bubbles" was commoner with larger aneurysms. It did not, in itself, appear to indicate any greater likelihood of rupture.

The critical size—that is, the size beyond which aneurysms suddenly become unstable and likely to rupture—was a maximum external diameter of 4 mm.

Aneurysms can probably form or begin at any age and may enlarge and rupture rapidly.

In patients with multiple aneurysms the largest aneurysm had ruptured in 88%.

When combinations of middle cerebral and internal carotid aneurysms were present on the same side the proximal aneurysm was more often ruptured.

Infiltration of the wall of the aneurysm with white blood cells and fibrin was thought to precede enlargement or rupture. This was thought to be due to endothelial damage as a result of pulsation, turbulent flow, or ischaemia resulting from mural thrombosis.

My thanks are due to the Wolfson Foundation and to the National Institute of Health, Bethesda, Maryland, U.S.A., for grants which made this study possible. I am also indebted to Professor T. Crawford, of the Department of Pathology, St. George's Hospital, London, and Mr. Wylie McKissock, Mr. L. S. Walsh, and Mr. A. E. Richardson, of the Neurosurgical Department of St. George's Hospital at Atkinson Morley's Hospital, London, for their interest, encouragement, and skilled criticism. Finally, my thanks are due to the technical, photographic, and secretarial staff, whose asistance was invaluable.

REFERENCES

Björkesten, G. af, and Troupp, H. (1962). *J. Neurosurg.*, **19**, 583.
Crawford, T. (1959). *J. Neurol. Neurosurg. Psychiat.*, **22**, 259.
Crompton, M. R. (1964). *Brain*, **87**, 491.
Falconer, M. A. (1954). *Proc. roy. Soc. Med.*, **47**, 693.
German, W. J., and Black, S. P. W. (1954). *Trans. Amer. neurol. Ass.*, **79**, 163.
Graf, C. J., and Hamby, W. B. (1964). *J. Neurol. Neurosurg. Psychiat.*, **27**, 153.
Hassler, O. (1961). *Acta psychiat. scand.*, **36**, Suppl. No. 154, p. 108.
Jain, K. K. (1963). *Surgery*, **54**, 347.
Krauland, W. (1957). In *Handbuch der speczíellen pathologischen Anatomie und Histologie*, by O. Lubarsch, F. Henke, and R. Rössle, Band 13, Teil 1, Bandteil B, p. 1511. Springer, Berlin.
McKissock, W., Paine, K. W. E., and Walsh, L. S. (1959). *Neurochirurgia (Stuttg.)*, **2**, 25.
Nyström, S. H. M. (1963). *J. Neurosurg.*, **20**, 329.
Sheehan, H. L., and Davis, J. C. (1958). *J. Path. Bact.*, **76**, 569.
—— and Moore, H. C. (1953). *Renal Cortical Necrosis and the Kidney of Concealed Accidental Haemorrhage*. Blackwell, Oxford.
Stehbens, W. E. (1963). *Arch. Neurol. (Chic.)*, **8**, 272.
Wood, E. H. (1964). *J. Neurosurg.*, **21**, 182.

Dogger Bank Itch: Survey of Trawlermen

M. L. NEWHOUSE,* M.D., M.R.C.P.

Brit. med. J., 1966, **1**, 1142–1145

Bonnevie (1948) gave the first account of an allergic dermatitis known as Dogger Bank itch or weed rash. In the late 1930s an increasing number of fishermen based on the port of Esbjerg in Denmark complained of a skin complaint which they alleged was due to contact with a seaweed known as the sea-chervil. This proved to be a coralline of the phylum Bryozoa identified as the *Alcyonidium hirsutum* (Fig. 1) found in the shallow waters of the North Sea, particularly on the Dogger Bank. Patch tests with the alcyonidium on affected patients were positive. According to Bonnevie, it is the only known allergic dermatitis caused by a live animal.

In 1939 the Danish Workmen's Compensation Act was extended to include skin diseases caused by this organism. Under the Danish law only men incapacitated for three months or longer, or those requiring change of occupation, are eligible for compensation. During the war the North Sea was mined and there was no fishing. After the war Guldager (1959) reported 95 cases seen at his fishermen's hospital in Esbjerg, and during this same period 15 to 20 fishermen were compensated annually. In more recent years the number receiving compensation has dropped to 4 to 6 a year (H. F. H. Reiter, personal communication, 1965).

In the East Coast ports of England the condition is well known and has become much more common, particularly among fishermen based on Lowestoft. The sea-chervil has again been incriminated, but specimens from the Dogger Bank brought home by English fishermen have been identified as *Alcyonidium gelatinosum* (Ryland, 1962). There has been no recent Danish work on the alcyonidium (Bonnevie, personal communication, 1965), and the prevailing species in the North Sea may have changed in the intervening years. This organism flourishes in the summer and dies off in the winter; the areas infested by it appear to vary from year to year and the impression among Lowestoft fishermen is that it covers an increasing area. The survey described in this paper was undertaken in May 1965 at the request of the White Fish Authority because many trawlermen were being incapacitated by Dogger Bank itch.

* Department of Occupational Health and Applied Physiology, London School of Hygiene and Tropical Medicine.

Trawl Fishing

At Lowestoft there are approximately 1,200 fishermen, of whom about 900 are in trawlers and the remainder in drifters. In drifting the net does not reach the sea-bed and there is no contact with the sea-chervil, which is sessile. Thus drifter-men are not affected by Dogger Bank itch. The Lowestoft trawlers are owned by various fishing companies, but the design of ships and the pattern of fishing are common to the port. The trawlers spend 12 days at sea and two in port; this rhythm is maintained throughout the year unless the vessel is sent for a refit. Although there is good fishing for cod and haddock within six to seven hours' sailing off the East Anglian coast, their main fishing-grounds are on the Dogger Bank and in its vicinity, where the more lucrative catches of plaice are found (Fig. 2).

In trawl fishing the net is shot over the side or stern of the ship. Two flexible steel wire ropes trail down under the water to the two otter boards weighted to float upright. As the ship moves slowly forward these boards are held open by the pressure of the water, and open the mouth of the net. The lower half of the net is weighted with wooden rollers and steel bobbins, which help to avoid obstructions in towing it along the sea-bed. Fish swim into the mouth of the trawl