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The Symptomatology with the Most Severe Clinical Course of Spontaneous Subarachnoid Hemorrhage

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Summary. The symptomatology of 18 patients with the severest clinical course after subarachnoid hemorrhage (SAH) is described and analyzed. Seventeen patients died, five with an acute bulbar syndrome with cardiac arrest, and 12 with irreversible breakdown of brain function. One patient had an apallic syndrome with minimal signs of remission, who died 4 months after the first rebleeding.

The cases are divided into five clinical groups on the basis of the acute brainstem symptomatology which set in instantly or after temporary adaptation, or following a second hemorrhage. Pathological examination revealed that 16 patients had a brainstem pressure cone, 14 with marked edema and signs of herniation; one patient had only cisternal tamponade while another had predominantly brain edema with herniation. There was striking parenchymatous damage of the brain of the patient with the apallic syndrome.

There was a marked analogy between the material reported and the symptomatology described by Plum and Posner (1972).

Key words: Subarachnoid hemorrhage – Brainstem tamponade – Pressure cone – Ventricular rupture – Midbrain syndrome – Bulbar syndrome.

Zusammenfassung. Es wird die klinische Symptomatik der schwersten Verlaufsform einer SAB anhand von 18 Patienten einer genauen Analyse unterzogen. 17 Patienten verstarben, davon 5 durch Herzstillstand im akuten Bulbärhirnsyndrom, 12 nach Eintreten des irreversiblen Zusammenbruchs der Hirnfunktionen. Bei einer Patientin hatte sich ein apallisches Syndrom entwickelt, das geringe Remissionszeichen aufwies. Die Patientin verstarb 4 Monate nach dem Aktualereignis. Nach dem klinischen Verlauf ließen sich 5 Gruppen unterteilen, bei denen die akute Hirnstammsymptomatik entweder sofort, nach einer vorübergehenden Adaptation oder erst im Rahmen einer

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Nachblutung eingetreten war bzw. erst die Nachblutung zur Hirnstammsymptomatik geführt hat.

Die morphologischen Untersuchungen ergaben, daß bei 16 Patienten eine massive Hirnstammtamponade, davon in 14 Fällen gleichzeitig ein ausgeprägtes Hirnödem mit Einklemmungszeichen vorlag. Bei einem Patienten bestand nur eine Zisternentamponade, bei einem anderen stand das Hirnödem mit Einklemmung eindeutig im Vordergrund. Bei der Patientin mit apallischem Syndrom fand sich ein ausgeprägter Parenchymschaden des Großhirns.

Es wird auf die weitgehende Analogie des hier beschriebenen Verlaufs zu der von Plum und Posner (1972) dargelegten Symptomatik hingewiesen.

Introduction

There are relatively few statements in the literature about the clinical symptomatology of the severe forms of SAH. Details of the clinical course have been presented in only few descriptions of cases [2, 3]. Plum and Posner [6] are the only one who have discussed the clinical picture and the pathological mechanism of spontaneous SAH associated with coma.

The classification of SAH into grades of severity is important, especially as regards the indication or contraindication for operation. Norlen [5] has recently again declared that coma is a contraindication for operation in a patient in whom an aneurysma is the source of bleeding. He has also stated that the operative mortality of stuporous patients is higher than for those who are clearly conscious. For the operative management of SAH it is important to know if, in addition to the focal symptomatology caused directly by the bleeding, diffuse brain damage has already occurred, especially in the brainstem. Finally, from the subsequent report of observations on our material, one may see how the symptoms of an acute brainstem syndrome occur.

Case Reports

The clinical histories of 18 patients who died after spontaneous SAH, which caused a severe form of the clinical course, have been studied. There were 16 patients with an aneurysm, one with an arteriovenous angioma, and one with massive intracerebral hemorrhage. In the analysis of the cases special attention has been paid to the acute brainstem symptomatology in the forms of a midbrain syndrome (MBS), or bulbar syndrome (BS).

