SURGICAL TREATMENT OF SPONTANEOUS SUBARACHNOID HAEMORRHAGE

PRELIMINARY REPORT

BY

**MURRAY A. FALCONER, M.Ch., F.R.C.S.
F.R.A.C.S.**

(From the Neurosurgical Unit, Dunedin Hospital and Otago
University, New Zealand)

Between May, 1946, and August, 1949, 40 consecutive patients with recent subarachnoid haemorrhage were referred to this unit for surgical investigation and treatment. This preliminary report outlines the results obtained, and advocates the routine surgical management of all cases of this condition.

Prognosis of Subarachnoid Haemorrhage Treated Conservatively

Spontaneous subarachnoid haemorrhage is a treacherous and deadly condition, and occurs at all age periods. It is usually treated by rest and quiet, and statistics from various sources show that in most general hospitals between 50 and 60% of patients die and only 15 to 30% make a satisfactory recovery (Taylor and Whitfield, 1936; Richardson and Hyland, 1941; Magee, 1943; Hamby, 1948). The Dunedin Hospital figures for 1943-7 were 45% and 19% respectively. A striking feature of the condition is the frequency with which bleeding recurs. In this connexion the above statistics show that about one-third of the patients die within a few days of their initial attack; one-half of the survivors then develop another attack while they are still in hospital and two-thirds of these die. The incidence of these recurrent attacks reaches its maximum peak about the third week after the initial attack, and falls sharply after the first month, but a few patients are still liable to a later attack (Magee, 1943). Ultimately only 40 to 50% of those who are stricken by subarachnoid haemorrhage leave hospital, and a large proportion of these are disabled.

Aetiology.—The lesion commonly responsible for the bleeding is a leaking congenital aneurysm on the circle of Willis or adjacent arteries. Several well-documented clinico-pathological studies have established this (Symonds, 1924; Richardson and Hyland, 1941; Magee, 1943). Uncommon causes include leaking arteriovenous malformations (arteriovenous aneurysms) and rupture of an atheromatous artery. Many cases of leaking congenital aneurysm also have a massive intracerebral haemorrhage associated with the subarachnoid haemorrhage (Richardson and Hyland, 1941).

Recorded Surgical Experiences

Several surgeons have explored the possibilities of operative intervention in subarachnoid haemorrhage. The methods tried range from carotid artery ligation (Dott, 1933) to direct intracranial measures, such as wrapping of hammered muscle around the aneurysm (Dott, 1933), incision of the aneurysm with packing of muscle into its interior (McConnell, 1937), clipping of the neck of the aneurysm (Dandy, 1938), trapping of the aneurysm by occluding the parent artery on either side of it (Fincher, 1939), and shrivelling of the aneurysmal sac with diathermy (Dandy, 1944). There are two recorded cases of excision of the aneurysm itself (Cone, 1939; Dandy, 1944).

