

## CONTRAST MEDIUM EXTRAVASATION FROM ANEURYSMAL RUPTURE DURING CEREBRAL ANGIOGRAPHY

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The radiographic demonstration of contrast medium extravasation from a rupturing intracranial aneurysm is rare. We have detected this occurrence a mere thrice in 15 years, a period in which angiography was routinely used for the investigation of subarachnoid haemorrhage. We have added these three cases to 14 others that were previously reported in the literature and summarised the total. There does not appear to be a common aetiological factor, though further experimental studies on intracarotid pressures distal to the site of injection seem to be warranted. Ventricular opacification during cerebral angiography occurred in five of the 17 cases: in our most recent case the contrast medium outlined intraventricular haematomata, a finding not hitherto reported in the literature and a feature of prognostic significance. Mortality in this series was extremely high and in excess of that usually found either with an aneurysmal re-bleed or an intracerebral haematoma or both. However, although a combination of these two lesions is notoriously hazardous, perhaps the added insult of the contrast medium extravasation is the cause for the inordinately high fatality rate. Only three of these 17 cases survived, two of whom had a significant residual neurological deficit.

Ever since attention was drawn to the relationship between subarachnoid haemorrhage and a leaking intracranial aneurysm, arteriographic techniques have been the only indirect method for safely delineating the causative lesion pre-operatively. Over the years opinion of the hazard of cerebral angiography has varied particularly when initial attempts were fraught with many complications. In 1934, Egas Moniz reported a mortality rate of 2–3%. More recently Feild *et al.* (1962) evaluated the risks of cerebral angiography in a series of 2000 patients of all ages. In the 2332 arteriographic studies performed for a variety of clinical diagnoses, the total incidence of all complications was 2.1%; 1.4% were mild, 0.34% were severe and 0.34% were fatal. In this latter category, two deaths were attributed to a ruptured aneurysm, but in only one of these was the aneurysm shown to be bleeding at the time of the angiogram. Sutton (1969) emphasised that this is an excessively rare event.

We have detected extravasation from an aneurysm three times over the past 15 years, a period in which 775 aneurysms were demonstrated angiographically, giving an incidence of 0.38%. The most recent example stimulated a review of the literature in an attempt to find a common aetiological factor. Although in many cases morbidity and mortality were directly attributed to this complication during angiography (Gallagher and Yamamoto, 1956; Liu, 1965) only 14 unequivocal examples of radiographic visualisation of intracranial contrast medium extravasation from a

rupturing aneurysm could be collected. To these cases we contribute our three cases and summarise the total as shown in Table 1.

### CASE REPORTS

**Case 1.** — A male (H.G.) aged 87 years, was found unconscious and was brought to hospital where he was found to be unconscious, but responding to deep pain with feeble movements of the arms. The left pupil was pinpoint and the right pupil was of moderate size. Both pupils were fixed. Examination of the right fundus showed haemorrhages and papilloedema. The patient was deteriorating steadily and an emergency right carotid angiogram was performed (see Figs. 1, 2). The anterior cerebral artery was displaced to the left of the midline; there was also displacement of peripheral vessels of the middle cerebral group away from the inner table of the vault over the mid and low convexity due to a large subdural haematoma. The aneurysm arose from the internal carotid artery at the origin of the posterior communicating artery and contrast medium was seen to extravasate posteriorly from the aneurysm. There was no spasm of the adjacent vessels.

The patient was taken to the operating room where a right fronto-temporal boneflap was turned. After evacuation of a large intracerebral haematoma, the aneurysm was exposed but when clips were applied to it, it ruptured; the internal carotid artery was therefore clipped. The patient continued to deteriorate and died the following day.

**Case 2.** — A male (R.K.) aged 30 years, was well until he suffered a sudden onset of severe headache and became drowsy. He was admitted to hospital where a clinical diagnosis of intracerebral or subarachnoid haemorrhage was made. Eleven hours after the initial symptoms, he was found to have a left sided hemianopia with a left hemiparesis involving the arm more than the leg, and a central facial weakness. The morning after admission a right carotid