The symptomatology and clinical course of these cases will be reported and a characteristic case will be described in detail from each of the groups into which they have been divided. The cases are assembled in Table 1 according to the symptomatology and clinical course and, in Table 2, according to the pathological findings. It should be mentioned that details of the course of the illness before admission to the clinic were not always complete. Table 1 shows that the material has been divided into six groups, depending on the onset and cause of the SAH. In the *first* group (Cases 1–4) the MBS and BS developed immediately, in Case 4 without any initial symptoms. An irreversible breakdown of brain function (brain death) developed rapidly. Focal signs were obvious at the beginning of the hemorrhage in two cases. Tentorial and foraminal herniation of varying grade was found at postmortem examination in all four cases. The weight of the brain of Case 3 was elevated to 1900 g, with which there were hemorrhages in the midbrain and pons due to pressure. The clinical course of Case 1 was typical for the groups as it went through every stage of development of the MBS, so it will be described in detail.

Case 1. A man, aged 31, suddenly experienced severe left frontal headache and shortly thereafter became giddy, had a stiff neck, and was restless. One hour later there was lessening of consciousness, the left pupil was dilated, and there was extension of the legs and mass movements of the arms. Within another hour he was more deeply unconscious and there was flexion of the arms and extension of the legs. Then there was extension of all extremities with spontaneous extensor synergies, divergence of the eyes, the left pupil was dilated and the right was narrowed and reacted poorly to light. There was greatly increased tonus of the muscles of the extremities and trunk, exaggerated reflexes, bilateral Babinski phenomena, tachycardia and rapid respiration. The BP was 200/110. The CSF was bloody. Four hours later there was gasping respiration and fewer extensor seizures; 30 min later he was flaccid, areflexic, with the eyes divergent and fixed, and there were no pyramidal tract signs. A half hour later there were signs of irreversible breakdown of brain function and, after another hour, spinal reflexes were demonstrable.

The pathological findings included an aneurysm of the middle cerebral artery, softening of the left frontal pole, left frontobasal bleeding which had broken into the ventricle, internal hematocephalus, tamponade of the cisterns, extensive edema of the brain with herniation at the tentorium and foramen magnum.

The *second* group consisted of Cases 5–7 (Table 1) and there was, likewise, the development of a MBS and BS immediately after the onset of the SAH, but in all three cases there was transitory stabilization and partial regression. In Case 5 there was regression from stage I of the BS to the full picture of MBS, and in Case 6 from a complete picture of BS to stage I of BS. Cardiac arrest occurred in Case 7 before the later developing regression. The initial symptoms were found only in Case 5 which died of cardiac arrest. The two remaining patients developed irreversible breakdown of brain function.

Pathological examination revealed foraminal compression signs which were very marked in Cases 5 and 7, the latter having hemorrhagic infarcts of the tonsils. There was rupture into the ventricle in all three cases. Case 5 will be described in detail for the second group.

Case 5. A man aged 31 years had SAH in 1961, at which time carotid angiography was negative. While at work in February 1962 he had sudden onset of headache with rapidly developing loss of consciousness, collapse, deep breathing and vomiting. There was extension of all extremities and opisthotonus within 10 min, followed shortly thereafter by extensor synergies which were intensified by painful stimuli. There were spasms of the chewing muscles synchronous with the extensor synergies. There was divergence of the eyes, both pupils were in mid position with poor reaction to light and the ciliospinal reflex was weak. Tone of the masseter muscles was increased and the masseter reflex was exaggerated. There was marked increase of the tone of the body musculature and exaggeration of the tendon reflexes, which were partially not elicited because of the increased tone. There were bilateral Babinski phenomena and absent abdominal reflexes, rapid rhythmic machine-like breathing (rate 26), slight hyperthermia, pulse 120 and BP 250/170. Respiration of Cheyne-Stokes type set in after 2 h, then became gasping and simultaneous the extensor spasms diminished. The pupils were widely dilated and reacted poorly to light, and the pulse and BP fell. After 30 min newly marked extensor spasms and machine-like breathing occurred and 3 h later there was gasping respiration again, lessening of the extensor spasms, medium wide pupils and a fall in the pulse and BP. Respiration ceased 45 min later and artificial respiration was administered. The BP was 100/50. There was flaccidity of the body, cessation of extensor spasms, widely dilated fixed pupils, tachycardia, and, within 25 min, the pulse and BP dropped and the heart stopped.

