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Overdrainage Phenomena in Shunt Treated Hydrocephalus

By

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With 4 Figures

Summary

In addition to the high rate of shunt complications due to obstruction and infection there is a considerable number of undesirable side effects caused by excessive drainage of CSF. Four hundred shunt treated patients are analyzed for overdrainage signs: acute decompression symptoms produced by upward shifting of the brain stem, low pressure headaches (mostly transient), and microcephaly and head deformities like scaphocephaly in infants. Skull X-ray changes reflect adjustment to reduced intracranial content. Slit ventricles and a marked intolerance to minimal pressure rises may be quite troublesome. Subdural haematomas are only exceptionally space-occupying; in most instances they are space-filling. Causes, incidence, management, and prevention are discussed.

The introduction of valve regulated shunts in the treatment of hydrocephalus 20 years ago was certainly an incontestable progression, although shunt operations still have the highest complication rate in neurosurgery. Most problems in shunt surgery are related to obstruction and infection. There is, however, a considerable number of side-effects and complications, caused by excessive drainage of CSF.

All extracranial shunting systems work on the basis of a differential pressure valve. Not the ventricular CSF pressure itself, but the pressure gradient between the ventricles and the body space, where the CSF is diverted, determines CSF flow. Practically, pressure gradients arise from the following situations: 1. increase of CSF pressure, 2. negative pressure waves in the right atrium caused by profound ventilation, 3. siphon effect of the distal catheter in the upright position (Portnoy *et al.*, Fox *et al.*, Yamada *et al.*, and Epstein *et al.*).

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Those who have to deal with CSF shunts are glad when the shunts are working well. This might explain why overdrainage phenomena are only mentioned rarely in most follow-up studies. It is the intention of this paper to analyze 400 shunt treated hydrocephalus patients for clinical signs of excessive CSF drainage.

Own Clinical Material

Between 1964 and 1974 in the Neurosurgical University Clinic at Homburg/Saar 424 patients of all age groups were treated for progressive hydrocephalus of different aetiologies with ventriculo-atrial and, in a few cases, with ventriculo-peritoneal shunts. In addition there are five patients with lumbo-peritoneal shunts. Twenty four patients were eliminated because of poor documentation and inadequate information about postoperative course. Eight per cent of these patients died within three months of the first operation. In another eight per cent CSF-shunting was a palliative procedure for cerebral tumours; these patients died of their primary conditions within the same period. Three hundred and thirty six patients were seen regularly in our outpatient clinic. The average observation time was four years.

Table 1. *Types of Hydrocephalus*

Communicating hydrocephalus	167
Occlusive hydrocephalus	164
Hydrocephalus with dysraphic states	69
Total:	400 patients

Table 2. *Types of Shunt Systems, Primarily Used*

	Opening pressure	Number
Holter low pressure	11– 40 mm H_2O	190
Hakim low pressure	30– 45 mm H_2O	8
Hakim medium pressure	60– 80 mm H_2O	75
Hakim high pressure	95–125 mm H_2O	115
Raimondi-Unishunt	20– 50 mm H_2O	12
		400

Results

1. Changes of Cranial Size and Volume

What are the sequels of the above mentioned overdrainage mechanisms? First of all a normalization of the CSF volume and an

adequate reduction of ventricular size is desirable. In babies such a decrease of volume clearly shows in the head circumference curve. Besides the slow approximation to the normal we often see a rapid decline into the *microcephalic* range. In babies the rapid and ex-

