Intracerebral haematoma complicating ruptured cerebral berry aneurysm

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This paper is an account of the morbid anatomical findings at necropsy in a study of intracerebral haematomata in 103 consecutively examined cases of ruptured cerebral berry aneurysm. In the majority of these cases the site of the ruptured aneurysm had been demonstrated by carotid angiography during life but in a few the aneurysm was only shown at necropsy. It is by studying these lesions at necropsy that information will be gained concerning some of the lesions probably present in patients surviving with a neurological deficit but it must be remembered that the severity and frequency of the lesions in cases coming to necropsy will not necessarily be the same as in those surviving though the nature of the lesions will probably be very similar.

As pointed out by Robertson (1949) and again 10 years later by Tomlinson (1959), the factual information to be found in the literature on the intracerebral sequelae of ruptured cerebral aneurysm is very sparse. Many of the conclusions drawn have resulted from impressions gained from a small number of cases rather than from factual observations on a larger series.

This paper is primarily concerned with intracerebral haematomata, but figures relating to cerebral infarction and subarachnoid haematomata in the same series will be given for comparison, and also because it is important to avoid giving the impression that intracerebral haematomata are the

only complication of the rupture of cerebral berry aneurysms.

INCIDENCE

One hundred and three cases of ruptured cerebral aneurysm were examined at necropsy. Sixty-two of them had intracerebral haematomata and 62 had cerebral infarction. This implies definite, large regions of cortical infarction, involving at least one-third of the region of supply of one of the cerebral arteries or ganglionic necrosis at least 5 mm. in diameter. Of those 62 cases with intracerebral haematomata, 32 also showed cerebral infarction.

AGE AND SEX DISTRIBUTION

Table I shows the age distribution of the 103 cases in decades. These have been separated into sub-groups and it can be seen that nearly all the cases over 60 years of age were in women.

The age distribution for cases with infarction and cases with intracerebral haematomata is similar to that of the total series, except that infarction is somewhat more common in the older age groups than are haematomata and a large number of these older cases with infarction are in women. The respective incidence of haematomata and infarction in men and women is summarized in Table I, and

TABLE I
AGE DISTRIBUTION OF MALES AND FEMALES IN TOTAL SERIES AND IN CASES WITH INTRACEREBRAL HAEMATOMATA AND CEREBRAL INFARCTION

Age Groups (years)	Total Cases (103 cases)		Intracerebral Haematomata (62 cases)		Cerebral Infarction (62 cases)	
	Males	Females	Males	Females	Males	Females
21-30	2	1	2	0	1	0
31-40	8	6	4	3	6	5
41-50	11	12	9	8	5	7
51-60	18	19	12	9	8	11
61-70	4	19	3	11	3	14
71 plus	0	3	0	1	0	2
	43	60	30	32	23	39

there appears to be a greater tendency for women to suffer infarction and for men to develop haematomata.

DISTRIBUTION OF ANEURYSMS

Table II shows the distribution of ruptured aneurysms in the total series and in the cases with intracerebral haematomata. Only aneurysms on the anterior half of the circle of Willis are described as the one aneurysm on the posterior half did not produce an intracerebral haematoma. The figures in this table suggest that anterior communicating and anterior cerebral aneurysms are more prone to produce haematomata than are other aneurysms.

TABLE II
DISTRIBUTION OF ANEURYSMS

	Total Necropsied Series	Intracerebral Haematoma
Total cases	102	62
Anterior communicating	35	27
Middle cerebral	34	16
Posterior communicating (at its origin from the internal carotid)	21	9
Anterior cerebral (peripheral to the anterior communicating)	6	6
Internal carotid bifurcation (between the origin of the middle and anterior cerebral)	6	4

DISTRIBUTION OF INTRACEREBRAL HAEMATOMATA

Table III shows the number of intracerebral haematomata at different sites and the aneurysms producing them. The designation of the haematomata refers to the following sites:—'Frontal', in the frontal lobes; 'caval', in the cavum of the septum pellucidum; 'external capsule', in the region of the external capsule or claustrum; 'temporal', in the temporal lobes; 'callosal', in the corpus callosum; and 'parietal', in the central parietal white matter or corona radiata.