Pathological examination disclosed a ruptured, partially thrombosed anterior communicating aneurysm, internal and external hematocephalus with extension into both frontal lobes, right more than left, and rupture into the right ventricle. There was edema of the brain and herniation into the tentorium and foramen magnum, the latter very marked.

In the *third* group (Cases 8–11) general symptoms of SAH occurred first without acute MBS (Table 1). After stabilization of the clinical picture and regression of the general acute symptomatology, there was rebleeding which led to acute BS. This progressed to an irreversible breakdown of cerebral function, which, with cardiac arrest, led to death (Case 11).

Table 1. Summary of clinical course in 18 patients with spontaneous SAH

Group	Case	Age	Sex	Number of bleedings	Initial symptoms		Clinical course	
					general	local	Rebleeding	Restitution
1/ 1	K.S.	31	♂	1	+	–	–	–
2	E.J.	28	♂	3	+	l.f.p.	–	–
3	F.P.	36	♂	2	+	l.f.	–	–
4	H.Sch.	54	♀	1	–	–	–	–
2/ 5	A.S.	31	♂	2	+	–	–	–
					–	–	–	–
6	H.K.	35	♂	1	–	–	–	–
					–	–	–	–
7	G.H.	30	♀	2	–	–	–	–
					–	–	–	–
					–	–	–	–
3/ 8	B.L.	60	♀	1	+	+	–	–
							6 d	–
9	G.L.	32	♀	5	+	l.f.p.	–	–
					–	l.f.p.	16	–
10	A.T.	48	♂	2	++	–	–	–
					–	–	18	–
11	A.H.	54	♀	1	+	l.p.	–	–
					++	–	3 d	–
4/12	P.W.	35	♂	2	+	–	–	–
								26 ←
							28	–
13	M.S.	43	♀	2	+	–	–	–
					–	–	–	24 ←
					+	–	24 d	–
					–	–	–	56 ←
					–	–	40 d	–
5/14	P.K.	44	♂	1	+++	l.f.	–	–
					+++	–	6 d	–
							–	–
15	F.K.	60	♂	1	++	+	–	–
							10 d	–
							–	24 ←
							12 d ↙	–
16	H.S.	61	♂	1	+++	l.f.	–	–
							9 h	–
							–	36
					+++	–	4 d	–
							–	24 ←
						l.f.	9 d	–
17	I.W.	57	♀	2	++	–	–	–
							6 h	–
							–	–
							–	–
6/18	D.J.	52	♀	2	–	l.f.p.	–	–
					++	–	22 d	–
					+++	–	–	–
							–	–

Numbers without other designation = hours, d = days, mo = months, f = frontal, p = parietal, l = left