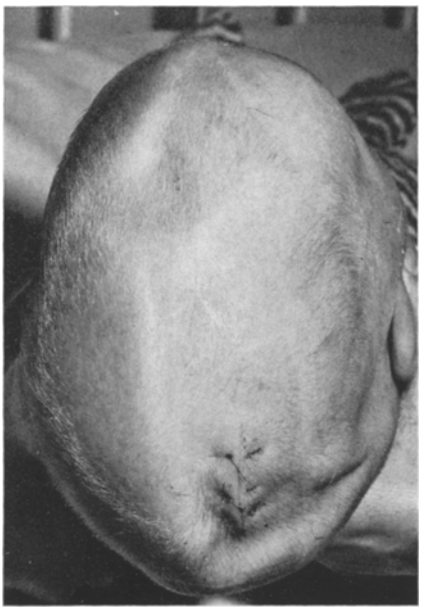

Fig. 1. Acute decompression. Deep sunken fontanelle, overriding of the parietal bones

cessive drainage of large ventricles causes the well known clinical picture of deep-sunken fontanelle, over-riding of the parietal bones, and finally dolichocephalic, and rarely brachycephalic, deformation of the skull (Fig. 1). In this sometimes grotesque deformation the skull consolidates because of premature synostoses (Fig. 2). The skull deformation is also irreversible, because surgical opening of the sutures does not help to normalize the cranial appearance when over-drainage of CSF persists. These misshapen heads are not only a cosmetic problem; they also express the simultaneous evacuation of the spatial buffer, which is mainly represented by the CSF.

Thirty three of the shunted babies developed marked micro-cephalus (distinctly below the 5 percentile). Eighteen of these had scaphocephaly with synostosis of the sagittal suture. The other cases had oxy- or brachycephaly.

2. Slit Ventricles

Another problem arises from the constriction of the shunted ventricle, and this is quite common as we now know from computerized tomography. The close contact of the ventricular wall with the catheter favours occlusions of the catheter. Fortunately not all cases of hyper-drained small ventricles are followed by ventricular catheter obstruction. Other factors like foreign body reaction and infection may be of some importance in the development of this complication. In our 97 cases of ventricular catheter occlusion the ventricular constriction was certainly one of the most prominent causes. In 12 cases recurrent catheter occlusions led to the clinical diagnosis of slit ventricle, mainly because of the difficulty of localizing the ventricle during operation. In one case of repeated catheter occlusions an involuntary third ventriculostomy with placement of the catheter in the cisterna interpeducularis definitely helped the patient. In another case we were unable to find the ventricles and we had to perform a bitemporal decompression, as suggested by Epstein. In addition we have seen five children, who intermittently showed the picture of increased intracranial pressure. We think that in these cases complete occlusion of the ventricular catheter leads to dilatation ventricles, and this again causes opening of one of the perforations of the catheter. Contrast medium studies in these cases seem to prove such a theory.

3. Negative Pressure Syndromes

Over-rapid decompression of a large hydrocephalus may cause an acute clinical syndrome with vegetative signs such as tachycardia, and also severe neurological signs such as unconsciousness and other brain stem signs. These symptoms are caused by upward shifting of the brain stem. They are completely and easily reversible by lowering the head. In most instances, however, these syndromes are caused by uncontrolled loss of CSF during operation. This can be prevented with appropriate operative technique and postoperative care.

Low pressure headaches were complained of by 13 treated patients, and in some cases nausea and vomiting were present. Diagnosis was proved by measuring the ventricular pressure in the flat and sitting positions. In two cases only did the shunt system have to be removed completely because of untolerable headaches. In two more cases valves with higher opening pressures were inserted. The interposition of an antisiphon device helped in only one case. In another case it was ineffective, and in a third case it caused obstruction of the system.

