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VENTRICULAR HEMORRHAGE: A SYMPTOM-GROUP*

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Hemorrhage in the brain may be extraventricular or intraventricular. In the latter case the blood may originate in the ventricle itself and produce a primary ventricular hemorrhage. In the first, the original seat of the hemorrhage is the tissue surrounding the ventricle and the ventricle itself is only secondarily involved. Secondary effusion of blood into cerebral ventricles is not an infrequent phenomenon, while a primary hemorrhage within the ventricular cavity is rather a rare occurrence, judging from the meager literature on the subject. Not many records are to be found on primary intraventricular hemorrhage. Nevertheless, sufficient data have been accumulated to deserve a special description.

The mechanism of formation, the pathology, the course, the symptomatology of ventricular hemorrhages have been somewhat differently considered by various authors who have had the opportunity to observe such findings. All writers, however, concur in the belief that this form of cerebral hemorrhage is quite infrequent. In view of this infrequency it may be of interest to place on record twelve personal cases of extraventricular and intraventricular hemorrhage studied from anatomical and clinical standpoints. The study of this series suggests a remarkable uniformity in the pathological aspect and in the manifestations during the patients' lives, so that diagnostic inferences appear to be admissible.

Out of the twelve cases seven presented secondary effusion into the ventricles from an original extraventricular area situated close in the vicinity of the cavity. The remaining five cases are examples of primary intraventricular hemorrhage.

I propose to consider first the latter group and the mechanism of formation of the hemorrhage before several other interesting features.

PATHOLOGY

In Cases 2 and 3 sections of the brains were made on the third day of hardening; the blood was washed out thoroughly from the lateral

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ventricles and the source of bleeding could be distinctly seen in the choroid plexuses. The choroid plexuses have been considered by many observers as the principal source of intraventricular hemorrhages. From the time of Morgagni, who uniformly considered those plexuses as being the unique source of the bleeding, up to the present time, the majority of observers believe that even in cases in which the seat of the bleeding could not be discovered, the blood vessels of the choroid plexuses are the origin of the ventricular hemorrhage. Degeneration, chronic inflammation, fatty changes, calcareous deposits, dilatation—all these conditions have been observed in the vessels of the plexuses. Thrombosis, aneurisms of the blood-vessels, angiomatous tumors and cysticerci attached to the plexuses have been observed in a few cases (Broca). Serous cysts originating in the walls of the blood vessels have been found in aged individuals also in cases of atrophy of the brain (Wilks). In the Cases 2 and 3 in which rupture of vessels of the plexus was present, the vessel wall showed fatty degeneration and rounded yellowish masses were seen in the posterior portions of the plexus. These masses were very likely remains of former blood effusions. In all of them pronounced changes were evident in the intima, such as disappearance of the endothelium, degeneration and thickening. The elastica interna usually followed the changes of the intima. The adventitia was found thickened in some vessels, but the media was frequently seen altered, viz., granular and calcareous masses were present. Some vessels were greatly distended with blood, but not to the extent of formation of aneurysm. On none of the sections could a true or false aneurism be detected.

In Cases 1 and 4 after the blood was washed out erosion of the ventricular walls could be seen. Here the blood vessels were found enlarged and distended with blood. Some of them showed calcareous deposits; in others complete occlusion with thrombi was evident. Both individuals were of somewhat advanced age, one 63 years old, the other 67. The ulcerations mentioned were seen on the wall of the corpus striatum in the lateral ventricle.

Case 5 is most interesting from the standpoint of pathogenesis. Nothing abnormal could be found macroscopically, but under the microscope very small aneurysmal enlargements could be seen.

As is well known, miliary aneurysms were considered by Charcot and Bouchard as the chief cause of cerebral hemorrhage. Before them distention of blood vessels and aneurysms were mentioned as causes of cerebral hemorrhage. But since these two observers published their monograph in 1868, the subject of miliary aneurysms was indiscriminately emphasized as being the sole etiologic factor. In their collection of eighty-four cases of cerebral hemorrhage, in all of them multiple

miliary aneurysms were found. In their study they confined themselves to gross pathological changes. They placed the brains in water and small floating masses of tissue with their vascular attachments were picked out and examined under magnifying glasses.

