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Balloon Catheter Technique for Dilatation of Constricted Cerebral Arteries After Aneurysmal SAH

By

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With 4 Figures

Summary

The authors describe the technique and explain the reason for the use of an intravascular balloon catheter technique in the treatment of vasospasm in cases of subarachnoid haemorrhage due to rupture of a cerebral aneurysm.

In 33 patients with SAH 105 major cerebral arteries were dilated (in the system of internal carotid and basilar arteries). Only in one case vasodilatation could not be accomplished. The effect of dilatation of vasospastic arteries was stable and the functional state of the brain was ameliorated, local and general cerebral disfunctional signs and symptoms due to vasospasm regressed.

The authors discuss indications and contraindications for intravascular balloon dilatation of spastic arteries, the time for the operation, the length of arteries to be dilated, and the possible mechanism of amelioration of cerebral blood flow to the brain stem.

Keywords: Subarachnoid haemorrhage; cerebral aneurysm; vasospasm; balloon catheter technique; intravascular stretching.

During the last two decades a great number of studies have been undertaken to elucidate the pathophysiology of cerebral vasospasm and delayed ischaemic dysfunction after aneurysmal subarachnoid haemorrhage (SAH). The significance of various mechanisms possibly involved in the phenomenon remains controversial. A large number of different substances have been suggested to account for the pathogenesis of cerebral vasospasm.

The presence or absence of cerebral vasoconstriction after the aneurysm rupture implies an interaction between a vasoconstrictor

substance in the post-haemorrhagic period and protective mechanisms in the arterial wall to maintain normal vascular tone.

Experimental SAH in dogs and monkeys produces severe ultrastructural vascular changes which can be demonstrated 3 to 7 days after SAH[1, 2, 3]. These changes consist of vacuoles and dense bodies, infrequently detached endothelial cells, an appearance of intimal hyperplasia, and platelet adherence. The morphological findings indicate that the intima, where prostacyclin (PGI 2) is synthesized, is specifically subjected to progressive injury after aneurysmal SAH. This may cause a decrease in PGI 2 synthesis and release which may result in a sustained contraction of the cerebral arteries and arterioles, as suggested by Sano *et al.*[4, 5].

In the treatment of cerebral vasoconstriction after SAH different procedures have been advocated (0.5–1 per cent solution Novocain i.v. or intraarterially, novocain blocks of the cervical sympathetic chain, application of novocain, papaverin, kanamicin solutions on major cerebral arteries during operation on aneurysms, early surgery on the ruptured aneurysm with washing out of blood clots from the basal cisterns[14, 15] and so on). It has been also shown that Ca^{++} antagonists induce relaxation of arteriolar constriction (L. Brandt 1981. More recent literature is collected in "Aneurysm Surgery in the Acute Stage", Acta Neurochirurgica 63, Fasc. 1–4, 1982.) The results of these procedures have been contraversial; in some cases the effect of treatment was very encouraging, while in others no result was obtained.

During the past 14 months we have been treating patients with intracerebral vasospasm after aneurysm rupture with good results by intra-arterial balloon dilatation of constricted major cerebral arteries.

Clinical Material and Method

The procedure was performed in 33 consecutive patients.

The site of the ruptured aneurysm was a follows: supraclinoid—6, anterior communicating artery (ACoA)—18, middle cerebral artery (MCA)—7, posterior cerebral artery (PCA)—1, vertebro-basilar system—1. Age distribution and localization of vasospasm treated by vasodilatation are shown in Table 1.

The indication for balloon-catheter intravascular stretching was local segmental, bilateral segmental, local diffuse, and bilateral diffuse vasospasm of cerebral arteries with signs of impairment of cerebral blood flow and local cerebral disfunction: aphasia, hemiparesis or plegia, and signs of brain stem involvement.

Angiography was performed in most cases under local anaesthesia, a solution of 60% verographin was used, usually 10 cm^3 for an injection.