Clinical course						Brain death	Cardiac arrest
MBS				BS			
I	II	III	IV	I	II		
½	1	1½	2	3	6	6½	–
–	–	½	1	3	8	8½	–
–	–	–	4	8	12	24	–
–	–	–	½	8½	23	23½	–
–	–	–	¼ ←	2	–	–	–
–	–	–	2½	5½	6¼	–	6¾
–	–	–	½	2½ ←	5	–	–
–	–	–	–	6	24	24½	–
–	–	–	1	–	5	–	9
–	–	–	–	– ←	9¼	–	–
–	–	–	–	10	17	17½	–
–	–	–	–	–	–	–	–
–	–	–	½	3	6	6½	–
–	–	–	–	–	–	–	–
–	–	½	1	21	26	27	–
–	–	–	–	–	–	–	–
½	–	3	4	4½	5	5½	–
–	–	–	–	–	–	–	–
–	½	1		14	–	–	14½
–	–	– ←	2	–	–	–	–
14 ←	8 ←	4	–	–	–	–	–
–	–	–	30	31	87½	88	–
–	–	– ←	1	–	–	–	–
10 ←	4 ←	2	–	–	–	–	–
1	2	2½	–	–	–	–	–
32 ←	26 ←	–	–	–	–	–	–
–	–	1	1½	5½	6½	–	7
–	–	–	–	–	–	–	–
–	–	–	1	1½ ←	8	–	–
–	–	–	–	9	27	27½	–
–	–	–	–	–	–	–	–
–	–	–	– ←	½	–	–	–
–	–	1½ ←	1	–	–	–	–
	–	– →	2	16	54	–	54½
–	–	–	–	–	–	–	–
–	2	3	–	–	–	–	–
15½	15 ←	–	–	–	–	–	–
– ←	1	–	–	–	–	–	–
12	–	–	–	–	–	–	–
–	–	1	25	35	36	–	36½
–	–	–	–	–	–	–	–
–	–	–	–	–	6	–	6
6½ ←	–	–	–	–	7½	–	7½
–	–	–	8 ←	15	29	29½	–
–	–	–	–	–	–	–	–
–	–	1	2	–	–	–	–
–	40 ←	4 ←	–	–	–	–	–
–	– →	47	48 →	–	–	–	–
				10 d AS	20 d R	4 mo Ex.	

MBS = midbrain syndrome, BS = bulbar syndrome, AS = apallic syndrome, R = remission, Ex = death

Table 2. Summary of neurological findings in 18 patients with spontaneous SAH

Case	Etiology (aneurysm)	SAH	Brain hemorrh.	Rupture into ventricles
1	A. cer. med. l.	base	l. frontal	l. AH
2	AV-Angiom l. Stggl temp.	base	l. Bsgl. temp. H.	l. lat. V.
3	A. com. ant./A. cer. ant. r.	base	l. frontobas. ant. N. lentif	r. AH.
4	Hyperton. mass hemorrh.	–	Bsgl. r. lat. V	lat. V bilat.
5	R. com. ant.	base	front bilat. r./l.	r. AH
6	R. com. ant. A. cer. ant. l.	Conv. + base	S. perf. ant. l. fornix ant. Ccal.	l. AH
7	A. carot. int./A. cer. med. r.	front + base	ant. N. lent. r. frontobas	
8	R. com. ant.	front + base	frontobas Ccal. cist int. hem.	l. AH
9	A. carot. int./A. cer. med. l.	base	med. bas. temp. lobe, hippocamp	l. TH
10	A. cer. ant.	base	frontal	r. AH
11	A. basil. (walnut size)	base	–	–
12	R. com. ant./A. cer. ant. l.	front + base	ant. mesencephalon-frontobas	l. AH
13	A. cer. med. and nat. r. (mult. An.)	base	frontobas (old SAH)	–
14	R. com. ant./A. cer. ant. r.	Conv. + base	Knee Ccal cist. int.-hem., AH	r. AH
15	A. cer. ant.	base	r. front.	r. AH
16	R. com. ant.	base	l. front.	l. AH
17	A. cer. ant. l.	base	l. frontobas. l. AH	l. AH
18	R. com. ant.	base	–	–

Abbreviations: A = artery; ant. = anterior; Ccal = corpus callosum; cer. = cerebral; cist. int. hem. = cisterna interhemispherica; hem. = hemorrhage; cbl = cerebellum; R = ramus; S. perf. = substantia perforata; SAH = subarachnoid hemorrhage; Bsgl = brainstem ganglia; lat. V = lateral ventricle; TH = temporal horn; AH = anterior horn

In all cases of this group (Table 2) there was striking bulbar foraminal pressure coning and minimal tonsillar pressure signs, although there was hemorrhagic infarction of the tonsils in Cases 8 and 9.