Table 1

No.	Reference	Age/sex	Aneurysms	Site of leak	Size	Extravasation			Spasm
						Subarachnoid space	Intra-ventricular	Local	
1	Jamieson (1954)	36 M	1	(L) M.C.A.				+	
2	Jenkinson <i>et al.</i> (1954)	49 F	?2*	(R) I.C.A.		Down pontine cistern to foramen magnum			
3	Jackson <i>et al.</i> (1960)	61 M	4†	(R) I.C.A.	8 mm			+	
4	Feild <i>et al.</i> (1962)	? F	1	(R) posterior communicating				+	Right middle fossa
5	Triska (1962)	58 F	1	(L) M.C.A.				+	Left temporal lobe
6	Pool & Potts (1965)		2	Posterior communicating				+	Temporal lobe
7	Goldstein (1967)	51 F	1	(R) posterior communicating	Large				
8	Murphy & Goldberg (1967)	23 M	1	(R) I.C.A.	Large		Pontine cistern S.A.S.	+	3rd ventric.
9	Beamer <i>et al.</i> (1969)	55 F	3	(R) I.C.A.			Over hemispheres	+	Right cerebral hemisphere
10	Ferrari & Vio (1969)	38 F	1	(L) I.C.A.			S.A.S.		Right temporal lobe
11	Hoff & Potts (1969)	46 M	1	Fusiform dilated lower basilar artery	Large		Pontine cistern		4th ventric.
12	Vines & Davis (1971)	42 F	1	(R) posterior communicating			Basal S.A.S.		
13	Lehrer <i>et al.</i> (1972)	84 F	1	Anterior communicating	Large			+	
14	Teal <i>et al.</i> (1973)	41 F	2	(R) I.C.A.	Large		Floor of right middle cranial fossa	+++	Right temporal lobe
15	H.G.	87 M	1	(R) posterior communicating	0.7 cm			+	Right temporal lobe
16	R.K.	30 M	1	(R) M.C.A.	2.2 cm			+	Right temporal lobe
17	R.J.	31 F	1	(L) M.C.A.				+++	

No.	Reference	Artery injected	Needle/catheter	Contrast medium	Quantity of contrast/ml	Injection method	Fate
1	Jamieson (1954)	L.C.C.A.	Percutaneous puncture, 3 in., 18 gauge needle	Uridone 40%	15	Manual	Died 10 h later after aneurysm clipped
2	Jenkinson <i>et al.</i> (1954)	R.I.C.A.	Percutaneous puncture	Diodrast 35%	8	Manual	Died 32 h later
3	Jackson <i>et al.</i> (1960)	R.C.C.A.	Percutaneous puncture	Hypaque 50%	5	Manual	Died 2 days later after aneurysm clipped
4	Feild <i>et al.</i> (1962)	R. Innominate and L.C.C.A. →	Catheter simultaneously	Hypaque →	? →	?	Died <24 h
5	Triska (1962)	L.C.C.A. →	18 gauge needle	→	8 →	Manual	Died 36 h later
6	Pool & Potts (1965)	L.C.C.A.	Percutaneous puncture	Urografin 60%	5		Died few hours later
7	Goldstein (1967)	R.C.C.A.	Percutaneous puncture (both common carotids)	Renografin 60%	8	Manual	Died 17 h later
8	Murphy & Goldberg (1967)	R.C.C.A.	Percutaneous puncture	Hypaque 50%	15	Manual	Died 6 days later
9	Beamer <i>et al.</i> (1969)	R.C.C.A.	Teflon catheter needle			Manual	Died rapidly
10	Ferrari & Vio (1969)	L.C.C.A.	Percutaneous puncture	Conray 60%	8	Manual	Right flaccid hemiplegia
11	Hoff & Potts (1969)	L. vertebral	Percutaneous puncture	Hypaque 50%	6	Manual	Died 12 h later
12	Vines & Davis (1971)	R.I.C.A.	Teflon catheter needle	Conray 60%	9	Pressure injector at 200 lb pressure	Aneurysm clipped
13	Lehrer <i>et al.</i> (1972)	R. brachial	No. 17 Cournaud needle	Hypaque 50%	50	Automatic injector at 400 lb/in <sup>2</sup>	Died 3 days later
14	Teal <i>et al.</i> (1973)	R.I.C.A.		Conray	10	Automatic injector at 125 lb/in <sup>2</sup>	Died 71.5 h later
15	H.G. (1977)	R.C.C.A.	Percutaneous puncture	Hypaque 50%	10	Manual	Died 2 h later
16	R.K. (1977)	R.C.C.A.	Percutaneous puncture	Hypaque 50%	10	Manual	Left lower quadrant field defect
17	R.J. (1977)	L.C.C.A.	Percutaneous puncture	Hypaque 50%	10	Manual	Died 36 h later

\* No aneurysm shown at site of extravasation.

† Only two were shown on the angiogram.

L.C.C.A. = left common carotid artery.

R.C.C.A. = right common carotid artery.

R.I.C.A. = right internal carotid artery.

L.I.C.A. = left internal carotid artery.