Table 1. *Age Distribution and Localization of Spasm Treated by Vasodilatation*

Age	21–30	31–40	41–50	51–60	Total
Spasm					
Internal carotid artery system					
Unilateral segmental	1	1	1	4	7
Bilateral segmental	—	1	—	—	1
Unilateral diffuse	1	2	2	2	7
Bilateral diffuse	1	3	9	3	16
Vertebro-basilar system					
Diffuse	1	1	—	—	2
Total	4	8	12	9	33

The intravascular dilatation was performed under neuroleptanalgesia and local anaesthesia. The balloon catheter was introduced into the internal carotid artery through a needle puncture of the common carotid artery, while in cases of spasm in the vertebro-basilar system the retrograde catheterization technique of Seldinger was used.

The particular construction of the balloon catheter is such that the catheter has different elasticity along its length. The main catheter is elastic, while its end, to which the balloon is attached (20–30 mm) is more elastic. The balloon used in these cases is small (3–4 mm in length) and its diameter after dilatation does not exceed the diameter of normal intracranial basal arteries that have to be dilated by 10–15%. This permits the introduction of the balloon catheter to the site of vasodilatation without increasing the balloon in size, so that there is no risk of rupturing the spastic artery.

The vasodilatation is performed in the X-ray room. A Siemens angiograph has been used with television control of vasodilatation.

The dilatation of vasospastic arteries was performed in stages beginning with the proximal portion of the artery. The procedure was controlled optically by angioscopy through the electronic optic system (television control) or angiographically. The effect of vasodilatation is achieved by one dilatation of the balloon at a time, for a few seconds. Using this technique we have not had any complications so far, and the cerebral blood flow was not impaired during the procedure.

In most cases the operation was performed under control of the functional state of the brain, and the cardio-vascular system (EEG, ECG, photoplethys-

mography). Before and after the intravascular operation not only angiographic control was performed, but in some cases the functional state of the internal carotid artery and regional cerebral blood flow were investigated using the technique of intra-arterial Xe-133 injection. Also repeated postoperative arteriograms were performed during the first month after the operation.

105 vessels were dilated. In 5 patients the cerebral arteries were dilated after clipping of an aneurysm.

In 6 patients not only vasodilatation was achieved but the aneurysm was excluded from the circulation by the introduction of the balloon catheter into it or into its neck.

Results and Discussion

Dotter *et al.* (1964) proposed dilatation of the segmentally stenotic major arteries of the extremities using the technique of gradual internal dilatation utilizing biliary duct dilators inserted into the artery and passed to the level of stenosis, and later by the balloon catheter technique of Fogarty.

Suzuki *et al.* (1967) have tried to dilatate the vasoconstricted segment of the internal carotid artery using saline solution containing heparin that was introduced into the vessel under pressure, while the proximal and distal ends of the vessel were temporarily occluded. The saline solution dilatation of the vessel was associated with the excision of the upper cervical sympathetic ganglion.

The use of the balloon catheter technique for the dilatation of a stenotic internal carotid artery was reported by Mullan *et al.* (1980).

As far as we know there are no reports of the use of the balloon catheter technique for the dilatation of vasoconstricted arteries of the brain after aneurysm rupture.

In all patients but one the vasodilatation was performed without difficulties. In our opinion these results could only be achieved by using the special balloon catheters. In one patient with a saccular aneurysm of the ICA it was impossible to dilate the supraclinoid portion of the ICA. The operation was performed at the end of the third week after SAH. The most probable reason for the unsuccessful intervention was the advanced age of the patient and a probable stenosis of the artery due to atherosclerosis or to organic changes in the arterial walls as described by Sakaki *et al.* (1979). Maybe the reason for this unsuccessful operation was our lack of experience, as it was our second operation of this kind.

In Table 2 the time interval between the SAH and vasodilatation of spastic cerebral arteries is shown.

In 18 patients the spasm has been demonstrated during angiography performed on the 1–2 day of hospitalization. In these patients vasodilatation has been performed immediately after angiography. In 15 patients the indications for vasodilatation were signs of aggravation of cerebral ischaemia observed during clinical

examination and medical treatment, in 10 patients before direct surgical intervention on the aneurysm and in 5 cases after the operation. Vasodilatation in this group was performed during the first 3 days after deterioration (on the first day—11 patients, on the second—1, and on the third—4 patients).