The total number of haematomata in Table III is greater than the number of ruptured aneurysms pro-

ducing them in Table II. This is because in 16 of the 62 cases with intracerebral haematomata there were multiple haematomata. Three cases had three haematomata and 13 cases had two haematomata. Examples of such cases were anterior communicating aneurysms rupturing into both frontal lobes and up into the cavum of the septum pellucidum, or an aneurysm at the bifurcation of the internal carotid artery forming a frontal haematoma and a haematoma extending back along the external capsule.

The high incidence of haematomata in the cavum of the septum pellucidum and the frontal lobes is of course associated with the high incidence of anterior communicating aneurysms producing intracerebral haematomata, as these are the sites of predilection for haematomata due to ruptured aneurysms in this situation.

INTRACEREBRAL HAEMATOMATA RESULTING FROM
ANEURYSMS AT DIFFERENT SITES

ANTERIOR COMMUNICATING ANEURYSMS These most frequently rupture into one or both frontal lobes and then into the frontal horns of the ventricles. They may rupture directly into the inferomedial portion of the frontal lobe, with a minimum of subarachnoid bleeding, and in this case the aneurysmal sac is often found to be adherent to the posterior end of the gyrus rectus. Alternatively they may form a subarachnoid haematoma between the medial surfaces of the frontal lobes (interfrontal). The blood from this haematoma may then distend and enlarge the sulci on the medial aspect of the frontal lobes, especially the sulcus above the gyrus rectus, and may mimic intracerebral haematomata (Tomlinson, 1959) but they are surrounded by intact, often necrotic cortex. Interfrontal subarachnoid haematomata may burst into one or both frontal lobes and then into the frontal horns.

The next most frequent site for intracerebral haematomata due to the rupture of anterior communicating aneurysms is in the cavum of the septum pellucidum. After the formation of an interfrontal

TABLE III
FREQUENCY AND SITES OF INTRACEREBRAL HAEMATOMATA

Site of Aneurysm	Site of Intracerebral Haematomata						Total
	Frontal	Caval	External Capsule	Temporal	Callosal	Parietal	
Anterior communicating	22	12	2		1	2	39
Middle cerebral	3		8	6	1	1	19
Posterior communicating	1		1	6			9
Anterior cerebral	4				2		8
Internal carotid bifurcation	2	2	2				6
	32	17	13	12	4	3	81

FIG. 1

FIG. 1. *Anterior communicating aneurysm rupturing into the cavum of the septum pellucidum.*

FIG. 2

FIG. 2. *Peripheral anterior cerebral aneurysm forming a supracallosal or intercingulate subarachnoid haematoma which has extended downwards into the lateral ventricles through a necrotic corpus callosum.*

FIG. 3. *Anterior communicating aneurysm which has ruptured into the frontal lobe and extended back along the external capsule.*

subarachnoid haematoma the blood passes backwards beneath the tough bundle of the rostral fibres of the corpus callosum and ruptures upwards into the cleft-like, or often only potential space of the cavum of the septum pellucidum (Fig. 1), distending it until one or both walls of the septum

rupture and the blood bursts into the lateral ventricles. Very occasionally the walls of the septum remain intact and the blood extends into the corpus callosum but only if this structure is necrotic. When the lateral ventricles are distended with blood it may be easy to overlook the evidence of there having been an antecedent intracaval haematoma. Tomlinson (1959) stated that in seven of his 17 cases of intracerebral haematoma following ruptured cerebral aneurysms the subarachnoid blood ruptured through the rostrum of the corpus callosum into the lateral ventricles. It seems unlikely that the subarachnoid blood should rupture through one of the strongest fibre bundles in the brain when the path of least resistance is behind these fibres and up into the cavum. The rostral fibres of the corpus callosum may suffer some laceration as the blood passes behind them, especially if they are necrotic, and this may suggest that the rostrum itself has been ruptured.

Less frequently the subarachnoid blood between the frontal lobes may pass over the dorsum of the corpus callosum to form a subarachnoid haematoma beneath the cingulate gyri (intercingulate) (Fig. 2). This haematoma may then burst laterally beneath one or both cingulate gyri into the parietal white matter or down into the corpus callosum, but again only if this naturally strong structure is necrotic as a result of the disturbance of blood flow when the subarachnoid blood may break through it into the lateral ventricles. Conversely, there is also evidence that when the ventricles are distended with blood it may rupture through the corpus callosum if it is necrotic and form an intercingulate haematoma above it.

Another of the more infrequent sites for a

FIG. 4. Middle cerebral aneurysm rupturing directly into the external capsule and then into the frontal horn.

