SPONTANEOUS RUPTURE OF THE CEREBRAL VENTRICLES

ARNE TORKILDSEN, M.D.

Neurological and Neurosurgical University Clinic, Oslo, Norway

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In cases of stenosis of the Sylvian aqueduct or occlusion of the 3rd ventricle, the cerebrospinal fluid that is produced within the cavities of the lateral ventricles is prevented from arriving at the place of resorption, that is, the subarachnoid space.

While normally resorption of the fluid takes place at the same rate of speed as production, in cases of obstruction the subarachnoid space is not filled with cerebrospinal fluid in proportion to its production. A condition arises which is characterized by a pressure that is higher above the place of obstruction than below. With occlusion of the foramina of Monro, a relative hypertension exists within the lateral ventricles, and with occlusion of the Sylvian aqueduct, relative hypertension exists within the lateral ventricles and the 3rd ventricle as compared with the pressure in the subarachnoid space.

In cases of relative intraventricular hypertension the walls of the ventricular system are under abnormal tension, and if the difference between the pressure inside and outside the ventricular system is great enough, a rupture of the ventricular wall may take place.

The result of such a rupture depends entirely upon the details concerning its location and its relationship to the membranes covering the central nervous system.

Occasionally hydrocephalic patients give a history of sudden subsidence of the signs of increased intracranial tension which conceivably may be due to ventricular rupture with formation of a short circuit giving the cerebrospinal fluid direct access to the subarachnoid space. This explanation is likely in the cases published by de Lange, van Stockum, and Sweet.

Indeed, it is surprising that ruptures of this kind do not occur more frequently. The translucent film constituting the anterior wall of the 3rd ventricle (lamina terminalis) would seem to make it a common location for such spontaneous rupture. However, only 1 instance of rupture in this location (Sweet) was found in the literature.

Until recently ruptures of the cerebral ventricles have been accidental findings at autopsy. However, observers aware of this kind of lesion must have encountered it not infrequently. Penfield, for instance, mentions that he has found 5 such cases. The literature on this subject is scarce. Judging by the cases that have been published the rupture occasionally takes place bilaterally in the posterior wall of the lateral ventricle (Penfield, Childe and McNaughton). The temporal lobe is another known location (Cruveilhier). That the posterior wall of the 3rd ventricle may be the site of a spontaneous rupture is evident from 2 of my cases.
The time has now come for the clinical recognition of this condition. Pneumographic air studies should move the place for such diagnosis from the postmortem room to the x-ray department. In order to widen our experience concerning these lesions it may be justifiable to publish case reports of some patients who have come under my care.


_Anamnesis._ For 2 years she had suffered from periodic headaches, especially in the back of the head. On 2 such occasions she had lost consciousness, and 4 or 5 months before admission a spell occurred during which she was unable to move limbs or body for about 5 min., although she was mentally clear. There was gradual loss of vision, starting on the left, and exophthalmos, loss of hearing and impairment of memory developed.

_Examination._ Vision of left eye was reduced to light perception; on the right, visual acuity was 6/12. There was bilateral papilledema with choked discs of about 3 D., and also left optic atrophy. There was bilateral exophthalmos, more marked on right. The corneal reflex was diminished and there was decreased sensation on right side of face. Coordination tests showed slight general disturbance, especially on left side. There was also slight hypotonia on the left. Deep and superficial reflexes showed no certain changes. Cerebrospinal fluid, urine, blood, and thoracic and abdominal organs were normal.

Roentgenograms of skull showed the bony walls to be thin, and there were rather marked convolutional impressions. The sella showed considerable destruction and faint contours. The optic foramina seemed to be enlarged but were not clearly visible because of generalized atrophy of the cranium.
SPONTANEOUS RUPTURE OF CEREBRAL VENTRICLES

Ventriculography, Dec. 11, 1945, disclosed widely dilated symmetrical lateral ventricles. No air could be seen in the 3rd ventricle, the Sylvian aqueduct or in the 4th ventricle. In the lateral view a large bubble of air was seen superimposed on the posterior and lower portion of the lateral ventricles. In the position with the brow down, air appeared in the midline at the place where the shadow due to air in the 3rd ventricle usually is found (Figs. 1 and 2). With the brow up and the x-rays in vertical direction, no such shadow could be detected.

At the time of the examination, a diagnosis of tumor in the 3rd ventricle was made because of the pneumographic findings. The bubble of air just described was diagnosed as a subtentorial cyst due to spontaneous rupture of the cerebral ventricles.