Table 2. *Number of Days Between SAH and Vasodilatation*

	Number of days after SAH						
	1–3	4–7	8–10	11–14	15–21	over 21	Total
Vasodilatation before operation on aneurysm	—	2	11	7	7	1	28
Vasodilatation after operation	—	1(1)	1(1)	2(4)	1(14)	—	5
Total		3	12	9	8	1	33

Figures in brackets indicate the day of direct operation after SAH.

Table 3. *Number of Days Between Vasodilatation and Direct Operation on Aneurysms*

Number of days after vasodilatation				Total
1–3	4–5	8–10	11–14	
7	6	3	2	18

Clinically, after vasodilatation the patients noticed a decrease and subsidence of local headache and regression of focal neurological signs. The positive effect of vasodilatation was also determined by investigation of cerebral blood flow and EEG (more detailed information will be given in another report).

In our opinion the vasodilatation treatment of patients in a grave condition due to cerebral ischaemia permitted us to hasten the direct operation on the aneurysm because of the improvement of the functional state of the brain.

Table 3 shows the time in days between vasodilatation and

clipping of the aneurysm in 18 patients. In 6 patients the prolonged occlusion of the aneurysm was performed simultaneously to vasodilatation, in 5 vasodilatation was done after intracranial clipping of the aneurysms, 4 others were not operated on after vasodilatation because of their poor condition (in two of them blood clots had occluded the ventricles, while in two others there was extended ischaemia of the brain).

Out of 33 patients who underwent vasodilatation 7 died: 2 after clipping of the aneurysm, one after balloon occlusion of the aneurysm because of thrombembolic occlusion of the pulmonary artery; the cause of death in 4 other patients was mentioned above.

The total death rate in this group of patients is 21–.

To determine the effectiveness of the method of treatment of prolonged vasospasm after aneurysm rupture it is important to discuss the following questions:

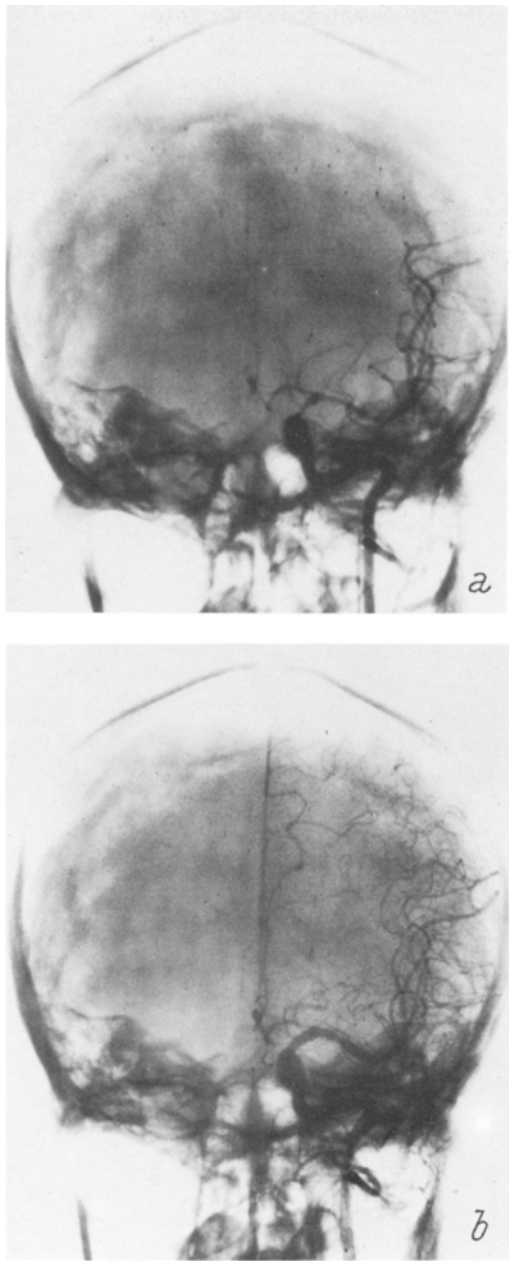
1. Is the effect of vasodilatation persistent?
2. The length of constricted vessel to be dilated.
3. Indications and time limits for intra-arterial vasodilatation in cases of cerebral arterial aneurysm rupture.
4. What is the pathogenesis of dilatation of constricted arteries after the endovascular interventions?