4. Intolerance of CSF Pressure Elevations

Patients with long-standing, excessive CSF drainage may become extremely sensitive to minimal intracranial pressure rises. In these patients an increase of CSF pressure within the normal range may

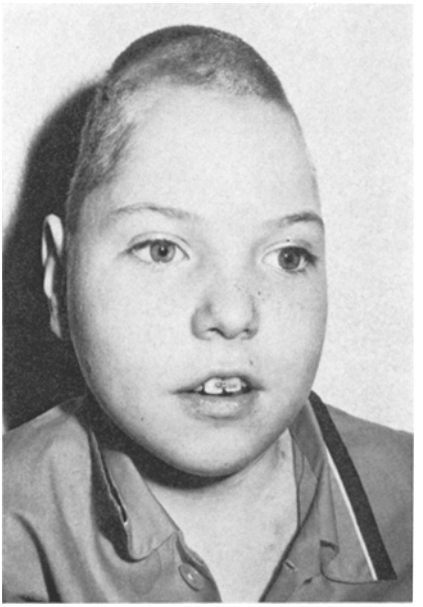

Fig. 2. Scaphocephalic head deformation. Twelve-years-old boy, who proved to be extremely sensitive to pressure elevations within the normal range, when his cardiac catheter was obstructed

cause clinical pressure symptoms up to respiratory arrest. We have seen cases where attempts to raise the pressure above 60–80 mm H_2O led to clinical pressure symptoms and impairment of consciousness.

Observations were possible on nine patients. Four of these were connected to a pressure-controlled external CSF drainage (one patient because of valve infection and ventriculities, three patients on account of the treatment of subdural haematoma). In five more patients the occlusion of the distal shunt catheter produced severe pressure signs. The recording of ventricular pressure showed normal pressure values within the range of 50 to 150 mm H_2O.

Why do some of the patients, who have been accustomed over many years to low intracranial pressure, lose the ability to tolerate minimal pressure elevations? Two factors may be important for such a loss of adapting properties: 1. The evacuation of the spatial buffer,

which was mentioned already above, and 2. the alteration of the biochemical quality of brain parenchyma (decrease of brain compliance).

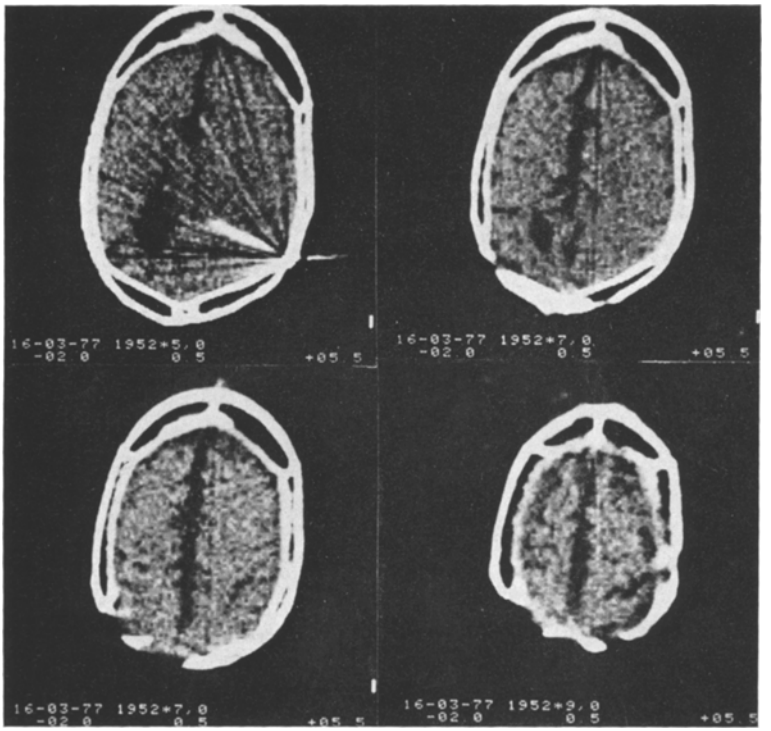

Fig. 3. Computerized tomography of a 15-years-old girl, who has developed a second inner skull to adapt her cranial vault to the reduced intracranial volume