- Code, C. F., Evans, C. L., and Gregory, R. A. (1938). *J. Physiol.*, **92**, 344.
- Cullumbine, H. (1947). *J. Path. Bact.*, **59**, 477.
- Carter, C. E., and Cohn, W. E. (1949). *Fed. Proc.*, **8**, 190.
- Dekanski, J. (1945). *J. Physiol.*, **104**, 151.
- Drury, A. N. (1936). *Physiol. Rev.*, **16**, 292.
- Dubois-Ferrière, H. (1945a). *C.R. Soc. Phys. Hist. nat. Genève*, **62**, 23.
- (1945b). *La Maladie post-opératoire et le Shock Traumatique*. Lausanne.
- Duncan, G. W. (1943). *Arch. Surg.*, **46**, 214.
- Dyckerhoff, H., Schörcher, F., and Torres, J. (1939). *Biochem. Z.*, **300**, 193, 198.
- Ederstrom, H. E., DiGangi, M. R., and Calman, C. (1948). *Amer. J. Physiol.*, **155**, 434.
- Emmelin, K., and Emmelin, N. (1947). *Acta physiol. scand.*, **14**, 16.
- Emmelin, N., Kahlson, G. S., and Wicksell, F. (1941). *Ibid.*, **2**, 110.
- Folkow, B., Haeger, K., and Kahlson, G. (1948). *Acta physiol. scand.*, **15**, 284.
- Goranson, E. S., Hamilton, J. E., and Haist, R. E. (1948). *J. biol. Chem.*, **174**, 1.
- Green, H. N. (1943). *Lancet*, **2**, 147.
- (1945). *Brit. med. Bull.*, **3**, 102.
- Bielschowsky, M., and Stoner, H. B. (1947). Unpublished report to M.R.C.
- Holdsworth, F. W., and Parkin, T. (1942). Unpublished report to M.R.C.
- and Stoner, H. B. (1944). *Brit. J. exp. Path.*, **25**, 150.
- (1947). *Ibid.*, **28**, 189.
- (1950). *The Biological Actions of the Adenine Nucleotides*. Lewis, London.
- and Bielschowsky, M. (1949). *J. Path. Bact.*, **61**, 101.
- Whiteley, H. J., and Eglin, D. (1949). *Clin. Sci.*, **8**, 65.
- Haist, R. E., and Hamilton, J. I. (1944). *J. Physiol.*, **102**, 471.
- (1945). *Canad. J. Res. (E)*, **23**, 89.
- Irish, U., Hamilton, J. I., Haist, R. E., and Jacques, L. B. (1945). *Ibid.*, **23**, 119.
- Kalckar, H. M. (1944). *J. biol. Chem.*, **154**, 267.
- and Lowry, O. H. (1947). *Amer. J. Physiol.*, **149**, 240.
- Kellaway, C. H. (1947). *Edinb. med. J.*, **54**, 333.
- and Trethewie, E. R. (1940). *Aust. J. exp. Biol. med. Sci.*, **18**, 63.
- and Turner, A. W. (1940). *Ibid.*, **18**, 253.
- Knisely, M. H., Bloch, E. H., Eliot, T. S., and Warner, L. (1947). *Science*, **106**, 431.
- Eliot, T. S., and Bloch, E. H. (1945). *Arch. Surg., Chicago*, **51**, 220.
- Kwiatkowski, H. (1941). *J. Physiol.*, **100**, 147.
- Le Page, G. A. (1946a). *Amer. J. Physiol.*, **146**, 267.
- (1946b). *Ibid.*, **147**, 446.
- Lewis, T. (1927). *Blood Vessels of the Human Skin and Their Responses*. Shaw, London.
- Lowry, A., and Code, C. F. (1948). *Amer. J. Physiol.*, **155**, 452.
- Macdonald, A. D., and Woolfe, G. (1938). *J. Physiol.*, **93**, 58P.
- Macfarlane, R. G. (1937). *Lancet*, **1**, 10.
- and Spooner, S. J. L. (1946). *Brit. J. exp. Path.*, **27**, 339.
- McShan, W. H., Potter, V. R., Goldman, A., Shipley, E. G., and Meyer, R. K. (1945). *Amer. J. Physiol.*, **145**, 93.
- Mazur, A., and Shorr, E. (1948). *J. biol. Chem.*, **176**, 771.
- Menkin, V. (1940). *Dynamics of Inflammation*. Macmillan, New York.
- Mylon, E., and Winternitz, M. C. (1946). *Amer. J. Physiol.*, **145**, 254.
- and de Sütö-Nagy, G. J. (1943). *Ibid.*, **139**, 299.
- Prinzmetal, M., and Bergman, H. C. (1945). *Clin. Sci.*, **5**, 205.
- and Hechter, O. (1944). *Surgery*, **16**, 906.
- Rose, B., and Browne, J. S. L. (1942). *Ann. Surg.*, **115**, 390.
- Stoner, H. B., and Green, H. N. (1944). *J. Path. Bact.*, **56**, 343.
- (1945a). *Ibid.*, **57**, 337.
- (1945b). *Clin. Sci.*, **5**, 159.
- (1949a). *J. Path. Bact.*, **61**, 114.
- (1949b). *Ibid.*, **61**, 111.
- and Threlfall, C. J. (1948). *Brit. J. exp. Path.*, **29**, 419.
- Tabor, H., and Rosenthal, S. M. (1947). *Amer. J. Physiol.*, **149**, 449.
- Trueta, J., Barclay, A. E., Daniel, P. M., Franklin, K. J., and Prichard, M. M. L. (1947). *Studies of the Renal Circulation*. Blackwell, Oxford.
- Yudin, S. S. (1937). *Lancet*, **2**, 361.
- Zipf, K. (1931). *Arch. exp. Path. Pharmacol.*, **160**, 579.
- (1932). *Ibid.*, **167**, 60.