The angiographic investigation of patients who underwent intraarterial vasodilatation showed that the segments of arteries that were stretched by balloon catheter sustained their form during the follow-up period, that is one month after the operation. So we can conclude that the effect of vasodilatation is persistent.

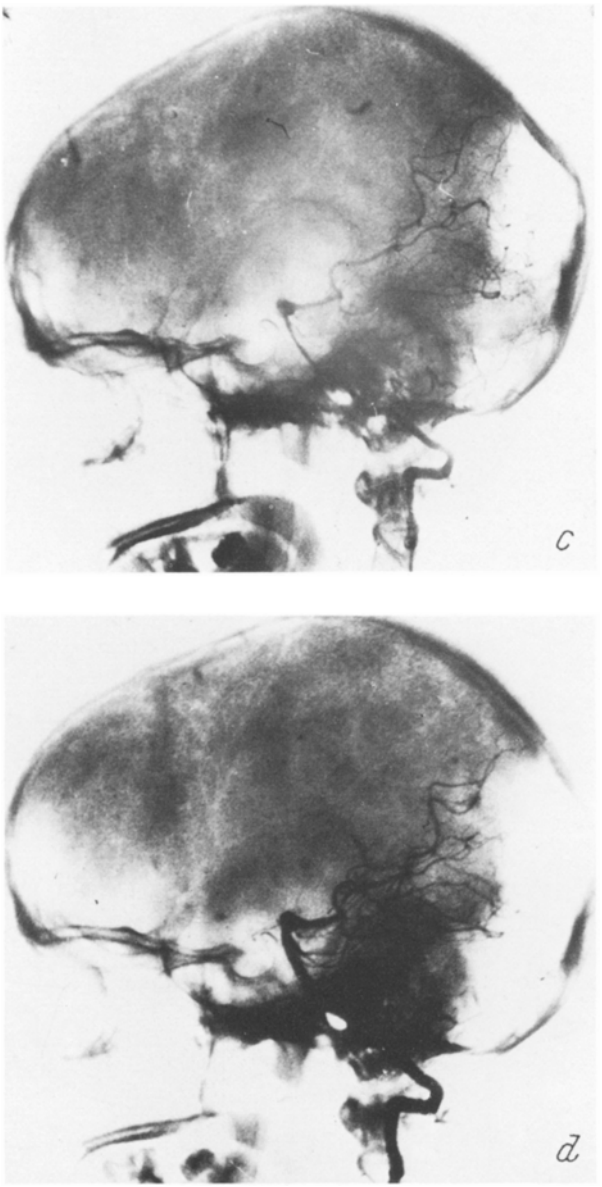
We managed to abolish the vasospasm of the ICA, vertebral and basilar arteries (in major trunks and primary branches of these arteries) (Figs. 1 a, b, c, d). A very important point in this connection is the use of appropriate balloon catheters. In the case in which the diameter of the dilated balloon is larger than the lumen of the vessel to be dilated, the patient may complain of local pain during the operation, which is due to overdistention of the vasoconstricted artery (Yu. N. Zubkov 1974, A. G. Lysatchev 1979).

It is particularly interesting to notice that the diameter of segments of arteries not dilated during the procedure have the same configuration in control angiograms during the days following the operation, being near to the dilated portion of the artery.