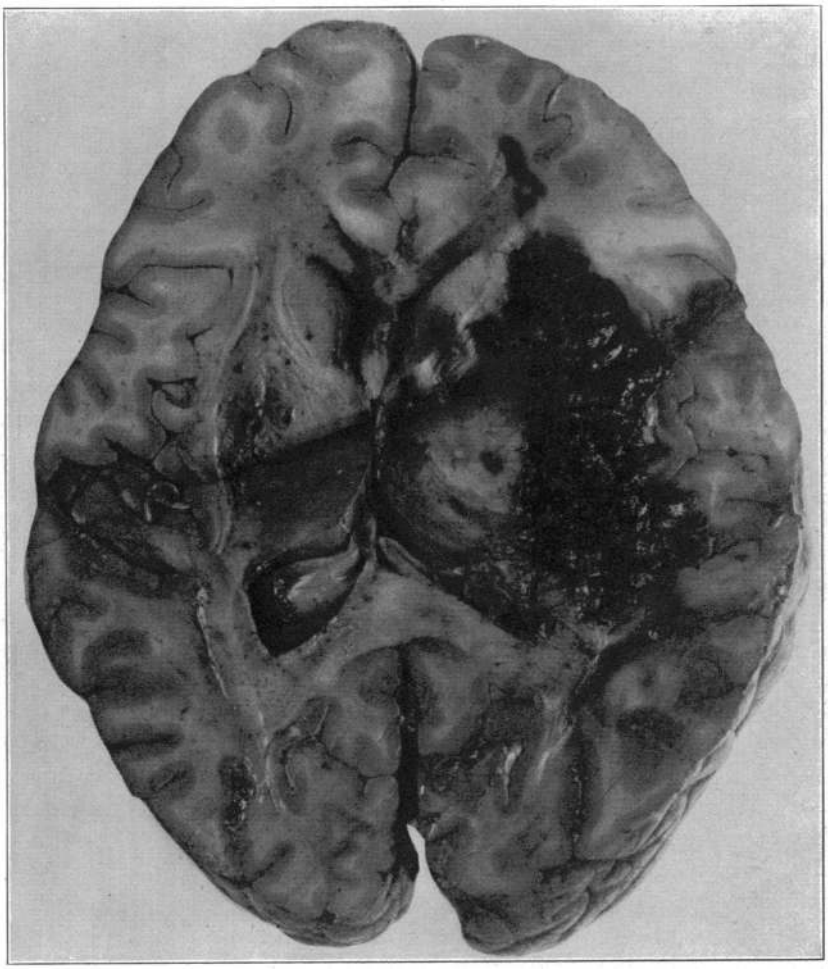

Fig. 1.—Primary intraventricular hemorrhage (in lateral cornua on right). Note deviation of the opposite side. Patient operated on.

The subsequent writers on the subject studied miliary aneurysms from the histological standpoint. The intima, the adventitia and the muscularis have all or individually been considered as the origin of aneurysmal dilatation of the blood vessel. Lowenfeld[1] from a study

1. Lowenfeld: Studien über Aetiologie u. Pathogenese der spontanen Hirnblutungen, Wiesbaden.

of seventeen brains, all with miliary aneurysms, found in every one the three vessel coats perfectly intact. The weight of opinion of the majority who believe in the existence of miliary aneurysms, however, is that the changes begin first in the media. The latter degenerates, becomes atrophic, the entire vessel wall becomes then nonresistant, and aneurysmal dilatation follows. This condition occurs especially in the very small cerebral arteries. In a large number of cases no miliary aneurysms were found and the rupture of the blood vessels was due to a diffuse degeneration of their walls. Kaufman,[2] for example, finds atheromatous changes in the large cerebral blood vessels and hyaline degeneration in the small ones.

Finally, the existence itself of miliary aneurysms is disputed by some writers.