The Prime Minister of Northern Ireland, Sir Basil Brooke, has accepted from the National Corporation for the Care of Old People a gift of £50,000 for the establishment of rest homes in Northern Ireland. Some time ago the Corporation received £170,000 from the gift of £1,000,000 to the people of Britain from the people of South Africa, and the whole of the allocation has been earmarked to provide rest homes for the infirm in Britain. The sum given to Northern Ireland is to be used to build one or two homes.

In assessing the risks of carotid ligation for intracranial aneurysm, a distinction must be made between ligation for leaking aneurysm and ligation for non-leaking aneurysm. In the presence of subarachnoid bleeding, carotid ligation has usually carried a mortality of between 20 and 30% or even higher. Thus Schorstein, in 1940, in reporting the collective experience of the Society of British Neurological Surgeons, recorded five deaths in nine cases of bleeding aneurysm, as opposed to three deaths in 22 cases of non-bleeding aneurysm. Dott (Hermann, Obrador, and Dott, 1937) has recorded a mortality of 25% in 12 cases of leaking aneurysm, and Kraysenbühl (1946) a mortality of 30% in 20 cases. Most of these cases have been treated by internal carotid ligation. Rogers (1949) performed common carotid ligation in six cases of leaking aneurysm with one death.

Dott, in 1933, was the first to deal successfully with a leaking intracranial aneurysm which he exposed via a frontal craniotomy. His case was one of bleeding aneurysm on the intracranial internal carotid artery, which he wrapped with hampered muscle, and his patient subsequently lived an active life for nine years, dying of another cause (Dott, 1948—personal communication). Several other surgeons, including Tönnis (1936), Ccne (1939), Jefferson (1947), and Swain (1948), have also had success with an occasional case of bleeding aneurysm treated by an intracranial procedure. Dandy (1944), with the largest experience of intracranial attacks on aneurysms, encountered at operation 36 cases, 30 of which he treated by various local measures, with nine deaths. Few of his cases, however, had a history of recent bleeding.

Case Material and Results in this Series

The 40 consecutive cases of subarachnoid haemorrhage which form the subject of this report were investigated and treated surgically, with five deaths (12.5% mortality rate) and 26 good recoveries, the patients resuming their full lives without appreciable disability (65% satisfactory recovery rate). Five further patients, although disabled, are still able to lead useful lives. The ages of the patients varied from 9 to 67 years, and 18 were males and 22 females. Included in the group were: (a) 30 cases of leaking intracranial aneurysm (31 demonstrated aneurysms)—four died and 26 recovered; (b) three cases of leaking arteriovenous malformation—all cured; (c) one case of massive intracerebral clot associated with subarachnoid haemorrhage occurring from a leak of the circle of Willis without aneurysmal formation—cured; and (d) six cases in which the bleeding-point was not disclosed by carotid arteriography—one died and five recovered.

In most cases surgical investigation was started within a few days of the last attack of bleeding, and in all except three cases the cerebrospinal fluid was still blood-stained or xanthochromic. Most of the patients (62%), having developed recurrent bleeding while still in hospital, were regarded by medical colleagues as being in grave jeopardy. Half the patients were transported to this unit by air ambulance, and they stood the journey well.