In the *fourth* group (Cases 12 and 13) the complete picture of a MBS took place without

Int. and ext. hematoma	Brain vol.-wt.	Cbl.BS wt.	Tent.-obl. coning		Remarks
3+	3+	3+	3+	3+	–
3+	3+1650 g	2+190 g	3+	1+	–
3+	4+1900 g	3+230 g	4+	3+	Old basal hemorrhages, coning hemorrh. midbrain pons tegue
3+	3+	2+	2+	2+	Old hemorrh cysts, frontal white matter
3+	3+	3+	3+	4+	–
3+	3+1650 g	3+240 g	1+	3+	Diff. granular layer necrosis cbl artex
3+	2+1330 g	3+230 g	3+	4+	Hemorrh. infarct, cbl.-tons. diffuse, cbl. necrosis, cbl. artex
1+	2+1350 g	3+200 g	1+	3+	Tons. infarct diffuse cbl. necrosis
3+	3+1500 g	3+230 g	1+	4+	Hemorrh. infarct. cbl. tons
3+	3+	2+	2+	2+	–
+–	3+	1+	1+	3+	Displacement ventr. pons by aneurysm
3+	4+1800 g	3+230 g	1+	r. 3+ l. 2+	–
–/3+	3+	3+	3+	3+	–
3+	3+1550 g	3+210 g	1+	3+	–
3+	1+	1+	1+	3+	–
3+	1+	1+	3+	3+	–
3+	3+	1+	1+	3+	–
–	–	–	–	–	Bilat. cortical necrosis (Stage II–III) int. hydrocephalus, older coning necrosis thalamus and hippo-campus

marked initial symptoms. In both cases the MBS receded completely within 26 and 24 h. There was rebleeding after 28 h in Case 12 and after 24 days in Case 13. In Case 12 this led to the picture of MBS within 2 h and, 1 h later, to BS in which the patient remained for about 56 h, then quickly went into the state of irreversible breakdown of brain function. The complete picture of MBS developed at first in Case 13, then regressed completely within 24 h. The first rebleeding occurred after 24 days, caused MBS, then regressed again completely. The second rebleeding took place after approximately 40 days. This caused the full picture of BS within $6\frac{1}{2}$ h and the patient died shortly thereafter of cardiac arrest.

Pathological examination of Case 12 revealed striking foraminal and moderate tentorial compression signs as well as ventricular hemorrhage and increase of the weight of the brain to 1800 g. In Case 13 there was striking edema of the brain with marked compression signs, but no rupture into the ventricle.

In the *fifth* group (Cases 14–17) an initial hemorrhage without MBS was recognizable throughout. Hours to days later, after regression of the general acute symptoms, a new hemorrhage occurred which resulted in MBS. After transitory remission in Case 14, the BS became more intense after a second hemorrhage. In Case 15 there was complete remission of the symptomatology of the BS which followed the first rebleeding. The second rebleeding first led by way of MBS to the complete picture of BS in which cardiac arrest occurred. There was complete remission of the brainstem symptomatology after both first and second rebleeding. Only after the third rebleeding did cardiac arrest occur, twice within 90 min, with cessation of respiration and cardiac function due to acute BS. After successful reanimation, MBS set in but BS developed within 24 h to complete breakdown of brain function.

Pathological examination revealed rupture into the ventricle in all four cases with extreme internal hematocephalus and striking signs of basal and foraminal pressure. The weight of the brain was very much increased in Cases 14 and 17.