FIG. 5. Middle cerebral aneurysm forming a Sylvian, subarachnoid haematoma which has ruptured medially into the external capsule.

haematoma after the rupture of an anterior communicating aneurysm is in the external capsule. This is usually the posterior extension of a frontal haematoma (Fig. 3).

MIDDLE CEREBRAL ANEURYSMS Unlike anterior communicating aneurysms these aneurysms most frequently form haematomata in the region of the external capsule. However, exactly like anterior communicating aneurysms, they may form haematomata by bursting directly into the brain (Fig. 4), often when the aneurysm sac is large and partly buried in the insula cortex or by first forming a subarachnoid haematoma, in this case in the Sylvian fissure, which then ruptures into the brain (Fig. 5). Less frequently, instead of rupturing into the external capsule, the aneurysm ruptures directly or via a Sylvian haematoma into the frontal and temporal lobes. From any of these three sites the blood may rupture into the lateral ventricle, almost always into the frontal horn or the trigone, as the bulky genu of the internal capsule lies between the haematomata and the lateral ventricle in the mid-parietal region. Very occasionally blood ruptures into the temporal horn or extends into the parietal white matter or across it into the lateral fibres of the corpus callosum.

POSTERIOR COMMUNICATING ANEURYSMS These aneurysms usually rupture directly into the temporal lobe and then into the temporal horn. The blood may form a subarachnoid haematoma above the incus (supra-uncal) (Fig. 6) and then extend into the temporal horn along the choroid fissure. From the temporal white matter blood may on occasion pass into the external capsule along the firm fibre bundles, passing between these structures. Occasionally subarachnoid blood extends forwards to form a subarachnoid haematoma beneath and between the frontal lobes, sometimes mimicking the sequelae of a ruptured anterior communicating aneurysm by going so far as to produce a caval haematoma.

ANTERIOR CEREBRAL ANEURYSMS These aneurysms behave almost exactly like anterior communicating aneurysms if they are situated inferiorly to the rostrum of the corpus callosum on the proximal portion of the anterior cerebral artery beyond the anterior communicating artery. Like anterior communicating aneurysms they most frequently rupture into the frontal lobes; they form interfrontal, subarachnoid haematomata and they rupture upwards into the cavum of the septum pellucidum. The minority of these aneurysms, which lie above the corpus callosum, far peripheral on the anterior cerebral artery, form intercingulate rather than

FIG. 6. *Internal carotid aneurysm, arising at the origin of the posterior communicating artery, forming a supra-uncal subarachnoid haematoma which has ruptured into the temporal horn of the lateral ventricle.*

interfrontal subarachnoid haematomata and rupture into the corpus callosum when this is necrotic.

ANEURYSMS AT BIFURCATION OF INTERNAL CAROTID ARTERIES These aneurysms usually rupture directly into the frontal lobe and then into the frontal horn but occasionally the blood may pass back along the external capsule. This behaviour is probably due to the fact that the aneurysm sac is very often adherent to or actually buried in the frontal lobe (Fig. 7) at the posterior end of the olfactory stalk (Robertson, 1949). In fact these aneurysms most characteristically form a subarachnoid haematoma which distends the olfactory sulcus and then ruptures into the frontal lobe. Anterior communicating aneurysms may behave similarly. Occasionally, like an anterior communicating aneurysm, these aneurysms rupture into the cavum.

Other rarer paths of rupture occur, such as the anterior communicating aneurysm rupturing back through the lamina terminalis into the third ventricle or the posterior communicating aneurysm forming a

subarachnoid haematoma between the cerebral peduncles which then ruptures into the third ventricle. Similarly, aneurysms at the bifurcation of the basilar artery (not in this series) may rupture into the third ventricle or the blood may burst up between

FIG. 7. *An unusually large internal carotid bifurcation aneurysm. The wide sac is buried in the frontal lobe and has ruptured into it and then into the frontal horn of the lateral ventricle.*

FIG. 8. *Aneurysm at the bifurcation of the basilar artery, rupturing between the cerebral peduncles and the blood tracking down into the mid-brain and pons. Haematoxylin and eosin $\times 4.5$.*

the cerebral peduncles and track back down the mid-brain and pons between the tegmentum of these structures and the cerebral peduncles and basipontis (Fig. 8), apparently tracking along the perivascular

FIG. 9

I.C. = intercingulate; C = caval; I.F. = interfrontal; S.R. = supra rectus; O. = olfactory; E.C. = external capsule (Fig. 9).
E.C. = external capsule; S. = Sylvian (Fig. 10).