As our experience grew we established a routine for investigating and treating patients. First, carotid arteriography is performed to locate and identify the lesion. Immediately after this, ligation of the common carotid artery on the appropriate side is performed in one or more stages, followed later in some cases by internal carotid ligation. Subsequently, a direct attack is made upon the aneurysm if it is accessible. A few cases, however, are attacked direct without preliminary carotid ligation, as when the arteriographic studies indicate that owing to a

brisk collateral circulation the blood flow through the affected artery will not be appreciably affected by the ligation.

Carotid Arteriography

The method of arteriography preferred is that devised by Moniz (1934), in which the common carotid artery is exposed low in the neck between the two heads of the sternomastoid muscle and injected under direct vision. The exposure is done aseptically, usually under local analgesia, with the patient in position on the radiographic table. In some earlier cases the internal carotid artery was exposed instead, and in an odd case a percutaneous arteriogram was taken. The great advantage of the Moniz procedure is that at the completion of the x-ray examination the common carotid artery is exposed, ready for prompt ligation. A 35% (occasionally 50%) diodone compound solution was the contrast medium used in most cases, as it is safe and is subsequently excreted in the urine.

Altogether carotid arteriography was undertaken in 38 of the 40 cases, the first two in the series being located by clinical signs alone. Localizing signs, however, were present in only half of my cases, and so both the right and the left carotid arteries were usually injected. In one case two aneurysms were disclosed, and the 31 aneurysms demonstrated in 30 patients were situated as follows: 14 on the intracranial internal carotid artery, five at the bifurcation of the internal carotid artery, three on the middle cerebral artery, and nine on the anterior cerebral artery. Of the three arteriovenous malformations, one was disclosed in the cortical distribution of the middle cerebral artery and two were within the lateral ventricle in the distribution of the posterior cerebral artery.

Carotid Ligation

This is the sheet-anchor of the operative treatment of intracranial aneurysm, but it has dangers and limitations. Internal carotid ligation in particular was apt to be followed by hemiplegia, and this complication was not always obviated by prompt release of the ligature as soon as the first signs of hemiparesis appeared, presumably because a spreading thrombosis of the artery had already developed. The principle of carotid ligation, of course, is to reduce the blood flow through the affected artery, and consequently to lower the arterial pressure within the aneurysm, in order to encourage sealing of the leak by intra-aneurysmal clotting.

After experience of internal carotid ligation in 10 cases, stage-ligation of the common carotid artery was adopted as our routine method. In this a silk ligature is passed around the common carotid artery, and the artery is then narrowed by one-third or one-half. Two or three days later the neck wound is reopened and the carotid tied tightly. It is often wise, at a later date, to expose the internal carotid artery in the neck and tie it completely, because after common carotid ligation a blood flow through the internal carotid artery becomes established from a retrograde flow down the external carotid artery. As a secondary effect of this collateral circulation we observed, in one case of aneurysm of the intracranial internal carotid artery, a third-nerve palsy (due to compression of the nerve by the aneurysm) appear within a week of common carotid ligation. Thus common carotid ligation, although generally effective, may not be sufficient of itself for the permanent relief of an aneurysm on the intracranial carotid artery, and in such circumstances should be followed by ligation of the internal carotid artery.

No instance of hemiplegia that could be ascribed to the ligation occurred in 14 cases treated by stage-ligation of the

common carotid artery, whereas four cases of hemiplegia due to spreading post-ligation thrombosis occurred, after primary internal carotid ligation, and one of these proved fatal. Altogether, carotid ligation, either common or internal, was performed in 24 cases with four deaths, but two of the deaths were due to the effects of an unrelieved massive intracerebral clot.

Intracranial Procedures

Although carotid ligation alone is effective in many cases, particularly internal carotid ligation for aneurysm of the intracranial internal carotid below the circle of Willis, its effect in other aneurysms may be only temporary. Thus aneurysms on the circle or its distal arterial branches may again cause trouble, once the collateral supply from the opposite carotid artery becomes fully established. Even an aneurysm on the intracranial internal carotid may fill by reflux from the circle of Willis, as demonstrated in one case in which percutaneous arteriography, performed on the opposite carotid 15 months after internal carotid ligation, again showed up the aneurysm, much smaller in outline but present none the less. Therefore it has been our policy, whenever practicable, to follow up carotid ligation with an intracranial approach to the aneurysm.