Ventriculocisternostomy was carried out immediately after the air study. Death occurred 2 days later.

Autopsy 335/45. There was enlargement of both lateral ventricles as had been diagnosed previously by ventriculography. It was seen to be due to an obstruction of both foramina of Monro, caused by ependymal changes (ependymitis granularis). The ependymitis extended all over the ventricular system, being especially pronounced at the foramina of Monro and the Sylvian aqueduct. At the region of the splenium corporis callosi there were signs of old hemorrhages antedating those due to the punctures in connection with the ventriculography and ventriculocisternostomy.

The cyst seen in the ventriculograms occupied the space above and behind the quadrigeminal plate. Apart from this structure it was surrounded by the cerebellar hemispheres, the lobus cerebelli anterior and, in the anterior direction, by the splenium of the corpus callosum, which had been pressed slightly upward. The hippocampal gyri also formed part of its walls. The roof of the cyst was formed by the tentorium and a distention of a membrane from the fornices to the posterior portion of the corpus callosum. The splenium corporis callosi as well as the pineal body had been dislocated somewhat forward and upward by the pressure of the cyst. A thin fibrous membrane of hemorrhagic appearance lined the cyst. In the posterior portion of the right lateral ventricle, near the midline, a funnel-shaped indentation ran toward the cyst. The communication gradually became narrower and finally it became a mere slit, showing signs of hemorrhagic inhibition.

The Sylvian aqueduct was narrow but not completely occluded. The 4th ventricle showed no signs of enlargement. Multiple subependymal hemorrhages were found in the walls of the lateral ventricles.

Comment. This case offers several points of considerable interest. The ventriculograms showed a cyst of the kind that is typical in cases where a spontaneous rupture of the cerebral ventricles has taken place, and the autopsy findings leave no doubt concerning this diagnosis. It is interesting to note that the patient had suffered from headaches for 2 years, the history thus having been of comparatively short duration. The anamnesis states that 4 or 5 months previous to admission she experienced an episode during which she was unable to move the limbs and the body. This may possibly have been due to the actual rupture. It is conceivable that the formation of the cyst temporarily exercised a compression of the brain stem sufficient to be responsible for temporary functional disconnection of the supratentorial from the infratentorial portion of the central nervous system. The autopsy record describes yellow-brownish discoloration due to old hemorrhages at the region of the splenium corporis callosi, and also states that the membrane lining the cyst showed signs of hemorrhages. It especially pointed out that these hemorrhagic signs were of older date than those due to the ventriculography and ventriculocisternostomy. The faint traces of abnormal
pigmentation could fit in well with a hemorrhage that had taken place some months before.

The formation of the cyst makes it reasonable to suppose that the pathological ependymal alterations originated in the region of the Sylvian aqueduct and spread secondarily to the foramina of Monro. In a case of this kind it would have been quite ineffective to perform ventriculostomy by cutting a window in the wall of the 3rd ventricle in order to create a communication between the 3rd ventricle and the adjacent cisterns. At the present time there is no operation other than ventriculocisternostomy (a communication between the lateral ventricle and the cisterna magna) that could possibly benefit the patient.


*Anamnesis.* For about 10 years she had noticed a peculiar sensation as if something were running in her head. It was not painful, and occurred especially when she was tired. For the last 7 years she had suffered from frequent headaches which became worse during the last year previous to admission.
Examination. Cerebration was somewhat slow. There were bilateral anosmia, bilateral papilledema, slight central facial palsy on right, and deviation of tongue to the right. In the Romberg test she showed a tendency to fall backwards and to the right. Otherwise findings were not abnormal. Roentgenograms of skull showed marked atrophy and separation of the sutures.

Encephalography was attempted 2 times, but no air was seen in the ventricular system. Ventriculography, Mar. 24, 1942, showed the lateral ventricles and the 3rd ventricle to be widely dilated (Fig. 3). No air could be seen in the Sylvian aqueduct or in the 4th ventricle. A large bubble of air was seen in the midline behind the 3rd ventricle, partly below and partly superimposed on the shadows caused by air in the lateral ventricles. In the pictures taken with the brow down and the x-rays in a vertical direction, the cyst was seen superimposed on the shadow due to the 3rd ventricle.