5. Subdural Haematoma

Chronic subdural haematoma or subdural effusion is thought to be the most severe sequel of excessive CSF drainage. We found in 17 of our cases subdural haematomas, effusions, or callus. In addition there was one case of epidural haematoma. Six subdural haematomas were discovered within the last two years by routine computerized tomography (Figs. 3 and 4). In seven cases the diagnosis has been established more casually after demonstration of capsular calcifications in the skull X-ray, typical findings in the brain scan, or accidentally during operative revision of the shunting system. This experience shows that subdural effusions in shunted patients in most instances did not cause specific clinical symptomatology, and

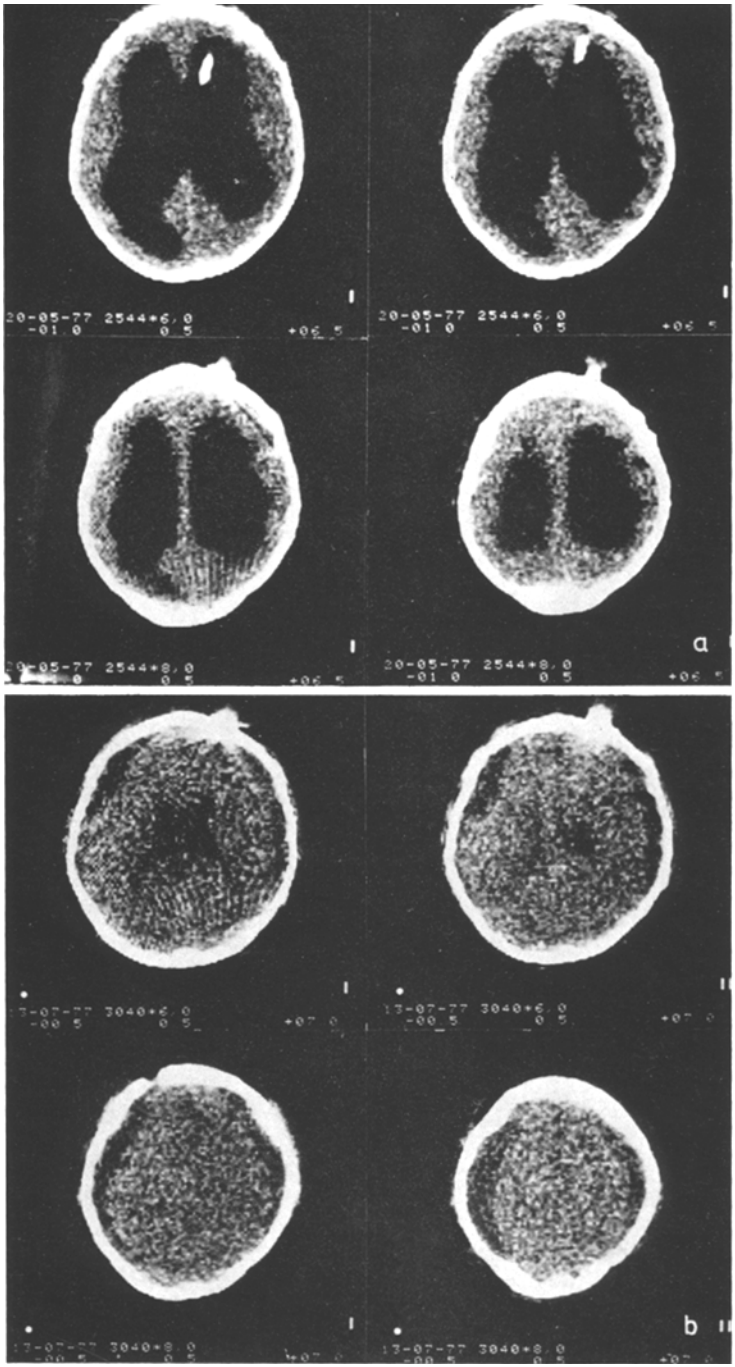

Fig. 4. Computerized tomography of a 12-years-old girl. a) Marked hydrocephalus due to aqueduct stenosis. b) Control eight weeks after ventriculoperitoneal shunt. Normalization of ventricular size. Bilateral subdural effusions. She is perfectly well. Further controls will show, whether surgical treatment is necessary

that its discovery is frequently accidental. We therefore presume that in our patient material the incidence of subdural effusions in shunt treated patients may be even higher.