L.M.C.A. = left middle cerebral artery.

R.M.C.A. = right middle cerebral artery.

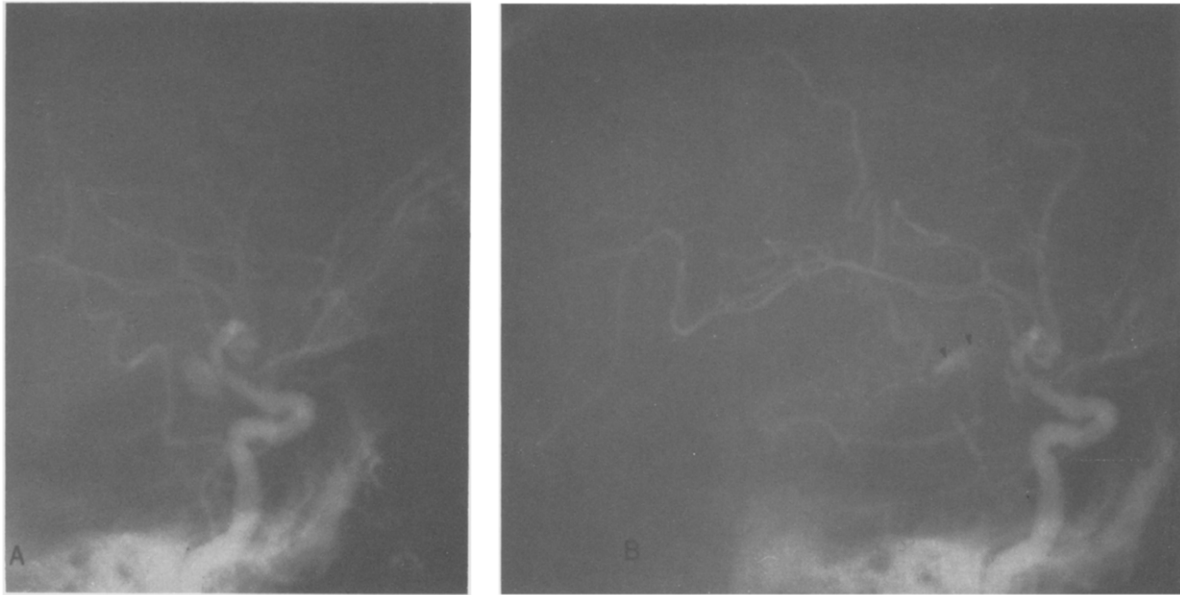


Fig. 1 — Lateral radiographs of right carotid angiogram (H.G.). (a) An aneurysm is shown to arise from the internal carotid artery at the origin of the posterior communicating artery. (b) Rupture of the aneurysm occurs and the arrowheads indicate the position of the contrast medium posterior to the aneurysm shown on 1(a). Irregularity at the origin of the posterior communicating artery is considered to represent the remnant of the aneurysm.

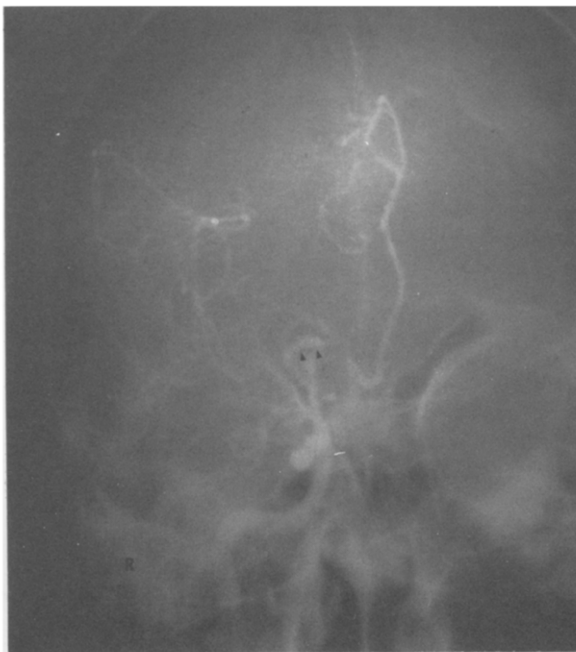


Fig. 2 — Antero-posterior radiograph of right carotid angiogram (H.G.) The anterior cerebral artery is displaced to the left and there is a large subdural haematoma. The arrowheads indicate the position of the extravasation.

arteriogram was performed (see Figs. 3, 4). There was displacement of anterior cerebral vessels to the left of the mid-line and a very marked displacement of middle cerebral vessels: the vessels in the insular area were displaced anteriorly and superiorly with the posterior temporal and angularis branches draped over a large avascular space-occupying mass in the mid-temporal area. This space-occupying mass was considered to be a haematoma related to the very large aneurysm which arose from the right middle cerebral group of vessels beyond its major bifurcation. In addition, contrast medium extended postero-superiorly from the aneurysm indicating extravasation.