Ventriculocisternostomy was carried out immediately after ventriculography, the diagnosis of non-neoplastic stenosis of the Sylvian aqueduct being made. At operation the stenosis was verified. Ringer's solution was injected into the lateral ventricles under slight pressure during the operation but no fluid was seen to run from the 3rd to the 4th ventricle.

During the postoperative period the signs of intracranial hypertension remained, the patient being troubled by headaches, vomiting and drowsiness. A subaponeurotical collection of fluid was present under the scalp along the rubber tube, giving cause for frequent punctures and removal of fluid, which was of yellow colour, with increased content of protein. The patient gradually became worse and died on the 47th day after ventriculocisternostomy.

Autopsy 126/42. There was a great dilatation of the first 3 ventricles. The posterior wall of the 3rd ventricle had undergone a perforation creating an opening into the interpeduncular fossa, from which a cystic collection of fluid extended below the tentorium between the cerebellar hemispheres.

The Sylvian aqueduct showed a complete occlusion. There was no neoplastic tissue.

Comment. It was found that the rubber tube had been placed in a wrong position with its cisternal orifice close to the soft tissues. This may have been the cause of the unsatisfactory effect of the operation.

While several instances are known in which rupture of the walls of the lateral ventricle had taken place,1 rupture of the filmy wall of the 3rd ventricle seems to be rare, as far as one can judge by the very limited literature on this subject. I could find only one previous example of such a case, the lamina terminalis having been the site of the pathological process (Sweet).2


Anamnesis. Three years previous to admission he began to suffer from headaches and vomiting. He spent a few weeks in bed, after which he felt well until about 4 months before admission, when he again began to suffer from spells of vomiting and headaches, usually lasting a few hours each time. About 4 weeks before admission, the headaches increased and the eyesight became blurred.

Examination. Visual acuity was 3/18 on either side. There was bilateral papilledema, with choked discs of about 5 D. Ocular movements were slightly limited on upward gaze. The right pupil was smaller than the left. Hearing was reduced on the right. There were some hypotonia and paresis of left arm and leg. Romberg test revealed a tendency to fall backward.

Ventriculography, April 19, 1939, showed wide enlargement of both lateral ventricles. No air was visible in the 3rd ventricle. In the lateral pictures (Fig. 4) a large bubble of air was seen superimposed on the posterior portion of the lateral ventricles behind the 3rd ventricle. In the position with the brow down (Fig. 5), this bubble of air appeared in the midline, below and between the lateral ventricles at the place where the shadow caused by air in the 3rd ventricle usually is seen.
Ventriculocisternostomy was performed immediately after ventriculography. The postoperative course was uneventful.

The effect of the operation was satisfactory. Already 6 weeks after operation there were no signs of papilledema. The optic discs became sharply outlined and showed definite signs of atrophy. Visual acuity was reduced to counting fingers at a distance of 2 m. on right and 3 m. on left.

The patient lived at home and was in good health apart from loss of vision. He died 3 years after ventriculocisternostomy, having shown signs of progression of the pathological process the last few weeks before his death.

Comment. In this case the diagnosis of the pathological process was not verified by autopsy. The ventriculographic findings indicate occlusion of the foramina of Monro and do not disclose a neoplastic process. The neurological findings do not necessarily lead to the diagnosis of a tumor in the 3rd ventricle, and might possibly be associated with non-neoplastic obstruction of the foramina of Monro.

After the ventriculocisternostomy all signs of intracranial hypertension disappeared and the clinical course of the case remained more or less stationary until a short time before death, when the patient became increasingly drowsy and stuporous without showing any of the symptoms which supposedly should have been present had intracranial hypertension again developed. It is likely, therefore, that death was due to further growth of a neoplasm with destruction of nervous centers of vital importance.

The cyst seen in the ventriculogram can hardly be mistaken for any other condition. Its appearance is so characteristic and so easily recognised,
once one is familiar with its appearance, that there can not be any doubt about its diagnosis.


Anamnesis. The patient was a full-term child of normal birth. Shortly after birth he started to squint, and the head became abnormally large. He would cry more than is natural. Gradually marked signs of hydrocephalus developed, the head assuming the shape of a pear, with large cranial contours and a small face. There was separation of the cranial sutures and the fontanelles were rather tense. At the age of 6 months he started to have attacks of vomiting and spells of twitching of both arms and legs.