By means of a frontal craniotomy similar to that used for a pituitary tumour, the whole course of the intracranial carotid artery can be readily exposed, as well as the proximal part of the middle cerebral artery and the anterior cerebral artery up to and including the anterior communicating artery. The distal course of the anterior cerebral artery can then be exposed by retracting the mesial surface of the frontal lobe from the falx, while the distal course of the middle cerebral artery can be exposed by splitting open the fissure of Sylvius. Most intracranial aneurysms causing subarachnoid haemorrhage are situated on one or other of these arteries.

A direct attack was carried out 19 times in 18 of the 30 patients with intracranial aneurysm. There was only one death. Of the 17 survivors 13 recovered without residual disability. Six patients were treated by an intracranial procedure alone, without resort to carotid ligation. Various local measures were employed once the aneurysm was exposed: in eight cases hammered muscle was wrapped around it; in five cases an aneurysm on the intracranial internal carotid was trapped between a ligature applied to the artery in the neck and a silver clip placed across the artery above the aneurysm but below the circle of Willis; in four cases (with five aneurysms) the neck of each aneurysm was occluded with a silver clip; while in the remaining case a clip was placed across the anterior cerebral artery immediately proximal to the aneurysm.

The methods most favoured are trapping of the aneurysm, whenever practicable, and wrapping of the aneurysm with muscle. The placing of a clip across the neck of an aneurysm is apt to tear it and cause bleeding. Three cases of intracranial aneurysm were associated with massive intracerebral haemorrhage, and in one of these the clot was successfully evacuated from within the temporal lobe, while the responsible aneurysm on the internal carotid artery was wrapped with muscle. The other two cases were not attacked intracranially, and both died of the effects of unrelieved raised intracranial pressure.

Besides the 18 cases of intracranial aneurysm, four of the 10 cases due to lesions other than aneurysm were also submitted to an intracranial procedure. All four recovered. Three were instances of bleeding arteriovenous malformation: one of these in the parietal region was excised as advocated by Olivecrona and Riives (1948), while the

remaining two cases, both situated within the lateral ventricle in the distribution of the posterior cerebral artery, were submitted to a lateral craniotomy with clipping of the posterior cerebral artery at a point lateral to the crus cerebri. The fourth case—one of massive clot within the frontal lobe due to a leaking point on the anterior cerebral artery without aneurysmal formation—was treated by evacuation of the clot and wrapping of the affected artery with muscle.

Fatal Cases

There were five deaths—Cases 2, 4, 22, 26, and 32 in the series. All were examined *post mortem*. In Case 2, explored intracranially without preliminary arteriography, torrential bleeding was encountered from an aneurysm of the internal carotid artery, which was then treated by an emergency internal carotid ligation in the neck, plus wrapping of muscle around the aneurysm. Death occurred from spreading thrombosis of the internal carotid artery leading to infarction of the brain. In Case 4 a midline aneurysm was present on the anterior cerebral system. One internal carotid artery was ligated, and subsequently the necropsy revealed an anomaly of the circle of Willis (disclosed previously by arteriography but misinterpreted), such that both anterior cerebral arteries were supplied by the artery which had been tied. In Case 22 no lesion was disclosed by arteriography, and necropsy revealed a primary intraventricular haemorrhage which was apparently secondary to a leaking artery in the septum lucidum.

Cases 26 and 32 were both examples of massive frontal lobe haemorrhage secondary to aneurysm of the anterior cerebral artery. In both cases the presence of a large intracerebral clot was indicated by the arteriograms, but in only one case were the outlines of the aneurysm visualized. Both cases were treated by carotid ligation on the side of the intracerebral haemorrhage, and both subsequently died of raised intracranial pressure. In Case 26 we were amazed to find that the aneurysm was situated on the anterior cerebral artery of the opposite side, and had bled into the frontal lobe across the midline. Carotid ligation had been performed on the wrong side.