In my case, after a thorough washing of the hemorrhagic lateral ventricle the brain tissue on the inner wall was seen torn and a number of exposed small vessels could be noticed. Portions of the lacerated tissue were placed in water so as to enable me to observe the blood vessels. Some of them were irregular and projections were seen on their walls. The remainder of the tissue was hardened in Müller's fluid and later examined microscopically. Imbedding was done in celloidin. Staining was done with osmic acid, Weigert's hematoxylin and ammonia carmin. Sections were made longitudinally and perpendicularly to the vessel. Marked changes of the intima and the elastica were found on many sections. Rupture of the intima and blood placed between the latter and elastica were seen on some sections. Swelling of the intima, disappearance of the endothelium, and hyaline appearance of the elastica could also be seen. Lesions of the media were always seen in conjunction with lesions of the intima; degeneration or calcareous deposits, thickening, were observed in the latter. The adventitia was seen split in one or two layers with blood between them. In the aneurysmal dilatations no clear distinction could be made of the individual membranes of the vessel wall. The latter consisted only of a few thin bands infiltrated with leukocytes. The aneurysms were filled with blood and in some sections blood was seen immediately around the vessel. In the latter cases the opening of the ruptured vessel could be traced. A gradual transition from the vessel wall to the aneurysm was noted. In all cases the changes of the intima become more and more marked as the aneurysmal sac is approached. In a number of sections the so-called dissecting variety of aneurysms could be seen, viz., a sacular dilatation of the vessel was present and blood had penetrated between the intima and the media or media and adventitia.

2. Kaufman: Specielle Pathologic Anatomie, 1907.

The histological findings in all the five cases permit the conclusion that as far as the mechanism of formation of the hemorrhage is concerned, the latter may be produced by simple rupture of the vessel wall or of a miliary aneurysm, but in both cases profound changes of some or all layers of the wall are always present and especially more pronounced in the intima.

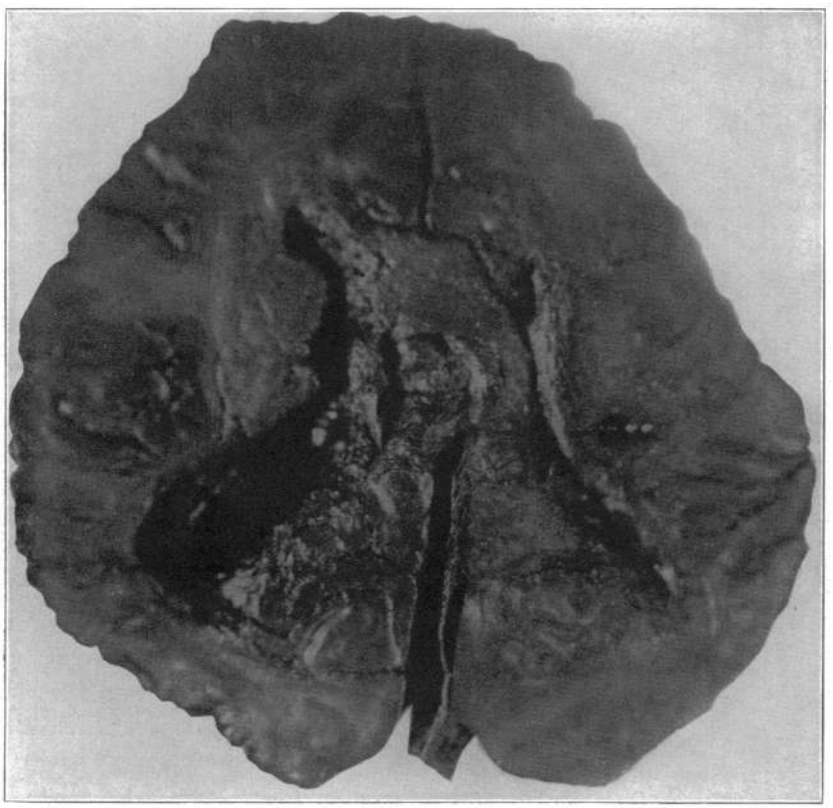

Fig. 2.—Primary intraventricular hemorrhage (on right, three cornua involved). Note deviation of the opposite side.