In only five cases did the subdural haematoma cause severe sequelae: impairment of consciousness, hemiparesis, and brain stem herniations. In these cases, of course, the operative removal of the space-occupying haematoma was necessary. Successful treatment, however, was only possible when, after removal of the haematoma, the collapsed ventricles were dilated by occlusions of the CSF shunt. Since most of these patients were accustomed over the years to extremely low intracranial pressures, the elevation of pressure into the normal range frequently caused pressure symptoms. Therefore it does not surprise us that four of these patients were left with permanent cerebral damage, although the haematoma was removed and the CSF shunt was later restored. Fortunately only few of the subdural haematomas or effusions are progressive space-occupying lesions, in which surgical intervention is inevitable. The long term follow up of the unoperated cases has shown us that progression up to the state of clinical symptoms is rare.

In view of such observations the question arises whether all subdural effusions must be considered as severe complications. Are they not rather adaptive mechanisms to the excessive reduction of volume in a system in which the total volume is determined by the rigid cranial vault? It seems important to point out that the five treated subdural haematomas and the two cases that were discovered accidentally, would be better classified as cases of subdural callus. The morphological aspects at operation distinctly differed from the classical chronic subdural haematoma or hygroma: they consisted of unusual, thick, fibrous membranes, containing pulpy and fatty material and little or no liquid. In all cases, therefore, burr holes would have been ineffective. A large craniotomy with removal of the capsule was necessary.

Discussion

In this investigation we have tried to list all those side effects and complications which are due to excessive CSF drainage occurring in different extracranial shunting systems.

It is astonishing that in most clinical reports of CSF shunts these aspects are only mentioned partly or not at all (Shurtleff and Foltz, Guidetti, Forster, Hemmer, Raimondi, and Becker). Detailed aspects of the hyperdrainage problem, however, are mentioned quite frequently in the literature. In the numerous case reports the com-

bined incidence of *craniostenoses* and hydrocephalus has been mentioned. Anderson was the first to recognize the relation between excessive CSF drainage and premature synostosis of cranial sutures; others wrongly consider craniostenosis to be a phenomenon associated with hydrocephalus (Hemmer).

Craniostenoses are only one aspect of the abundant skull changes. Systematic descriptions of these changes were given by Griscolm, Kauffmann, and Villani. Griscolm entitled the radiological sequels of hyperdrainage correctly as contracting skull. The *changes on plain skull X-ray* can be summarized as follows: 1. microcrania and skull deformities, 2. primature synostoses, 3. thickening of the cranial bones and, endocranial growth of the lamina interna, 4. hyperpneumatized sinuses and mastoids, 5. small sella and diminution of the foramina of the cranial nerves. All these changes indicate a reduction of the intracranial capacity due to extracranial CSF drainage. Unfortunately the X-ray findings in our documents are poor. In only a few cases was synostosis of cranial sutures described.

Roberts and Rickham have seen in a patient material of 800 children only 8 cases of secondary craniostenoses. They were all operated on. The value of such operations seems to be quite questionable, when they are carried out without urgency, without intracranial pressure signs. After all, the skull has been adapted to the decreased intracranial volume and when the CSF drainage is functioning after opening of the sutures the skull will not grow any more.

Bilateral craniectomy for *slit ventricles and repeated ventricular catheter occlusions* however is quite different (Epstein). In these rare cases this operation might be the only possibility of reducing threatening intracranial pressure and of providing slow widening of the ventricles.

In Torkildsen's ventriculo-cisternostomies ventricular catheter occlusions are unknown, which impressively proves that in the aetiology of this complication a foreign body reaction is of minor importance (Kirch).