Ventriculography was performed for the first time at the age of 4 months (June 22, 1942). The intraventricular pressure was abnormally high; 150 cc. of fluid were removed and a similar amount of air injected. The ventriculograms showed the usual findings in cases of hydrocephalus: widely dilated lateral ventricles and a dilated 3rd ventricle. No air was seen in the Sylvian aqueduct or in the 4th ventricle.

Course. The child was discharged without any special kind of treatment being attempted. He lived in a home for orphans, where he was kept under constant observation. The head gradually increased in size. On Nov. 4, 1943 the circumference of the head measured 55.5 cm. (normal: 48). He was readmitted to the ward on Dec. 9, 1943. There was ptosis on the left side. The left pupil was dilated and did not react to light or accommodation. Left leg and arm were paretic. Deep reflexes in left arm and leg were considerably brisker than on the right.

There was bilateral plantar extensor response.

Cerebellar exploration and ventriculocisternostomy were carried out on Dec. 10, 1943. The cerebellum and the 4th ventricle were of normal appearance. It was found that no fluid came from the 3rd to the 4th ventricle through the Sylvian aqueduct, even when the baby cried or vomited. Thus it was obvious that a stenosis of the Sylvian aqueduct was present.

During the operation a puncture of the left lateral ventricle was attempted. The needle met with firm resistance at a depth where this should not be found. The cranial burr hole was enlarged, and a lighted retractor was introduced into a cavity filled with clear fluid. The convex surface of a "tumor" was seen and a biopsy specimen was excised for microscopic study. It proved to be the surface of the left hemisphere. This interesting abnormality had not been observed at ventriculography on June 22, 1942.

Ventriculography was performed again on Jan. 5, 1944. This time the findings were indeed unusual (Fig. 6). While the previous ventriculograms had disclosed the alteration ordinarily seen in cases of hydrocephalus, this time the whole ventricular system was dislocated toward the right side. The right lateral ventricle was widely dilated and the left lateral ventricle had assumed a deformed shape and an oblique position, deviating towards the right side. The 3rd ventricle was seen in a similar oblique position, having its lower end fixed above the sella, and slanting with its upper portion towards the right side. Between the left surface of the head and the left lateral ventricle air was seen in the subdural space to cover the left hemisphere.

Osteoplastic craniotomy was carried out Jan. 10, 1944. A large left parietal flap was turned down, exposing most of the left hemisphere. There were dense adhesions between dura and skull. The dura was hyperemic and 2 or 3 times normal thickness. Between the dura and the surface of the hemisphere was a large cavity filled with clear, colourless fluid. Multiple, rather thick trabeculae connected the hemisphere to the dura and the falx. After these trabeculae were cut, the hemisphere could be moved in all directions. With a dissector an opening was made through the corpus callosum and a lighted retractor was introduced into the ventricular cavity, allowing inspection of the interior of the left lateral ventricle. The septum pellucidum was seen to be the site of large perforations, through which the rubber tube in the right lateral ventricle could be seen and palpated. Most of the lateral wall of the left lateral ventricle could be seen and appeared normal. The temporal horn could not be inspected. No other abnormal findings were detected. The rubber tube was apparently in good position and could presumably drain the ventricular fluid into the cisterna magna in a satisfactory way. The wound was closed in the usual manner.
The postoperative period was not associated with complications and the boy was discharged to the orphans' home Jan. 25, 1944 in a satisfactory condition. The circumference of the head was 58.5 cm.

On examination January 1946, 2 years after operation, he was found to be in excellent condition, having developed normally, and showing no evidence of paresis. The head measured 60 cm. in its greatest circumference, thus having enlarged by only 1.5 cm. in the course of the last 2 years.

The patient presented no signs of intracranial hypertension. The eye-grounds showed pale optic discs and no edema. The edges of the discs were sharp. It was difficult to estimate the exact visual acuity, but vision seemed to be normal.

Ventriculography was performed Jan. 23, 1946 (Fig. 7). The same changes as detected by the previous ventriculography were present, except that there was no longer a marked dis-
ventriculograms showed a collection of fluid in the subdural space, covering and compressing the left hemisphere and dislocating the brain toward the right. This finding was not present in the ventriculograms of June 22, 1942.