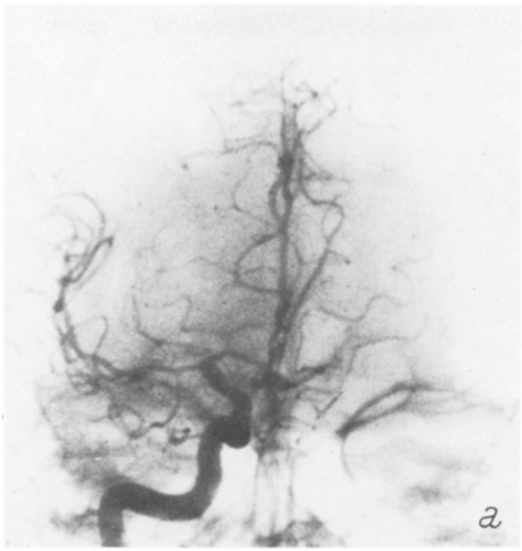
This is well illustrated by angiograms from a patient with vasospasm in the internal carotid arterial system, in whom after the first endovascular operation the



gs. 1 a and b. Angiogram before and after balloon catheter dilatation of arterial spasm of the ICA and the MCA



Figs. 1 c and d. Angiogram before and after balloon catheter dilatation of basilar artery



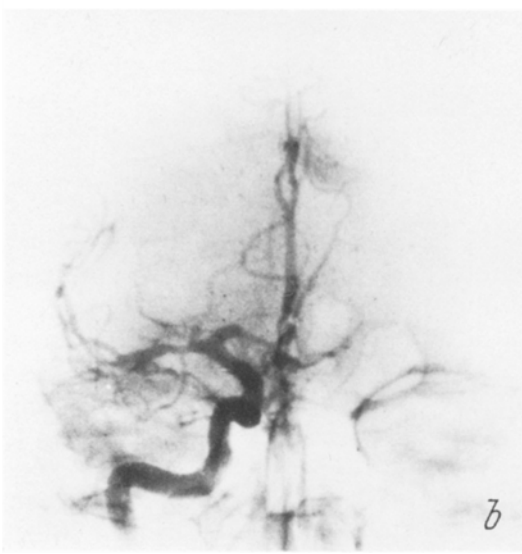


Fig. 2. a) Preoperative film showing vasospasm of internal carotid artery in a case of ruptured ACoA aneurysm. b) Angiogram after balloon catheter dilatation of the ICA, ACA (AI), and MCA. c) Angiogram after additional dilatation of the MCA and balloon occlusion of the AI and ACA (two days after first dilatation)

MI portion of the MCA was only dilated in segments throughout its length (Figs. 2a, b, c). During the second operation the AI portion of the ACA was dilated, as well as segments of middle cerebral artery previously not dilated, so that finally all the main trunk of the MCA was dilated.

In two patients with diffuse vasospasm due to SAH an aneurysm could be visualized only after vasodilatation.

Fig. 2c

In solving the problem of the extent of vasodilatation to be performed we took into consideration the data obtained from the literature that after SAH vasoconstriction is usually seen only in major cerebral arteries which are in the basal cisterns. In this connection it can be supposed that diffuse constriction of cerebral arteries in patients with ruptured cerebral aneurysms, frequently seen in angiograms, was due not to the decrease in the lumen of arteries that lie beyond the basal cistern but to the decrease in cerebral blood flow. This thesis was confirmed by our investigation. For example, after dilatation of the MI segment of the MCA only, one could see the dilatation of MCA beyond its trifurcation in angiograms (Figs. 3 a, b).

This permits us to come to the conclusion that the limit of endovascular dilatation to be performed should be vessels lying within the basal cisterns. It is probable that vasospasm of the ACA