The seat of the primary intraventricular hemorrhage, as seen from my five cases, is distributed as follows: Two cases in both lateral ventricles; in one of them blood was found in the three cornua of one ventricle and only in the anterior and posterior cornua of the other ventricle; in the second case the hemorrhage was seen in all the cornua of one ventricle and only in the posterior cornua of the other ventricle. Three cases presented hemorrhage in one lateral ventricle; in four out

of five cases the right lateral ventricle seems to be the predominant seat of hemorrhage. In only one case was the hemorrhage seen exclusively in the left lateral ventricle.

The seven cases of secondary ventricular hemorrhage present this peculiarity, that here hemorrhage in both lateral ventricles was seen in a larger number (five) than in cases of primary ventricular hemorrhage.

The blood in the ventricles was found coagulated in four cases and fluid in one case. In the latter, death ensued instantaneously. In the former in six, seven, twelve and twenty-four days, respectively. The patient in the latter case was operated on (see below) and at necropsy a firm clot was found. It is interesting to note that in the unilateral cases the ventricle which was free from blood was filled with serous fluid. The walls of the ventricles presented small erosions in three cases. In two cases with diseased blood vessels of the choroid plexuses, the ventricular walls were intact. It is to be presumed that the lacerated face of the ventricles was the result of the diseased vessels and of miliary aneurysms which eventually ruptured. Under the microscope destruction of tissue was seen, but there was no indication of a degenerative state which usually follows softening. The remainder of the brain with its meninges was intact.

In all the five cases the brain tissue in the immediate vicinity of the hemorrhage suffered destruction. In three cases the hemorrhage, besides destroying the surrounding brain tissue and pushing outward the remaining cortical substance, exercised also considerable pressure on the opposite hemisphere and disfigured it. This latter fact, observed at first in one case, suggested a certain surgical procedure for relief of increased intracranial pressure which will be discussed later. In the two other cases the hemorrhage was bilateral so that no marked displacement of brain substance to one or to the other side could be observed.

The pathology of the second series of my cases, seven in number, presents no special features deserving special mention. The original hemorrhage occurred in all in the internal capsule and inundated secondarily the lateral ventricles. Bilateral hemorrhage was present in five out of seven cases. In the two unilateral cases the right lateral ventricle alone was involved. In three cases were found degenerative changes of blood-vessels, especially of the intima, such as was described in the cases of primary ventricular hemorrhage. In four cases multiple miliary aneurysms were seen in the seat of original bleeding. The technic as to preparing the tissue hardening and staining was exactly the same as in the first series.

DIFFERENTIAL CLINICAL FEATURES

The most interesting manifestations in the primary ventricular hemorrhages of my five cases were: the sudden onset; the most profound coma from the very beginning; convulsions more marked on the side opposite to the lesion than on the same side in the unilateral