In the *sixth* group there is only Case 18, which should be included in group 3 because of the occurrence of rebleeding, but is being put into a special group because of the unusual course of the apallic syndrome. The first SAH caused left frontoparietal focal signs which did not disappear completely. Rebleeding 22 days later caused an acute MBS which had regressed within 40 h to the second stage. Presumably as the result of a second rebleeding, the complete picture of MBS developed 8 h later. An apallic syndrome set in during the course of the next 10 days. When this stabilized an operation was performed on the aneurysm of the anterior communicating artery which had been found in the meantime. A beginning remission of the apallic syndrome did not continue and the patient died after 4 months from a newly developed complete picture of the apallic syndrome. Because of the unusual nature of the clinical course this case will be described in detail.

Case 18 was a woman of 53 years who experienced nausea, vomiting, severe headache and an electrifying feeling in the entire body when she made physical effort, 4 weeks before admission. These symptoms subsided after 6 h but she had weakness of the right arm and leg. Later there was recurrence of headache. After lumbar puncture, which revealed bloody CSF, there was relief of her discomfort. She was transferred to the Neurological Clinic of the University of Vienna 3 weeks later and was found to have minimal signs on the right side with slight frontal symptomatology. There was sudden increasing diminution of consciousness 12 h later and within 2 h there was a complete picture of MBS, which, within 2 h moved into the third phase of MBS. An aneurysm of the anterior communicating artery was found on carotid angiography but there was no operative intervention. After another 36 h the picture progressed to stage II and 8 h later the complete picture of acute MBS was present again. There was regression of the MBS in the next 2 days with transition into an apallic syndrome which developed into the complete apallic picture after 10 days. Clinically there was striking increase of masseter tonus and the masseter reflex, atrophy of the upper and lower extremities, especially of the peroneal region. There was beginning remission of the apallic syndrome after 10 days and the aneurysm was operated upon without complication 33 days after the first rebleeding. Subsequently there was further improvement to the point that simple commands were performed with an undifferentiated emotional reaction with manifestations of the Korsakow syndrome. There was no change for 6 weeks, but she then developed high fever due to cystitis and recurrence of coma vigile and the rest of the apallic symptomatology. Death took place due to cardiac decompensation without a sign of MBS, 124 days after the first rebleeding.

The EEG was abnormal 6 weeks after the acute phase. There were bilateral occipital slow theta waves, sporadic delta waves scattered through the 7–8 c/s basic rhythm, bilateral frontotemporal to temporal scattered theta-delta groups, more striking on the left, with a tendency to extension into the frontal region.

The pathological findings included SAH from a clipped aneurysm of the anterior communicating artery, bilateral old frontal cortical necrosis in the area of the anterior cerebral artery, severe internal hydrocephalus with basal siderosis, old compression necrosis in the left hippo-

campus, multiple old necrosis due to vascular compression in the median nucleus of the right thalamus as well as in the base and sides of the third ventricle. There were no lesions in the midbrain or oblongata. There was polyneuropathy of the distal axonal type with ascending wallerian degeneration and marked denervation atrophy of the muscles of the lower extremities.