Discussion

The results achieved in this series of 40 consecutive cases indicate that surgery has a place in the treatment of spontaneous subarachnoid haemorrhage, and one which we think will become routine. Compared with a mortality rate of between 50 and 60% for conservative treatment and a satisfactory recovery rate of between 16 and 30%, the reduction of the mortality rate in this surgically treated group to 12.5% and the elevation of the satisfactory recovery rate to 65% are gratifying. With further experience this record should be improved.

In the period during which our first 20 cases were being collected my unit was asked by medical colleagues to see in consultation in this hospital six other patients with subarachnoid haemorrhage. One of these was moribund, and died within half an hour. The other five were all recovering from their first attack and seemed to be progressing well. We therefore decided to leave them alone, but to our regret no fewer than three of them had a second attack while they were still in hospital, and all three succumbed rapidly. Thereafter we regarded subarachnoid haemorrhage as a surgical emergency to be investigated and treated as soon as practicable, even if the patient was conscious and alert.

No instance of a leaking aneurysm on the vertebral, basilar, or posterior cerebral arteries was found in this

group, although two arteriovenous malformations were disclosed on the posterior cerebral artery. Possibly aneurysmal lesions were present in some of the five cases which recovered without a bleeding lesion being shown up by carotid arteriography. The remaining cases, of course, were accounted for. Post-mortem studies, however, indicate that most aneurysms which bleed are located on the anterior part of the circle of Willis (Turnbull, 1918).

Spontaneous subarachnoid haemorrhage is not uncommon. Precise statistics of its incidence are not available, but the New Zealand Health Department Statistician (Mr. C. E. Gardiner) informs me that 77, 82, and 75 deaths were certified in this country as due to this condition in the years 1946, 1947, and 1948 respectively. If we suppose that an equal number of patients had the condition and survived, then on an average 160 cases occur each year in New Zealand—that is, approximately 100 cases annually per million of population.

Here is a wide field awaiting surgery. Time alone will decide the worth of the various measures which have been used in this series.

The following case with two aneurysms, one on each side of the circle of Willis, illustrates some of the problems encountered with surgical treatment.

Case 17

A 38-year-old man was suddenly stricken, on June 20, 1948, with severe left-sided headache as he was getting out of bed. Within a few minutes he became very drowsy, and vomited several times. For three days he was confused and irrational, and on regaining orientation he could not recall what had happened to him, but complained of a generalized headache which continued for a few days.

When admitted to the Christchurch Hospital on the fifth day he was moving all his limbs normally, but the right plantar response was extensor, the right abdominal reflexes were absent, while some neck rigidity and Kernig's sign were present. Lumbar puncture disclosed a moderately blood-stained cerebrospinal fluid, and this cleared within a few days. Five years previously he had had a similar attack of sudden headache followed by confusion.

At the end of the third week, therefore, when acute symptoms seemed to have subsided, Dr. R. H. Q. Baxter had him transferred by air ambulance to the Neurosurgical Unit for further investigation. Two nights later, while lying in bed eating his evening meal, he had another attack of severe left-sided headache, this time without loss of consciousness or focal neurological signs. The lumbar cerebrospinal fluid was again blood-stained. Digital compression of each carotid artery in the neck for a period of ten minutes was well tolerated, the compression on the left side temporarily easing his headache.

Arteriographic Studies

Next morning bilateral carotid arteriography was carried out by the Moniz method under local analgesia. The left common carotid artery was injected first, and the resulting arteriograms showed a moderate-sized aneurysm projecting upwards in the angle between the diverging left anterior and middle cerebral arteries (Fig. 1). The internal carotid and middle cerebral arteries filled clearly, but the anterior cerebral artery was only very faintly discerned.