The patient was kept on bed rest for 3 days until he began deteriorating and was taken to the operating room. A right fronto-temporal bone flap was turned and a large intracerebral haematoma was evacuated; the aneurysm was ligated and clipped and gauze was packed about the proximal portion of the fundus. The patient tolerated the procedure well, and when last seen 8 months after the operation he was fully restored to his usual state of health except for a complete left lower homonymous quadrantic field defect.

**Case 3.** — A female (R.J.) aged 31 years, was well until early one morning she collapsed and went stiff for about 20 min. Subsequently she was drowsy and was taken to hospital where she responded to commands, but her voice was slurred. Ten minutes later and 14 h after her initial symptoms, her left hand went to her head and she had a grand mal seizure. Her breathing became stertorous and she went into spasm of all limbs. In the postictal period the patient stopped breathing and had to be intubated and ventilated. She became unresponsive to painful stimuli. The right pupil was fixed and of normal size and the left pupil was dilated and fixed. The right fundus was normal, but the left fundus showed a large subhyaloid haemorrhage which increased gradually until it covered the disc and the retina. Plantar responses were upgoing bilaterally. A clinical diagnosis of subarachnoid haemorrhage was made and an emergency left carotid angiogram was performed (see Figs. 5, 6, 7). This demonstrated a single irregular aneurysm arising from the left

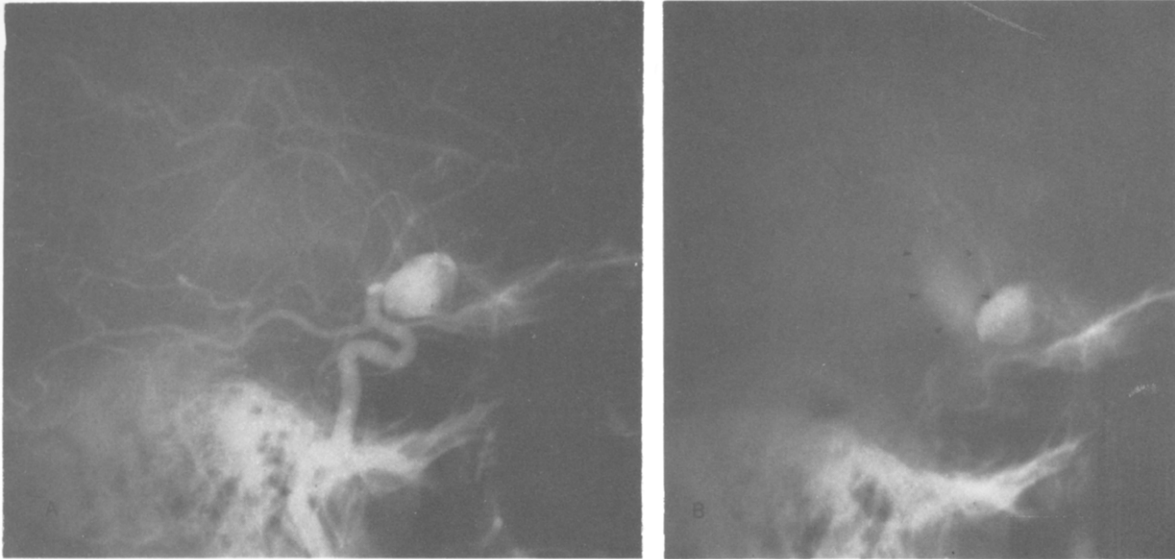


Fig. 3 - Lateral radiographs of right carotid angiogram (R.K.). (a) A large oval aneurysm is shown and there is elevation and stretching of middle cerebral arteries. (b) Extravasation of contrast medium postero-superiorly from the aneurysm is shown (arrowheads).

middle cerebral artery at its bifurcation. Prompt extravasation of contrast medium from the lateral aspect of the fundus was seen to extend into the anterior horn of the left lateral ventricle and subsequently into the right lateral and third ventricles. A slight trickle of contrast was also seen to pass down the aqueduct to the fourth ventricle. The contrast outlined haematomata within both lateral ventricles and probably also in the third ventricle. Some contrast, however, remained locally at the site of the extravasation.

The patient was taken to the recovery room, but died 2 h later.