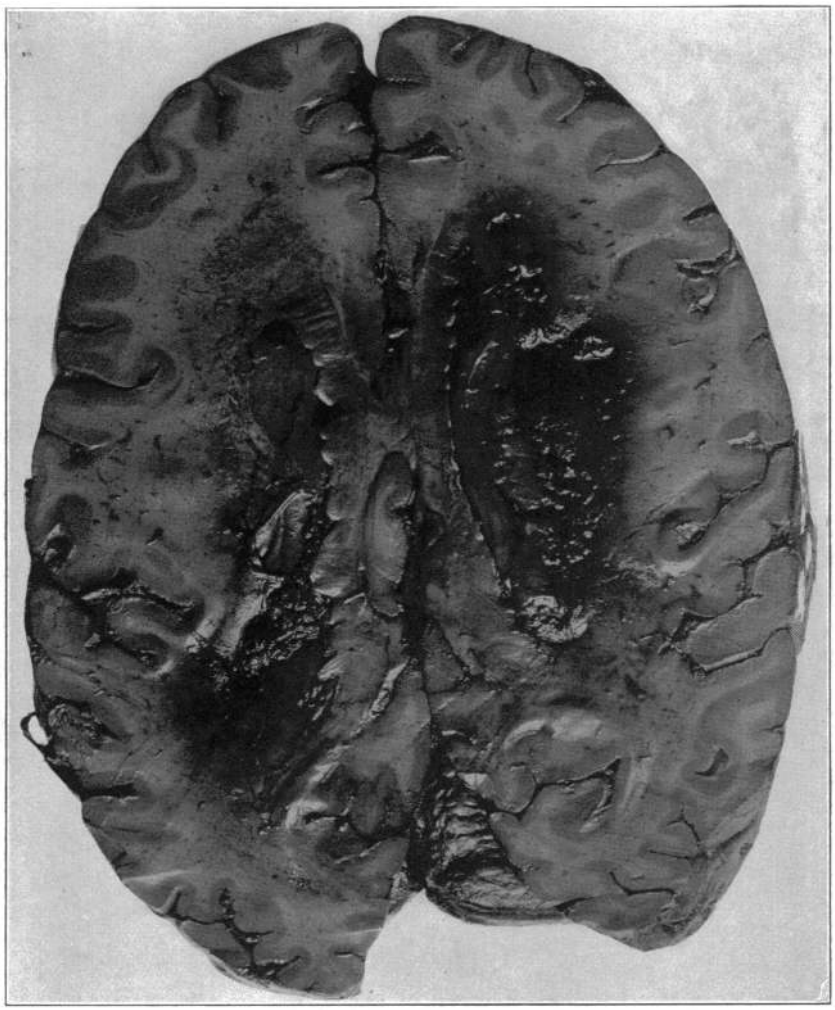

Fig. 3.—Primary intraventricular hemorrhage (both lateral ventricles).

cases, and on the side opposite to the seat of the largest hemorrhage in the bilateral cases; finally, absence of marked paralysis—the latter was very slight. These four symptoms were uniformly present in all the five cases at the time when the attack was ushered in.

During the comatose state two patients with unilateral hemorrhage had several convulsive seizures, always contralateral; the other three patients had but one initial attack. The most striking phenomenon is the absence of true paralysis. At first similarly to ordinary cerebral hemorrhage, the sudden loss of power was evident, but subsequently during the patient's short life the usual rigidity and contracture did not appear. The absence of rigidity is quite interesting, as since Durand-Fardel, early contracture of the limbs had been considered pathognomonic of primary intraventricular hemorrhage.

Loss of power is very slight. The latter becomes especially evident where the patient attempts in his comatose state to move his limbs or when he defends himself against external stimulations. As to the reflexes, the knee-jerk on the paralyzed side was not especially increased, although it was somewhat greater than on the normal side. Ankle-clonus was absent, the toe phenomenon was also absent: stimulation of the sole provoked no movement of the toes at all, neither was there any response of the toes with the test for the paradoxical reflex.

In the cases of secondary ventricular hemorrhage the already existing paralysis and contracture became markedly accentuated at the time the ventricles were invaded. The abnormal reflexes which are usually present in hemiplegias were manifest here. All these patients were comatose at the time of the ventricular attack and never regained consciousness during their remaining short life. Among other distinguishing although not constant features of primary ventricular hemorrhage may be mentioned the character of the premonitory symptoms and the duration of the comatose state.

In three of my five patients the attack was ushered in without the least preceding objective or subjective disorder. One patient complained for a few hours of a slight headache, and another patient of a slight vertigo during two preceding days. The attacks consequently bear no relation to the existence or nonexistence of premonitory symptoms. Two of the patients were aged individuals, above 60, and presented evidences of arteriosclerosis; three were of middle age without apparent arterial changes.

The character and duration of the coma are of interest. The latter appeared at the commencement of the seizure, remained complete throughout the short life. In the secondary ventricular hemorrhage the coma was also present with the irruption of the blood into the ventricle. Therefore the mere existence of unconsciousness is of little value in differential diagnosis. It is the sudden onset of profound coma without preceding hemiplegia that will determine the diagnosis of a primary hemorrhage in the ventricle.