FIG. 10

FIG. 9. Diagram showing haematomata produced by anterior communications, anterior cerebral and internal carotid bifurcation aneurysms. Their common directions of rupture are arrowed.

FIG. 10. Diagram showing haematomata produced by middle cerebral aneurysms. Their common directions of rupture are arrowed.

sheaths of basilar and posterior cerebral perforating vessels, passing down the brain-stem.

The more usual paths of intracerebral rupture are summarized in Figs. 9 and 10.

COMPARISON WITH HYPERTENSIVE INTRACEREBRAL HAEMATOMATA

It may be of value to compare the above description of the distribution and incidence of haematomata resulting from ruptured cerebral aneurysms with the distribution and incidence of the supratentorial primary intracerebral haematomata found in cases of vascular hypertension. According to the figures given by McKissock, Richardson, and Taylor (1961), by far the most common site for hypertensive haemorrhage is in the region of the external capsule, where 27 out of 54 occurred. This greatly outnumbered all other sites, the thalamus being next most frequent with nine out of 54 haematomata. In contrast, after the rupture of cerebral aneurysms the most common site for intracerebral haematomata is the frontal lobes. The external capsule is less than half as common and is exceeded by haematomata in the cavum of the septum pellucidum.

Certain haematomata are common to both aneurysms and hypertensive haemorrhage, namely, frontal (common with aneurysms, uncommon with hypertension), occipital (rare with aneurysms and uncommon with hypertension), temporal (fairly common with aneurysms and less common with hypertension), parietal or corona radiata (uncommon with aneurysms but fairly common with hypertension), and external capsule (moderately common with aneurysms and very common with hypertension). At all these sites the ultimate pathological diagnosis rests upon careful dissection to demonstrate or exclude a ruptured aneurysm. This is most important, as the appearance of the haematoma in the cut brain may be identical in a case of ruptured aneurysm (Fig. 4) and in one of hypertensive haemorrhage (Fig. 11). This is very striking in the external capsular haemorrhages, for a hypertensive haemorrhage in the external capsule may rupture into the subarachnoid space to form a Sylvian haematoma typical of a middle cerebral aneurysm. Conversely a middle cerebral aneurysm buried in the cortex may produce a lens-shaped, fibre-splitting haematoma in the external capsule which is typical of a hypertensive haemorrhage.

FIG. 11. Large, lens-shaped haematoma originating in the external capsular region in a case of vascular hypertension (compare with Fig. 5).

On the other hand, there are haematoma sites which are almost specific for aneurysms or for hypertensive haemorrhage. Caval haematomata are practically always due to a ruptured aneurysm, and very often to an anterior communicating aneurysm. Callosal haematomata are very unusual in hypertensive haemorrhage and nearly always indicate an aneurysm that has resulted in necrosis of the corpus callosum, namely an anterior communicating or anterior cerebral aneurysm. Conversely, thalamic haematomata are almost invariably hypertensive and isolated haemorrhages in the head of the caudate nuclei are not seen with aneurysms but are a recognized, if not very common, sequel of hypertension.

DIMENSIONS OF INTRACEREBRAL HAEMATOMATA Half of the intracerebral haematomata were over 2 cm. in diameter. The largest (up to 8 cm. in diameter) were in the external capsule where there is no easy path of rupture into the ventricles, the strong internal capsule lying medially. Frontal haematomata

were seldom over 4 cm. in diameter and caval haematomata seldom over 2 cm., as they soon ruptured into the lateral ventricles.

DIRECT AND INDIRECT RUPTURE INTO THE BRAIN In 36 of the 62 cases with intracerebral haematomata the blood ruptured into the brain indirectly after first forming a subarachnoid haematoma; in the remaining 26 cases the aneurysm burst directly into the brain. The frequency of these varying courses is shown in Tables IV and V. These figures all suggest that an important factor predisposing to intracerebral rupture of an aneurysm is the situation of the aneurysm (Robertson, 1949), in so far as if it bleeds into a relatively confined or circumscribed subarachnoid compartment, such as the Sylvian fissure or between the frontal lobes, the blood does not have rapid or free access to the basal cisterns or general subarachnoid space.