#### DISCUSSION

Intracranial contrast medium extravasation during cerebral angiography is rare considering the numbers of these studies performed annually and the relative paucity of documented cases in the literature, and this is borne out by the figures cited above.

Many factors contribute to rupture of an aneurysm, not the least being trauma or stress of any kind, presumably because of an abrupt elevation of the blood pressure. Whether an elevation of intracranial arterial pressure occurs in the carotid and/or the vertebro-basilar arterial tree during cerebral angiography has not been proven, though opinions seem to favour no significant elevation of pressure. There have been conflicting reports in the literature concerning this matter; Bakay and Sweet (1952) examined five patients and found no increase in internal carotid pressure when injecting a 10-12 ml bolus of either 35% Diodrast or physiological saline into the common carotid artery in 2-3 s. However, more recently Lin *et al.* (1964) reported a

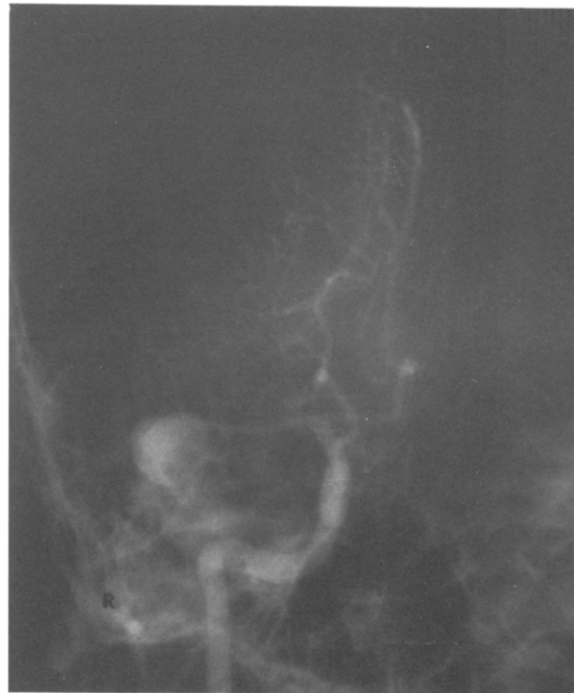
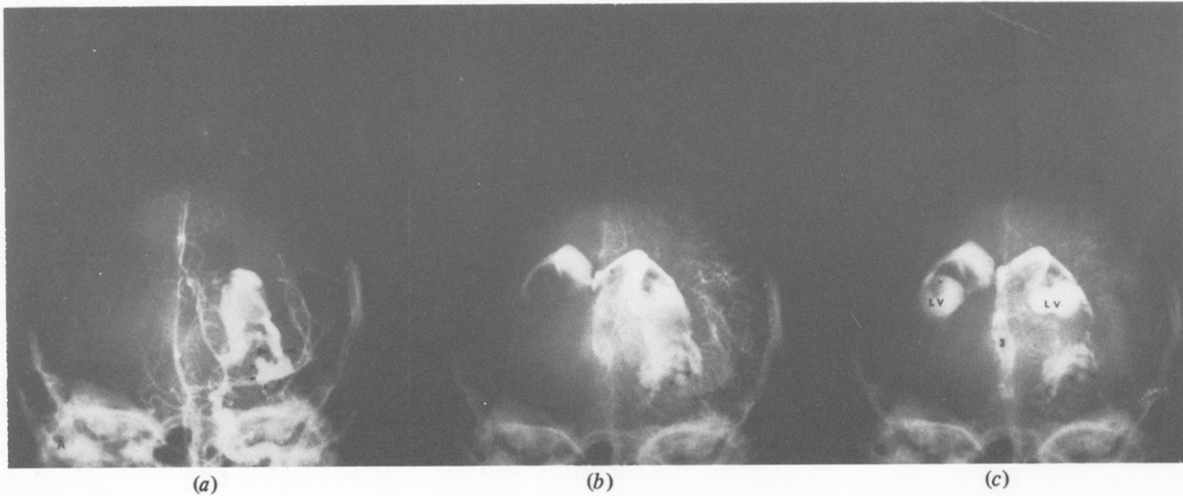


Fig. 4 - Antero-posterior radiograph of right carotid angiogram (R.K.). The large aneurysm is shown to arise from the middle cerebral artery distal to its bifurcation. There is displacement of middle cerebral vessels around an avascular space occupying lesion in the mid-temporal area which is causing displacement of the anterior cerebral artery to the left.