Between June 22, 1942 and Jan. 5, 1944 rupture of the ventricular system must have taken place, admitting the ventricular fluid into the subdural space covering the left hemisphere. This finding was verified by operation, after having been diagnosed by ventriculography. The pia-arachnoid was glued closely together, so that no subarachnoid space could be seen. It is possible that adhesions between these membranes were present at the place of the rupture previous to its occurrence. It is possible also that the membranes became glued together at the time when the rupture of the ventricular wall took place, perhaps because of simultaneous hemorrhage. Lep-tomeningeal adhesions and signs of meningeal inflammatory reaction seem to be a frequent finding in cases of spontaneous ventricular rupture and were observed in the cases published by Pennybacker and Russell. Similar alterations may have been present in my case and may have caused the subdural accumulation of the cerebrospinal fluid.

The exact place of the rupture can not be pointed out with certainty. At the time of the operative inspection of the interior of the left lateral ventricle the mechanism of the pathological alterations was not clearly understood and the place of the rupture was not actually looked for. The case report contains no information that the place of the rupture was observed. It is possible that it was overlooked, and it is possible that it was located at a place that could not be well seen, for instance the left temporal horn. The pathological alterations in this case had the consequence that the fluid in the lateral ventricles became evacuated into the subdural space, where insufficient resorption took place, leading to compression of the left hemisphere and generalized intracranial hypertension.

While nature's attempt at treatment of the aqueductal stenosis resulted in a failure, the treatment by means of an artificial aqueduct (ventriculocisternostomy) seems to have been a success. Since the operation the development of the patient has been normal. He has lost the hydrocephalic appearance, he has become relieved of the signs and symptoms of intracranial hypertension, his head has grown at a normal rate, and he has shown normal intellectual development (Fig. 8).

Case 5. 11836/47. Gunnar A., a 41-year-old man.

Anamnesis. The patient stated that his vision had been poor since childhood. It had gradually become worse in recent years. In 1943 he suffered from meningitis and was treated successfully by sulphathiaisol. At that time the spinal fluid contained 39,160/3 cells. The fluid was sterile.

In 1944 vision became so reduced that he was no longer able to read newspaper print.

In 1946 another attack of meningitis occurred. The cerebrospinal fluid contained 25,000/3 cells, mostly polynuclear in type. No microbes could be detected. He was again treated by sulphathiaisol and became free of symptoms. Following this attack of meningitis he was ad-
mitted to the department of neurology at Rikshospitalet, Oslo. Neurological examination revealed bilaterally reduced vision: 5/30 R., and 5/24 L. There was rather marked atrophy of both optic discs. Roentgenograms of the cranium showed increased convolutional markings and separation of the sutures. The maxillary sinus showed signs of sinusitis on the left side. Percutaneous cerebral angiography was carried out on the right side. The findings suggested distention of the lateral ventricles.

In March 1947 meningitis again developed. He became unconscious and was admitted to a local hospital. The cerebrospinal fluid contained 20,000/3 polynuclear cells. No microbes could be seen. He was given penicillin treatment and recovered satisfactorily. His vision gradually decreased and he was re-admitted to the neurological department on April 29, 1947.

On examination it was noted that he was constantly shivering as if feeling cold, although he denied that the temperature of the room was too cold to suit him. The optic discs showed the same atrophy as previously. Otorhinological findings were not definitely abnormal.

Ventriculography was performed May 10, 1947; 200 cc. of fluid were removed and a similar amount of air injected. The picture taken with the brow up showed both lateral ventricles to be widely dilated. Air was visible in the 3rd ventricle, giving only a faint shadow. With the brow down, the 3rd ventricle apparently was well filled with air (Fig. 9), but the lateral view disclosed that this air was due to filling of a cyst behind the 3rd ventricle (Fig. 10), the cyst being of the kind seen in cases of spontaneous rupture of the cerebral ventricles with formation of an infratentorial cystic collection of fluid.

After ventriculography the patient became comatose. Examination of the intraventricular pressure showed this to be rather low. Ringer’s solution was injected into the ventricles, the air was expelled, and the intraventricular pressure adjusted to a normal level. The patient gradually became worse, however, and died with hyperthermia May 18, 1947.

Autopsy 19/47. There was considerable dilatation of both lateral ventricles and of the 3rd ventricle. The posterior wall of the 3rd ventricle showed a rupture reaching from the pineal gland to the quadrigeminal plate (Fig. 11). Here there was wide communication between the cavity of the 3rd ventricle and a cystic formation lined by the leptomeninges. The walls of the cyst were surrounded by the tentorium cerebelli, the cerebellar hemispheres, the
Figs. 9 and 10. Case 5. Ventriculograms May 10, 1947. *Left:* Both lateral ventricles are considerably dilated. Air is seen also below and between the posterior portions of the lateral ventricles at the usual place of the 3rd ventricle when air is present in that cavity. The lateral view discloses that this shadow is caused by a collection of air behind the 3rd ventricle. *Right:* Lateral view with brow down shows no air in 3rd ventricle. A cystic collection of air is seen superimposed on the lower portion of the lateral ventricles. This picture is taken with patient in same position as in Fig. 9.