TABLE IV

NUMBER AND SITE OF SUBARACHNOID HAEMATOMATA RUPTURING INTO THE BRAIN

Site of Haematoma	Number of Cases	Total
Interfrontal	16	36 cases (58%) of the 62 cases with intracerebral haematomata
Sylvian fissure	10	
Olfactory sulcus	4	
Above corpus callosum (supracallosal or intercingulate)	4	
Supra-uncal	1	
Between cerebral peduncles	1	

TABLE V

FREQUENCY OF DIRECT AND INDIRECT INTRACEREBRAL RUPTURE FOR ANEURYSMS AT DIFFERENT SITES

Site of Aneurysm	Number of Cases	Direct	Indirect via Subarachnoid Haematomata
Middle cerebral	16	4	12
Anterior communicating	27	9	18
Anterior cerebral	6	3	3
Posterior communicating	9	5	4
Internal carotid bifurcation	4	3	1

EFFECT OF MULTIPLE BLEEDS FROM CEREBRAL ANEURYSMS ON INTRACEREBRAL RUPTURE, INTRAVENTRICULAR RUPTURE, AND DILATATION OF THE VENTRICLES

Twenty-nine of the 62 cases with intracerebral haematomata at necropsy had evidence of multiple haemorrhages from the aneurysm. This consisted of a clinical history of more than one intracranial haemorrhage with blood in the cerebrospinal fluid, or the pathological finding of dense arachnoidal

fibrosis and siderosis. On the other hand, only 12 of the 41 cases without intracerebral haematoma at necropsy, which had bled only into the subarachnoid space, had this clinical or pathological evidence of multiple haemorrhages. This supports the suggestion put forward by Robertson (1949) that the presence of previous bleeds predisposes to the formation of intracerebral haematoma as a result of subsequent haemorrhages, possibly by obstructing the rapid dissipation of subsequent bleeds through the subarachnoid space by the presence of altered blood, fibrin, and fibrous arachnoidal adhesions, resulting in confinement and loculation of the subarachnoid blood.

Similarly another important factor tending to predispose to intracerebral rupture of an aneurysm may be adhesion of the aneurysmal sac to the pia mater. In six of the seven cases with intracerebral rupture described by Tomlinson (1959) the aneurysm was adherent to the pia. This is often very difficult to assess in a ruptured aneurysm but 11 of the 62 ruptured aneurysms associated with intracerebral haematoma were found to be firmly adherent to the pia, whereas only two of the 41 ruptured aneurysms which had bled into the subarachnoid space only showed similar adhesions. These adhesions of the sac of the aneurysm to the pia may well be facilitated by previous haemorrhages. This is supported by finding that of the 13 cases in which the aneurysmal sac was adherent to the pia, eight had had previous haemorrhages, whereas this had occurred in only 35 of the 90 cases in which the sac was not adherent to the pia.

In 57 of the 62 cases with intracerebral haematoma examined at necropsy the haematoma had ruptured into the ventricular system. In 28 of these 57 cases the ventricles had been greatly enlarged by the blood, while in 13 of these 28 cases with enlarged ventricles the fourth ventricle was widely dilated.

Bebin and Currier (1957) state that massive bleeding into the ventricles commonly causes death in under six hours. Tomlinson (1959) described infarction around the fourth ventricle as a result of dilatation of this space by blood. He also described subependymal haemorrhages in the lateral ventricles following distension of the ventricles by blood. All these lesions have also been observed in the present series, especially acute necrosis of neurones in subependymal nuclei such as the hypothalamic nuclei around the third ventricle, thalamic and caudate neurones around the lateral ventricles and hypoglossal and vagal nuclei beneath the fourth ventricle. An impressive index of the distending force of the blood in the ventricles is the fact that occasionally it bursts out through the ependyma to form subependymal, intracerebral haematoma over 1 cm.

in diameter. In many cases, however, death occurs too rapidly for histological evidence of subependymal necrosis to be found.

Sixteen of the 28 cases with distension and enlargement of the ventricles by blood had evidence of multiple haemorrhages, whereas only 12 of the 29 cases with intraventricular rupture but without enlargement of the ventricles had this evidence. These findings suggest that apart from increasing the tendency to the formation of intracerebral haematoma, the presence of previous haemorrhages also increased the tendency to ventricular dilatation or enlargement following intracerebral and intraventricular rupture. This may be implemented by obstruction of the rapid escape of blood from the fourth ventricle through the exit foramina, as a result of arachnoidal fibrosis and the presence of the products of blood breaking down in the leptomeninges. Even when the exit foramina of the fourth ventricle are fully patent, blood cannot escape very rapidly from the ventricles (Symonds 1924) but it may rupture extremely rapidly into the ventricles from an intracerebral haematoma.