5–10 mmHg. elevation of blood pressure in the ipsilateral carotid artery in one patient after a brachial injection of a 30 ml bolus of contrast medium at 625 lb/in<sup>2</sup>: in four additional patients the contralateral carotid recordings showed elevations from 7 to 25 mmHg, the increases being in direct proportion to the injection pressures which were by hand and at 225 and 625 lb/in<sup>2</sup>. Further evidence suggesting that elevation of intracarotid pressure occurs during cerebral angiography include the statistics quoted by Saltzman (1959), who observed opacification of the posterior communicating or posterior cerebral arteries in 58% of patients with internal carotid injections and in only 28.6% in those with common carotid injections. Similarly, it has been noted that there is an increased incidence of transient opacification of the contralateral proximal anterior cerebral arteries during selective internal carotid angiograms compared to common carotid angiography. Furthermore, in vertebral angiography retrograde opacification of the contralateral vertebral artery is a common occurrence even in the absence of a 'steal' effect.

Intracranial contrast medium extravasation from an aneurysm has been reported only once (Case no. 13) during a retrograde brachial procedure when a bolus of 50 ml was injected at 400 lb/in<sup>2</sup>. Extravasation occurred locally and into the ventricular system from a large anterior communicating aneurysm: those authors believed that the procedure could have contributed to the aneurysmal re-bleed. Likewise, there is only one report (Case no. 11) of extravasation during vertebral angiography when 6 ml were manually injected into a vertebral artery following a percutaneous puncture; a large fusiform aneurysm of the lower basilar artery was shown to be the pathological lesion.

Fig. 5 – Antero-posterior serial radiographs of left carotid angiogram (R.J.). (a) Arterial phase; a single irregular aneurysm is shown to arise from the left middle cerebral artery at its bifurcation (small arrowheads). The extravasation courses upwards (large arrowheads) to enter the left lateral ventricle. (b) Capillary phase; some contrast medium persists within a portion of the aneurysm (arrowheads) but considerable opacification of both lateral ventricles has occurred. (c) Early venous phase; a portion of the aneurysm is still clearly visible (arrowheads) and some contrast remains locally. The lateral and third ventricles are now clearly delineated by the contrast medium.

In three cases (nos. 2, 12, 14) the contrast medium was injected into the internal carotid artery; in one of these (no. 12) an automatic injector at 200 lb pressure injected a bolus of 9 ml while the contralateral common carotid artery was being compressed; in another case (no. 14), a bolus larger than normal was used due to the internal carotid artery being inadvertently cannulated. It is noteworthy that in this latter case an automatic injector at 125 lb/in<sup>2</sup> was used and spasm of the middle and anterior cerebral arteries bilaterally and the supraclinoid left internal carotid artery was present. In one case (no. 14) the right innominate and left common carotid artery were reported to be injected simultaneously with a bolus of 8 ml by manual pressure on the left, but an unspecified bolus and injection method on the right; the rupture occurred from a right posterior communicating aneurysm. In the remaining 10 cases (excluding the brachial and cerebral angiogram and Case no. 6 in which the artery injected was not specified), the contrast medium was introduced into the common carotid artery and injected manually, a method comparable to that used in our department for many years.

Some authors including Ferrari and Vio (1969) have suggested that doses of contrast medium higher than those required for a normal procedure may be a