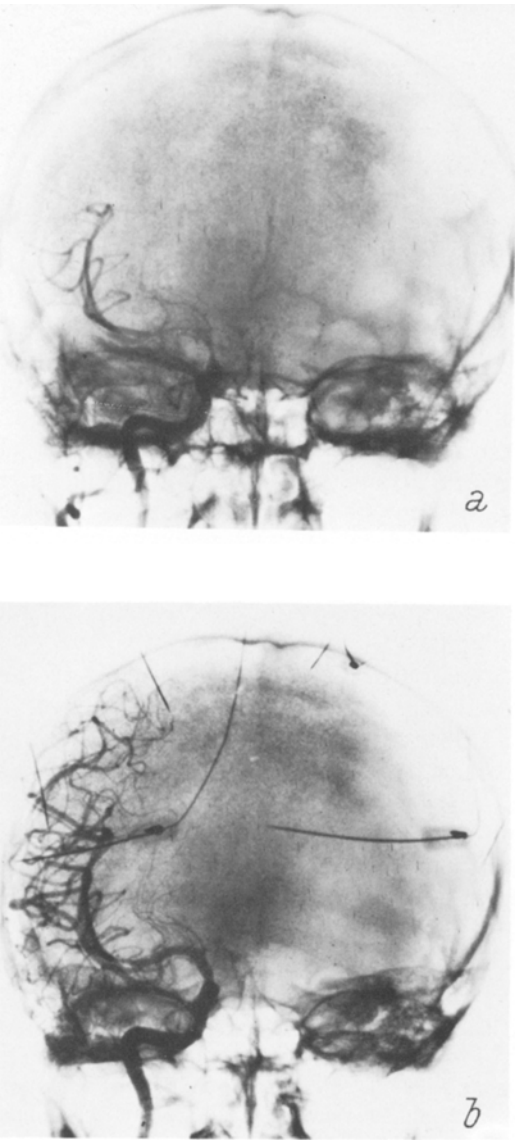


Fig. 3. a) Film demonstrating ICA spasm. b) Film after balloon catheter dilatation of the ICA and MI MCA. Vessels of the MCA are contrasted better

vessels is due to the prolonged contact of these vessels with blood that has invaded the longitudinal fissure, and that endovascular dilatation may not be very effective in these cases.

The most important questions to be answered are the indications and contraindications for vasodilatation and the appropriate time for the procedure.

The limited number of cases does not permit us to answer the question fully, but our experience shows that the operation can be performed in practically all the patients with SAH in the acute stage of the haemorrhage when there is a prolonged vasospasm in the angiograms, especially in association with signs of cerebral blood flow impairment and clinical signs of brain stem and cerebral hemispheric involvement. Vasodilatation was of temporary effect in patients with massive haemorrhage from the ruptured aneurysm complicated by intracerebral haematoma penetrating the ventricular system and also in cases of massive ischaemia due to spasm treated late.

The simplicity of the procedure permits it to be performed immediately after angiography. Moreover, in some patients the aneurysm can be occluded simultaneously by introduction of the balloon catheter into the aneurysm.

The same technique can be used also after the clipping of aneurysms. As it is known, in patients operated on for aneurysms soon after SAH in the postoperative period a vasospasm can develop. This may be the cause of worsening of focal and general cerebral signs and even lead to the patient's death.

In our opinion, post-operative vasospasm in most cases is the continuation of the pathological process seen in the arterial wall due to SAH. Hence the indication for vasodilatation can be broadened and the balloon catheter technique can be used after direct intracranial operations on aneurysms complicated by vasospasm. The following case illustrates the efficiency of such a procedure in the post-operative period.

This 40-year-old patient was admitted to the neurosurgical unit on the first day of illness complaining of acute pulsating headache and vomiting. On examination he was drowsy, with an upward conjugate gaze palsy. There was weakness of the left lower half of the face and a mild neck stiffness. The AP was 120/100, pulse 72, respiration was normal. On lumbar puncture haemorrhagic cerebrospinal fluid was obtained. Bilateral carotid angiography was done. On the right side there was hypoplasia of the AI segment, so that the internal carotid artery continued into the middle cerebral artery. On the left side an aneurysm was revealed of the AI portion of left ACA at the site of its bifurcation into A 2. There was a definite cerebral