In the series of secondary ventricular hemorrhage, death followed but a few hours after the inundation of the ventricle in every case. In the cases of the primary variety, with one exception of instantaneous death, life persisted from six to twenty-four days. The latter patient was operated on, which probably accounts for the longest duration. The subject will be discussed later. The other three patients lived

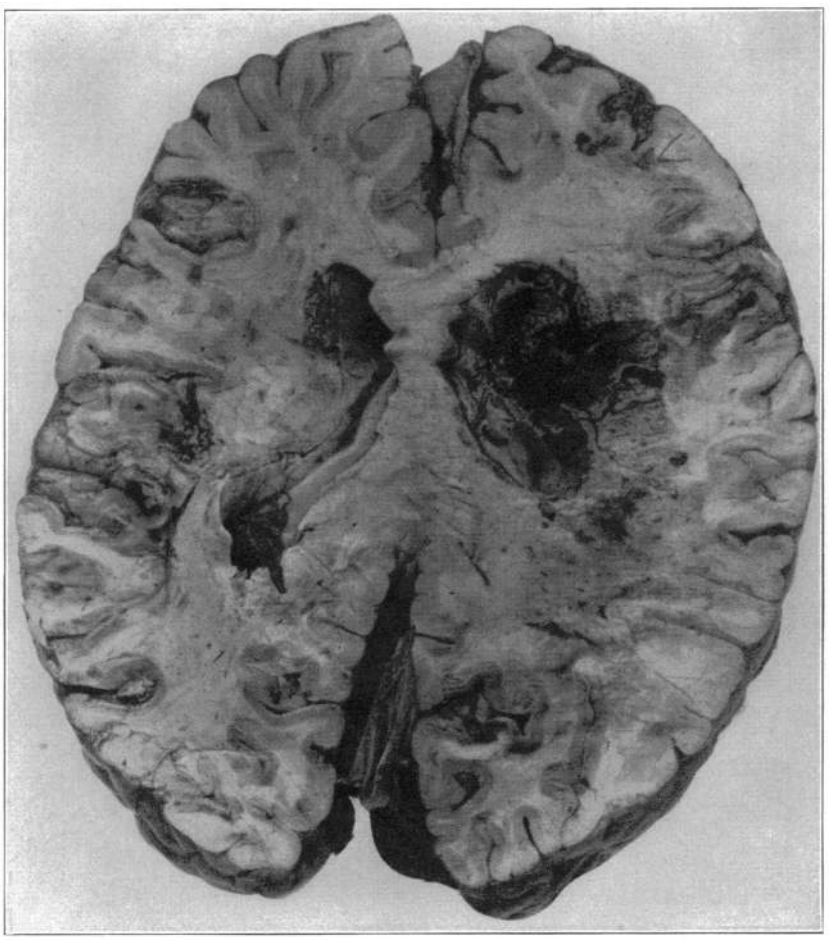

Fig. 4.—Primary intraventricular hemorrhage (on right).

though unconscious six, seven and twelve days, respectively. The seat and the size of the hemorrhage have no direct relationship to the duration of life. Thus, in Case 3 the hemorrhage was more extensive than in Case 4. Nevertheless, the former lived twelve days and the latter but six days. On the whole it seems that the fatal issue is less rapid in the primary than in the secondary variety of ventricular hemorrhage.

A rapid glance at the brains in the five cases of primary ventricular hemorrhage shows displacement of the brain tissue to the side opposite the blood in three cases, and consequently the possibility of the comatose state as due to sudden undue pressure on the normal side of the brain appeared to be highly plausible. Such a finding in one case suggested the idea of surgical intervention in other cases with the object of relieving the intracranial pressure on the sound side. Accordingly an attempt was made in one case for a decompressive operation. Although the patient (a woman of 45) did not recover eventually, nevertheless the duration of her life was prolonged to twenty-four days. Immediately after the operation there was a decided improvement in her respiration, in response to external stimulation, in the cardiac action. For several days she could open her eyes voluntarily and when called by her name; she could be fed more readily than before, as she would respond to requests to open her mouth, and she could swallow. On the twentieth day the coma returned and on the twenty-fourth day she expired. The improvement obtained was undoubtedly due to the relief of the intracranial pressure. Unfortunately the operation was consented to only on the fifth day after the apoplectic seizure, viz., after five days of a comatose state. Efforts were made to operate in the other four cases, but permission could not be obtained.