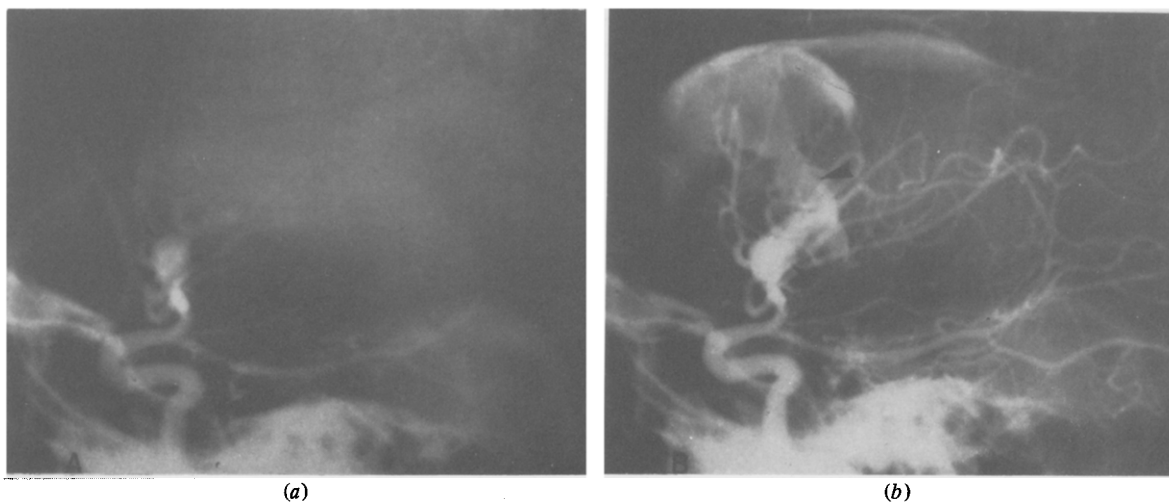


Fig. 6 – Lateral radiographs of left carotid angiogram (R.J.). (a) The aneurysm demonstrated in Fig. 5 is here shown on an early arterial phase film (arrowheads). (b) Rupture of the aneurysm has occurred and the course of the extravasation toward the anterior horn of the lateral ventricle is shown (large arrowhead).

contributing factor to aneurysmal rupture, particularly when injected into a small or spastic vessel. Our review of the literature does not confirm this impression. There were only three cases in which a bolus larger than normal was utilised; in two of these (nos. 1 and 8) 15 ml were injected into the common carotid artery, and in the third case (no. 14) 10 ml were injected inadvertently into the internal carotid artery. Intracranial arterial spasm was shown in two of these three arteriograms, which when combined with the large bolus, may have caused significant increase in the intracarotid pressure.

The summation effect of injections given too rapidly as suggested by Broman and Ollson (1949) may apply to Case nos. 1 and 2 when four and five series of films respectively were obtained. In 10 out of the 17 cases summarised in Table 1 the extravasation was seen after the initial injection on that side and the examination was consequently terminated. It would seem unlikely, therefore, that the summation effect is applicable as an aetiological factor.

Vines and Davis (1971) believed that contralateral carotid artery compression did not contribute to the aneurysmal rupture in their case, on account of this procedure being almost routine in their work without apparent complication. Wright (1962) recorded pressure readings during 89 carotid, nine vertebral and two subclavian angiograms while the contralateral common carotid artery was firmly compressed for periods of 30 s or longer. He calculated an average increase in

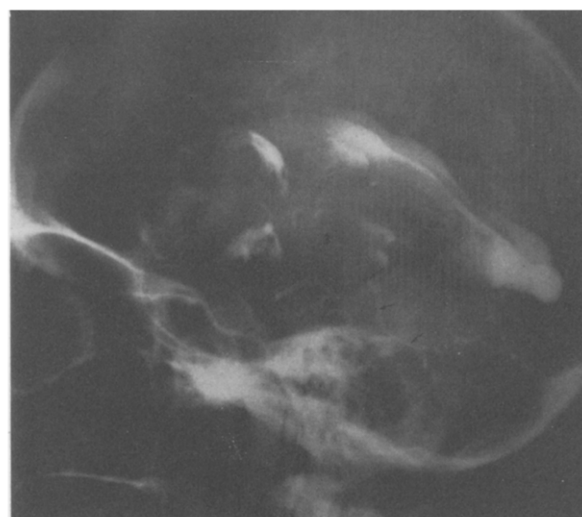


Fig. 7 – Lateral radiograph of left carotid angiogram (R.J.). The majority of the contrast medium is in the lateral ventricles within which masses are seen and considered to represent haematomata. A similar appearance to the third ventricle is shown (3). Some contrast is trickling down the aqueduct (arrows) towards the fourth ventricle.

intra-arterial systolic pressure of 4.2, 7.2 and 10.8 mmHg within the common carotid artery, 5, 15 and 30 s respectively after contralateral compression low in the neck. The response to compression over the region of the carotid bifurcation was variable, but was not considered significant when elevation of pressure did occur. He concluded that compression is a potentially dangerous procedure in patients with recent subarachnoid haemorrhage particularly when compression was applied to the carotid artery low in the neck. The case reported by Vines and Davis (Case no. 12) is the only one in this series in which

contralateral compression was apparently used. Hence, the insufficient evidence to implicate this procedure.

A refinement in contrast media has occurred since Moniz's initial successes with 70% strontium bromide. In his search for a better medium, he chose sodium iodide and colloidal thorium dioxide respectively, having reported a 2.6% mortality with the former agent and a 1.3% mortality with the latter. A wide variety of relatively non-toxic water-soluble agents are now available and as seen in this small series, no one agent dominates the market. One cannot incriminate any of these agents alone as a possible aetiological factor in a rupturing aneurysm during angiography.