The significance of these findings is that though it is almost certain that massive intraventricular haemorrhage is fatal, slower intraventricular haemorrhage is definitely survived in some cases, as is shown by finding old haemorrhagic lesions which are continuous with the ventricles in patients who had hypertensive haemorrhages or ruptured cerebral aneurysms years before death. However, it is less likely to be survived by an individual who has had one or more subarachnoid haemorrhages previously and whose fourth ventricle exit foramina are stenosed or blocked by adhesions and old blood. In such a person, the ventricles will become distended with blood, whereas this may not have occurred with a slow intraventricular haemorrhage if there had not been leptomeningeal adhesions or old blood as a result of a previous haemorrhage. Thus not only is the likelihood of intracerebral haematoma increased by the presence of previous haemorrhages, but the chances of a fatal issue from the intracerebral rupture are also increased by the presence of previous haemorrhages.

COMA FOLLOWING RUPTURED ANEURYSM

Forty-two of the 62 cases with intracerebral haematoma and 21 of the 41 cases with only subarachnoid haemorrhage were admitted after a coma-producing haemorrhage. This suggests that intracerebral and intraventricular rupture is an important factor in producing coma and supports the high incidence of coma-producing haemorrhages after rupture of anterior communicating aneurysms.

SECONDARY HAEMORRHAGE AND NECROSIS DUE TO DISPLACEMENT OF THE BRAIN-STEM

Fourteen of the 30 cases with intracerebral haematomata alone and 14 of the 30 cases with cerebral infarction alone had secondary haemorrhage and necrosis of the brain-stem. An almost identical proportion, namely 15 of the 32 cases with both infarction and haematomata, had similar brain-stem lesions. Thus both intracerebral haematomata and cerebral infarction were equally likely to produce this complication.

SUMMARY

Examination of the brains of 103 consecutive cases of ruptured cerebral berry aneurysm showed that 62 out of the 103 cases had intracerebral haematomata, of which the incidence was higher in men than in women.

Anterior communicating and anterior cerebral aneurysms resulted in intracerebral haematomata more frequently than aneurysms at other sites and commonly produced haematomata in the frontal lobes and cavum of the septum pellucidum and less commonly in the corpus callosum. Middle cerebral aneurysms most frequently produced haematomata in the external capsule and less frequently in the temporal and frontal lobes.

Internal carotid aneurysms at the origin of the posterior communicating artery usually ruptured into the temporal lobes. Aneurysms at the bifurcation of the internal carotid artery were often buried in the frontal lobe and ruptured straight into it.

Haematomata due to vascular hypertension were compared with those resulting from aneurysms. The main differences were the preponderance of external capsular haemorrhages in the hypertensive cases and of frontal haemorrhages in the aneurysmal cases. Discrete thalamic and caudate haematomata were considered as being almost always due to hypertension and caval and callosal haematomata to aneurysms.

The largest haematomata, following the rupture of an aneurysm, were in the external capsule. Those in the cavum were small.

In the large cerebral fissures aneurysms produced subarachnoid haematomata before rupturing into the brain in some cases, most frequently with middle cerebral and anterior communicating aneurysms.

Previous subarachnoid haemorrhages from an aneurysm were found to make it more likely to rupture into the brain with subsequent haemorrhages. They were also found to increase the likelihood of the ventricles becoming dilated with blood after intraventricular rupture thus probably increasing the chance of a fatal outcome from even a relatively slow intraventricular haemorrhage in subsequent haemorrhages.

A larger proportion of the cases with intracerebral haematomata were admitted to hospital after a coma-producing rupture of an aneurysm than was the case with aneurysms that ruptured into the subarachnoid space alone.

There was a similar incidence of secondary brain-stem haemorrhage and necrosis due to transtentorial herniation in cases with intracerebral haematomata and in the cases with significant cerebral infarction.

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 at St. George's Hospital, 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, Wimbledon, for their interest, encouragement and skilled criticism. My thanks are finally due to the technical, photographic, and secretarial staff whose assistance was invaluable.

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