The right common carotid artery was then injected, primarily to test out the collateral circulation through the circle of Willis (Fig. 2). To our amazement a second and smaller aneurysm was clearly displayed in the antero-posterior view, occupying the symmetrical site to that of the first aneurysm. The anterior, middle, and posterior cerebral arteries on the right side were well outlined, while the left anterior cerebral artery was also outlined, but faintly. The right lateral carotid arteriogram, however, taken while the left carotid artery was temporarily occluded, showed good filling of the anterior and middle cerebral arteries on both sides, while the

FIG. 1.—Left carotid arteriogram showing moderate-sized aneurysm (arrow) projecting upwards at angle between anterior and middle cerebral arteries. The anterior cerebral artery is only faintly discernible.

shadows of the two aneurysms were superimposed, the larger one on the left obscuring the shadow of the smaller one on the right.

Surgical Considerations and Procedure

The appearance of recurrent bleeding was a dire sign. Two aneurysms had been demonstrated, one on each side of the brain. We presumed that the left and larger aneurysm was the bleeding one, because the initial headache had been left-sided, minimal right-sided pyramidal signs had been observed after the attack three weeks before, while digital compression of the left common carotid artery had temporarily eased the headache of the third attack of bleeding. It was evident from the arteriograms that the aneurysms had narrow necks, and it was obvious from the right lateral carotid arteriogram that the collateral circulation between the two halves of the circle of Willis through the anterior communicating artery was a free one, invalidating carotid ligation. I therefore determined to attack each aneurysm directly.

FIG. 2.—Right carotid arteriogram. The A.P. shows small aneurysm (arrow) projecting upwards in angle between anterior and middle cerebral arteries.

At the conclusion of the arteriography, while the common carotid arteries were still exposed, the left common carotid artery was ligated completely. I realized it was only a temporizing measure, but I reasoned that if the direct attack could be deferred for a few days some healing might occur in the wall of the aneurysm and so lessen the risk of a torrential haemorrhage occurring from the aneurysm when it was exposed.

Two weeks later, by means of a coronal scalp flap and a left frontal bone flap performed under general anaesthesia, the left-sided aneurysm was attacked. The left frontal lobe was elevated off the floor of the anterior cranial fossa, as in a pituitary operation, and the left internal carotid artery was exposed as it lay immediately behind and lateral to the optic nerve. The artery was then followed to its bifurcation, and in the process of doing this the neck of the aneurysm was exposed in the angle between the diverging anterior and middle cerebral arteries. The neck was about 7 or 8 mm. in diameter. The fundus of the aneurysm was not seen, for it projected upwards into the frontal lobe. Two silver clips, each slightly longer than standard size, were then applied to the neck of the sac in such a way that they occluded it without narrowing the origins of the anterior and middle cerebral arteries. The clips were then buttressed by being wrapped round with muscle, and the craniotomy was closed.

Ten days later the coronal scalp flap was reopened and a right frontal craniotomy performed. The smaller aneurysm on the right side was thus located, being treated in the same way by the application of silver clips to its neck (Fig. 3).

FIG. 3.—Post-operative radiograph of skull showing position of silver clip placed on the necks of the two aneurysms, and also the position of the two bone flaps.

Convalescence from both these operations was straightforward, and the patient made a complete recovery except for bilateral loss of smell, indicating damage to his olfactory nerves during elevation of the frontal lobes. Within two months of his second craniotomy he had resumed his work as a company accountant, and one and a half years later he reported that he was symptom-free except for loss of smell, and that he had been most successful in his profession. His wife in an independent statement reported that he was mentally normal in every way.

I wish to thank those physicians who entrusted my unit with their patients. Fifteen patients were referred by colleagues in the Dunedin Hospital, 14 by colleagues in the Christchurch Hospital, and the remainder from other hospitals. Four of the cases were treated by my associate, Mr. Anthony James, who has kindly allowed me to include them in this report. I particularly wish to acknowledge

the encouragement which I received from Professor F. G. Bell, Dr. J. A. D. Iverach, and Dr. R. H. Q. Baxter during my first endeavours. Dr. A. C. Begg and Mr. M. Allen collaborated with the radiographic studies.