SUMMARY

The present study suggests the following interesting features in primary intraventricular hemorrhage:

1. From a diagnostic standpoint sudden onset without premonitory symptoms, profound coma at the outset and continuing for several days without improvement, absence of genuine paralysis, absence of rigidity and contracture during the days following the seizure, absence of the toe phenomenon—all of these manifestations speak in favor of primary ventricular hemorrhage.

It should be borne in mind that in all the five cases the hemorrhage occurred only in the lateral ventricles. The above mentioned phenomena were observed in but five cases. The number is too small to draw general conclusions. Nevertheless they appeared sufficiently uniform to permit the possibility of their pathognomonic nature, at least in cases when hemorrhage occurs in the lateral ventricles alone.

2. The next important feature of the subject is the surgical intervention on the sound side based on the existence of a displacement of the brain to that side. It is possible that the profound comatose state is due precisely to the sudden compression of the normal brain tissue. The amount of improvement obtained in one case was a sufficient encouragement in that direction. If a decompressive operation on the

sound side is to be undertaken, it must be done promptly after the onset. In my case the operation was performed on the fifth day and in spite of this delay some amelioration of the condition was decidedly

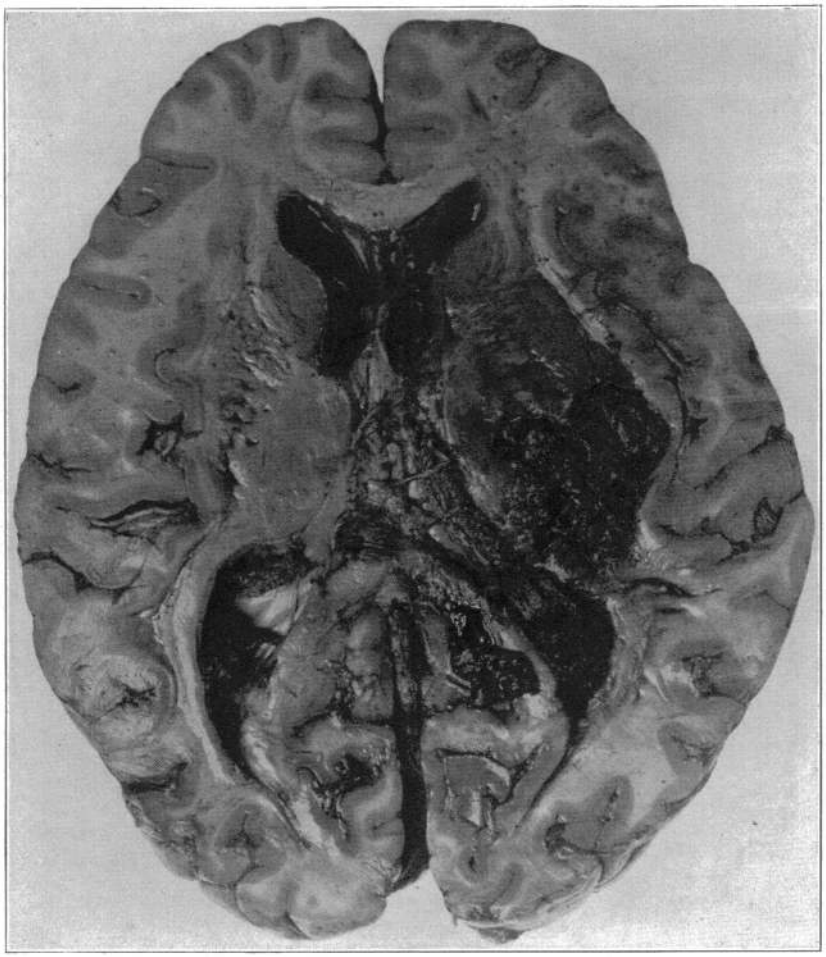

Fig. 5.—Primary intraventricular hemorrhage (both lateral ventricles).

manifest. It is to be presumed that the earlier relief from intracranial pressure is obtained, the more prolonged the favorable results that will be observed.

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