Taylor (1961) believed that the natural tamponade effect of the intracranial pressure and also the brain tissue around the aneurysm tended to prevent rupture. This theory is supported by this series and by the study of BJORKESTEN and TROUPP (1962) who found that aneurysms of the carotid and posterior communicating arteries which lie partly exposed in the subarachnoid space are more likely to rupture than aneurysms in the middle cerebral artery which are protected by the surrounding brain tissue. In this series, 11 aneurysms occurred on either the internal carotid artery or posterior communicating artery whereas only four occurred on the middle cerebral artery. It is clear, too, that the anatomical localisation of the aneurysm determines the pattern of contrast medium extravasation: those which lie in close proximity to the subarachnoid space invariably rupture into that space as seen in seven of the 11 aneurysms on the internal carotid and posterior communicating arteries as well as the basilar artery aneurysm. However, the pattern of contrast dissemination is not always typical, with extravasation from three of the posterior communicating aneurysms being confined to the local brain tissue and in one of the internal carotid artery aneurysms the leakage was confined locally. In contrast, the extravasations from the four middle cerebral artery aneurysms, did not pass into the subarachnoid space.

The opacification of a part or whole of the ventricular system from a rupturing aneurysm during cerebral angiography is an even more rare occurrence and was seen in only five of these 17 cases. In four, the opacification occurred as a result of direct flow of contrast medium from the site of rupture, whereas in one, the opacification occurred via the basal subarachnoid space and thence into the fourth ventricle. In our most recent case (no. 17) the contrast medium showed clear evidence of intraventricular haematomata, a feature which may have prognostic significance. In Pia's study (1968) of

intraventricular haemorrhage from all causes, he found a mortality rate of 100% when the entire ventricular system is filled with haematoma, 56% when blood masses fill circumscribed portions of the ventricular system, and only 14% when the ventricular cerebrospinal fluid contained fresh blood without signs of a haematoma. Our patient died less than 2 h following the angiogram, and despite the absence of autopsy proof it would appear to be the only case reported in the literature in which intraventricular haematomata have been demonstrated during cerebral angiography.

The comprehensive report by PERRET and NISHIOKA (1966) on the cooperative study of intracranial aneurysms and subarachnoid haemorrhage showed an overall incidence of complications of 7.6% with 7933 angiographic studies performed on 5484 patients. Interestingly, this figure does not include 67 cases in whom recurrent subarachnoid haemorrhage was stated to have occurred during or after angiography since it was considered by the authors that a re-bleed from an aneurysm was part of the natural course of the disease and therefore not necessarily attributable to the study. However, they reported a fatal complication with a plausible relationship between the angiogram and death in 64 patients representing a mortality of 1.17%. Within this group a re-bleed during angiography was reported to have occurred in seven cases, but in only one instance was extravasation of contrast medium out of the aneurysm seen on the radiographs. It was not feasible to add this case to the series in Table 1 because no further data were available in the report, though it is possible that the case may have been previously reported in the literature and, unknown to us, is already included in our series. We are aware of six further cases in which an intracranial aneurysm was shown to be bleeding at the time of angiography (TAVERAS, 1964; SUTTON, 1969; CHIU, 1973; STEVENS, 1974; TOLAND, 1974) but insufficient data prevented their inclusion in this series.

It has been well established that complications during cerebral angiography are more frequent with advancing age. In this series of 10 females and 6 males (in one the sex was not specified) the ages of 15 patients are known and range from 23 to 87 years with a marked predominance in the fourth, fifth and sixth decades, a pattern consistent with that usually seen in patients who present with subarachnoid haemorrhage. The average age of the three survivors was 36.6 years whereas in those who died (and whose ages are known) the average age was 51.8 years. Similarly, when cerebral angiography is performed for the investigation of subarachnoid haemorrhage the morbidity and mortality is increased, and the com-



plications are probably related to the injurious effect of the initial aneurysmal bleed.

When discussing the prognosis in spontaneous subarachnoid haemorrhage, Hyland (1950) stated that approximately 50% of patients recover from the attack that brings them under observation and that patients with multiple episodes of bleeding have only about one-half the chance of survival of those with a single episode. If one assumes that the request for an angiographic search for the source and site of intracranial bleeding is based on a clinical suspicion of subarachnoid haemorrhage, one must conclude that extravasation from an aneurysm shown on the radiographs is at least the second episode and consequently the relatively high mortality should be expected. Nevertheless, we suspect that the mortality in this series is inordinately high and suggest that the contrast medium extravasation may be a contributing factor to this high fatality rate.

**Acknowledgements.** — We would like to thank Drs T. Morley, W. Loughheed, and R. Tasker for permission to publish these cases, the Medical Photographic Department at the Toronto General Hospital, and Mrs B. Mistal who typed the final manuscript.

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