REFERENCES

- Cone, W. (1939). Quoted by C. K. Russel. *Trans. Amer. neurol. Ass.*, 65, 130.
 Dandy, W. E. (1938). *Ann. Surg.*, 107, 654.
 — (1944). *Intracranial Arterial Aneurysms*. New York.
 Dott, N. M. (1933). *Edinb. med. J.*, 40 (Med. Chir. Soc. Trans.), 219.
 Fincher, E. F. (1939). *Yale J. Biol. Med.*, 11, 423.
 Hamby, W. B. (1948). *J. Amer. med. Ass.*, 136, 522.
 Hermann, K., Obrador, S., and Dott, N. M. (1937). *Lisboa Méd.*, 14, 782.
 Jefferson, G. (1947). *Proc. R. Soc. Med.*, 40, 419.
 Krayenbühl, H. (1946). *Schweiz. med. Wschr.*, 76, 908.
 McConnell, A. A. (1937). *Zbl. Neurochir.*, 2, 269.
 Magee, C. G. (1943). *Lancet*, 2, 497.
 Moniz, E. (1934). *L'angiographie cérébrale*. Paris.
 Olivecrona, H., and Riives, J. (1948). *Arch. Neurol. Psychiat.*, Chicago, 59, 567.
 Richardson, J. C., and Hyland, H. H. (1941). *Medicine*, 20, 1.
 Rogers, L. (1949). *Lancet*, 1, 949.
 Swain, R. D. (1948). *Surg. Clin. N. Amer.*, 28, 396.
 Symonds, C. P. (1924). *Quart. J. Med.*, 18, 93.
 Taylor, A. B., and Whitfield, A. G. W. (1936). *Ibid.*, 5, 461.
 Tönnis, W. (1936). *Zbl. Neurochir.*, 1, 39.
 Turnbull, H. M. (1918). *Brain*, 41, 50.

PRESENT STATUS OF THE Rh-Hr BLOOD TYPES

BY

ALEXANDER S. WIENER, A.B., M.D.

(From the Office of the Chief Medical Examiner of New York City)

There have been considerable advances in our understanding of the Rh-Hr blood types during the past few years. The purpose of this article is to summarize the present status of the subject in order to take into account especially the newer knowledge concerning genes r^y , R^z , R^{1w} , and the R^0 variants.

At present the existence of three "standard" Rh antisera is recognized—namely, anti-Rh₀, anti-rh', and anti-rh". Sera giving reactions reciprocally related to those of the Rh antisera are designated as Hr antisera, and two such sera have been shown to exist—namely, anti-hr' and anti-hr". Despite claims to the contrary published in the literature, the existence of an antiserum and corresponding factor reciprocally related to Rh₀ has not been convincingly demonstrated to date (Wiener, 1950). With the aid of the three standard Rh antisera the existence of eight sharply defined allelic genes has been established. These genes can be identified by the reactions with the three Rh antisera alone (Wiener and Hyman, 1948), and the reactions with the Hr antisera merely define additional characteristics not essential for their differentiation (see accompanying Table).

Table Showing Present Status of Rh Series of Allelic Genes

Gene	Corresponding Agglutinin	Reactions with Rh Sera				Reactions with Hr Sera	
		Anti-Rh ₀	Anti-rh'	Anti-rh"	Anti-rh ^w	Anti-hr'	Anti-hr"
r^y	rh	Neg.	Neg.	Neg.	Neg.	Pos.	Pos.
r^y	rh'	"	Pos.	"	"	Neg.	"
r^y	rh"	"	Neg.	Pos.	"	Pos.	Neg.
r^y	rh ^w	"	Pos.	"	"	Neg.	Pos.
R^0	Rh ₀	Pos.	Neg.	Neg.	"	Pos.	Pos.
R^1	Rh ₁	"	Pos.	"	"	Neg.	"
R^2	Rh ₂	"	Neg.	Pos.	"	Pos.	Neg.
R^3	Rh ₃	"	Pos.	"	"	Neg.	Pos.
R^{1w}	Rh _{1w}	"	Neg.	Pos.	"	Pos.	Pos.
R^0	Rh ₀	Weak	Neg.	"	Pos.	Pos.	"
R^1	Rh ₁	"	Pos.	"	"	Neg.	"
R^2	Rh ₂	"	Neg.	Pos.	"	Pos.	Neg.