Fig. 11. Case 5. Section of the brain showing the ruptured posterior wall of the 3rd ventricle and the cystic formation below the tentorium.
quadrigeminal plate and the pineal body. The Sylvian aqueduct showed a complete occlusion of the lumen. Microscopical examination of the stenosis showed this to be due to a neoplasm of somewhat atypical appearance, ependymoma being the most likely differential diagnosis.

GENERAL COMMENT

The infratentorial cysts in question usually do not lead to direct communication between the ventricular cavities and the subarachnoid space. As a rule the infratentorial cyst is lined with a membrane keeping the intracystic fluid separate from the subarachnoid fluid. For that reason one may be justified in arguing that an actual rupture has not taken place in spite of the presence of an infratentorial cyst. For the same reason the formation of the infratentorial cyst does not, as a rule, lead to spontaneous cure of the hydrocephalus.

As far as experience goes, there seem to be two favored locations for the breakdown of the ventricular wall: (1) The mesial wall of the temporal horn where it joins the posterior horn and the body of the lateral ventricle. (2) The anterior or the posterior wall of the 3rd ventricle.

It is possible that these two groups have distinctly different backgrounds.

1. If an occlusion of the Sylvian aqueduct is present in prenatal life, it is conceivable that the increased pressure of the intraventricular fluid is great enough to prevent complete closure of the choroidal fissure. It seems not unlikely that under such circumstances the point of entry for the posterior choroidal artery may stay open after the rest of the fissure has become closed. Such developmental background would explain why the cyst has the above-mentioned location (1) and so frequently is bilateral and symmetrical.

2. If the occlusion of the Sylvian aqueduct takes place after closure of the choroidal fissure has been completed, increase of the intraventricular fluid pressure naturally will lead to distention of the ventricular walls. The thinnest parts of the walls will yield to the pressure more than any other place. This may explain why under such circumstances the filmy anterior or posterior wall of the 3rd ventricle is another favored location for spontaneous "rupture" and eventual cyst formation.

In case prenatal occlusion of the Sylvian aqueduct has taken place, the cerebrospinal fluid will be prevented from filling the subarachnoid space to a normal degree. Secondarily this may lead to a faulty development of the subarachnoid space and its absorbing mechanism. Under such conditions the subarachnoid space may not be prepared to receive and absorb the cerebrospinal fluid even if rupture of the ventricular wall takes place at a later stage of life and creates a communication between the intra- and the extraventricular fluid. This would also add to the explanation of why spontaneous cure of obstructive hydrocephalus rarely occurs.

Spontaneous "rupture" of the cerebral ventricles is a pathological anatomical condition which is easily diagnosed by ventriculography. The pres-
ence of such cyst formation frequently indicates a pathological condition that can be treated advantageously by ventriculocisternostomy. The recognition of the condition has consequently gained considerable practical importance.

SUMMARY

Five cases of spontaneous rupture of the cerebral ventricular system are described. In 4 cases the rupture resulted in the formation of a cyst occupying the interpeduncular space, extending below the tentorium and covering the quadrigeminal plate.

In Case 1 an aperture was seen in the posterior portion of the lateral ventricle near the midline. This seems to be the most common location, as far as one can judge by the scarce literature. In Cases 2 and 5 a rupture of the posterior wall of the 3rd ventricle had taken place. In Case 3 there was no postmortem examination. Case 4 differs from the others inasmuch as the rupture did not create a cyst below the tentorium. After the spontaneous rupture the cerebrospinal fluid filled the supratentorial subdural space on the left side, resulting in an accumulation of the cerebrospinal fluid causing cerebral compression.

In 1 of the cases only (Case 1) the anamnesis gives information about an episode that probably was related to the actual rupture of the ventricular wall.

Stenosis of the Sylvian aqueduct is a common cause leading to spontaneous ventricular rupture, but slowly growing tumors also frequently come into consideration. The accompanying intracranial hypertension should be treated by ventriculocisternostomy.

REFERENCES