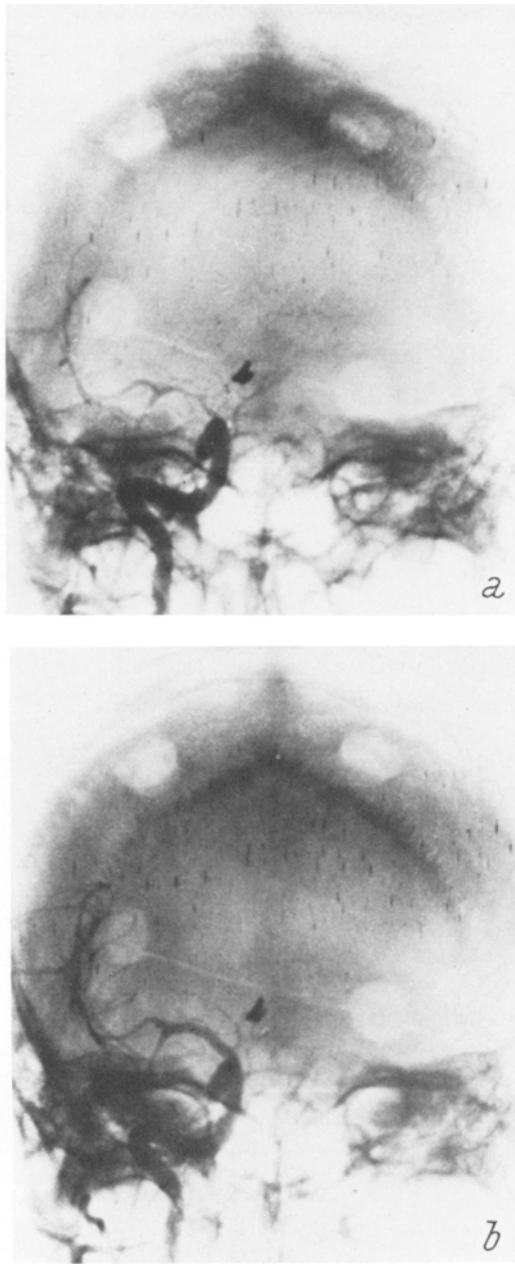


Fig. 4. a) Angiogram demonstrating ICA and MCA spasm in a patient operated on for an ACoA aneurysm. b) Angiogram after vasodilatation

vasospasm. On the 4th day of SAH direct surgery of the aneurysm was undertaken through a craniotomy in the left fronto-temporal region with the aneurysm neck clipping.

On the first post-operative day the patient's state was normal, but on the 10th post-operative day, 14 days after SAH, the patient's condition deteriorated with worsening of general and focal cerebral signs. He became drowsy, desorientated, his headache accentuated and a left-sided hemiparesis appeared. The right carotid angiogram revealed a diffuse spasm of the ICA and the MCA. After this investigation a balloon catheter dilatation of these arteries was performed (Figs. 4 a, b). The next day the focal and general cerebral signs started to regress.

The peculiarity of this case was that vasospasm developed on the side of hypoplasia of the AI and ACA, on the side not directly connected with the aneurysm and surgical manipulation.

The mechanism of endovascular dilatation of spastic arteries has not been elucidated. It can be supposed that during the prolonged cerebral vasospasm the walls of the cerebral arteries lose their elasticity, while the muscular layer becomes degenerated and loses its contractile ability after the degenerated muscle fibres have been stretched by balloon dilatation. This permits the re-establishment of the internal diameter of vessels by stretching their walls, while the effect of vasodilatation is maintained by endovascular blood pressure, for it is quite probable that the vessel for some time loses its ability to react to nervous and hormonal regulation of its tone. Otherwise it is hard to explain the efficiency of balloon stretching of cerebral vessels, if we take into consideration the theory of Mizukami and Shizuka advocating the leading role of vascular wall oedema, not the spasm itself, in the development of long-term vasospasm. It is quite possible that both these mechanisms play a role in stenosing the arteries after SAH.

However, though the pathogenesis of cerebral arterial spasm after cerebral aneurysm rupture and SAH is controvertial, the effect of the balloon catheter technique in obtaining prolonged vasodilatation of spastic cerebral arteries is obvious as well as the normalization of cerebral blood flow and the regression of focal and general cerebral signs is quite impressive, so this new method of treatment of prolonged vasospasm after cerebral aneurysm rupture in the acute stage may be considered to be quite promising.

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