The information gathered from the case material presented (Table 1) shows that a MBS occurred in all 18 cases. In group 1 the direct development of a BS after a MBS led to death while in group 2 death occurred after temporary stabilization of the MBS which was followed by reintensification of the MBS and BS. In Group 3 there were symptoms of SAH at first without midbrain symptoms and MBS and BS developed only after rebleeding. There was transitory stabilization in group 4. Four hemorrhages could be distinguished in Case 16. In Cases 14 and 15 there was a BS which, in Case 15, experienced a complete remission. There was also complete remission in Case 16, to be sure only from the third stage of MBS. Case 18 belongs in group 3 but is presented separately because the patient had a complete apallic syndrome with associated remission and died after 4 months. There was brain death in 12 cases and cardiac arrest in the remaining patients during the BS. Cardiac arrest was overcome twice in Case 17 and brain death occurred due to BS. Additional clinical data are to be found in Table 1. Table 2 contains the pathological findings and follows the same grouping as Table 1. As can be seen from the table there was rupture of a ventricle in 15 of the 18 cases and marked increase of the weight of the brain in 13 cases, moderate increase in Cases 7 and 8, and to a lesser degree in Cases 15 and 16. In Cases 1–4, where a particularly rapid development of the MBS occurred, there was, without expection, a striking increase in the weight of the brain. The volume of the structures of the posterior fossa (cerebellum and brainstem) was clearly increased in ten cases, and slightly in three cases. It was not possible to correlate the clinical course with the increased volume of the cerebellum and brainstem. A tentorial pressure cone was found in all cases with the exception of Case 18; it was especially marked in Case 3. There was pressure coning of the oblongata in all cases from clear to severe; it was moderate in Cases 4 and 10, and slight in Case 2. Internal and external hematocephalus with cisternal tamponade was found to a striking degree in 14 cases. It was present without exception in the first two groups where MBS and BS developed rapidly. The BS developed in a particularly short time in Case 8 due to rebleeding where there was only slight internal hematocephalus but striking tamponade. Case 11, with a basilar aneurysm, had slight internal hematocephalus although cisternal tamponade was of high degree. There was no rupture into the ventricle in Case 13, nor marked cisternal tamponade, but there was definite edema of the brain with signs of compression. No judgement could be made about acute compression in Case 18 because so much time had elapsed before death.

Discussion

As already mentioned in the introduction there are only cursory remarks in the literature about the symptomatology of the severe form of SAH. Only Plum and Posner [6] have described the development of the acute brainstem symptomato-

logy of patients with coma after SAH. They cite several factors as the cause of the comatose course after SAH and state that these include the mechanical effect of the formation of the hematoma on the brainstem, an increase of intracranial pressure leading to transtentorial herniation with compression of the brainstem, and direct pressure on the intracranial structures with resulting spasm of the arteries and consequent ischemia of the parenchyma of the brain. Plum and Posner also maintain that the blood found in the subarachnoid spaces causes a disturbance of the CSF circulation and a chemical meningitis [4] which directly influences the metabolism of the brain. The symptomatology of the severe clinical course of SAH described by Plum and Posner shows that the acute form of the MBS and BS develop after nonspecific general symptoms combined with increasing local difficulties that lead to the complete picture of MBS and BS (see Case 36, p. 213) and lateralization of a MBS can also be observed. Foltz and Ward [1] have reported severe residuals after SAH with prolonged coma which corresponds to the apallic syndrome.

In analyzing the cases with fatal SAH presented here it was found that in all instances the symptomatology of MBS and BS had developed and that the material could be divided into several groups according to the occurrence in time of the onset, development and course of MBS and BS. It was not possible to establish a relationship between the number of bleedings and the course of development of the damage to the brainstem. It is worth noting that complete regression of the acute brainstem symptomatology took place in all cases of group 4, as well as Cases 16 and 17, although there were massive midbrain signs and an already developed BS.

The pathological examinations revealed that there was a marked pressure cone of the brainstem in 16 cases and in 14 cases there was massive edema of the cerebrum, and the structures in the posterior fossa had suffered tentorial and foraminal pressure coning. Therefore there was increased volume with massive displacement as well as pressure coning of and direct pressure on the brainstem in all cases. Case 11 with an aneurysm of the basilar artery and only cisternal tamponade, and Case 13 with predominantly brain edema and pressure coning, were exceptions.

The development of MBS and BS in the most severe forms of SAH must be attributed to brainstem damage resulting from cisternal tamponade as well as from local pressure in the midbrain and bulbar region due to pressure coning. As judged by the clinical course, local pressure coning appears more important for the development of MBS and BS than direct brainstem tamponade.

For the existence of the apallic syndrome the direct pressure of the hematoma which Plum and Posner have mentioned, with resulting vascular spasm, has played a role. The same mechanism may also influence the development of edema of the brain. We find little to explain the disturbances of chemistry and of the CSF circulation.

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