Emery has described the *movements of the brain stem* caused by over-rapid CSF drainage in an informative analysis of over 60 autopsies on shunt treated children. Jackson *et al.* have pointed out that intracranial hypotension may produce *headaches.* Our experiences have shown that fortunately these complaints rarely persist, and that further operations are only exceptionally necessary.

The most serious complication of excessive CSF diversion is thought to be the *chronic subdural haematoma.* In 1952 Anderson noted the relation between overdrainage and haematoma in three cases. After that more reports were published in the literature. Becker

and Nulsen had 7 cases among 140 patients (5%). Illingworth found 8 haematoma patients in 175 cases (4.5%). Samuelson has published a remarkably high rate of secondary subdural haematomas (21%). In the older publications (Forrest, Shurtleff) this complication is almost never mentioned. It must be suspected that the true incidence is probably much higher, primarily because secondary subdural effusions frequently stay asymptomatic and because demonstration has not been possible until recently when easy screening tests like brain scan and computerized tomography became available (Katzner).

Generally, in most clinical reports (Hemmer, Illingworth, Samuelson, McCullough, Portnoy) the surgical evacuation of the haematoma or hygroma is advised. Indeed in some cases an operative intervention cannot be avoided. In most cases, however, an expectative attitude with adequate controls is justified. Our experience with 12 untreated subdural lesions in valve patients seems to confirm this regime. Emery, in the above-mentioned neuropathological study, has described chronic subdural haematomas which did not cause compression of the underlying brain. In addition he described in many cases an augmentation of the subdural connective tissue and a thickening of the arachnoid. He characterized these structures as space fillers. Our cases with subdural callus would not have profited from burr hole drainage, which was done by Illingworth and Samuelson in most of their cases. The latter were adults, whereas our patients were children between two and twelve years of age. Only the case of Hemmer described as haematoma was similar to ours. Hemmer, Illingworth, and Samuelson have occluded or removed at the same time the CSF shunt. Illingworth needed ventricular drainage in four cases. All our treated patients needed external ventricular drainage.

The poor results of Illingworth and Samuelson and also our depressing experiences with surgically treated subdural lesions in valve patients justify in many cases an expectant attitude after careful consideration of the risks.

Is there a possibility of avoiding the hyperdrainage effects presented in this paper? In the last years the shunt producing companies have offered valves with higher opening pressures in order to reduce the incidence of hyperdrainage signs. The elevation of the opening pressure, however, was only possible up to the range of the upper normal pressure. The siphon effect, or negative pressure waves in the right atrium, will easily overcome these resistances. In 1973 Portnoy *et al.* presented the *antisiphon device,* which indeed enables a reversible shunt occlusion when a negative pressure occurs within the system. The authors, however, admitted one year later

that the reversible occlusion valves were not effective in all cases, and this means that subdural haematomas cannot always be prevented (McCullough, Fox 1974). Our experiences with the antisiphon device have shown negative as well as positive results. Reports with larger experiences are not published as yet.

For several years constructional designs for diversionary systems with variable opening pressure, partially in combination with intracranial pressure recording have been in existence. Up to now such innovations did not appear on the market, probably because the technical realization or the clinical application is difficult.

Conclusions

Investigations in 400 hydrocephalic patients with valve regulated shunts show that, beside the conventional complications which necessitate a revision rate of 1.05 revisions per patient, there is a considerable number of complications and side-effects, which are due to excessive drainage of CSF. More or less all extracranial CSF shunts are concerned with these problems, since all shunts are working on the basis of differential pressure valves. Over-rapid drainage of CSF may cause brain stem shifting with corresponding clinical symptomatology. This complication, however, is frequently due to excessive CSF loss during operation.

Older children and adults often suffer from low pressure headaches, which fortunately are mostly transient. In five cases the valve was modified. In only two cases the shunt was removed because of persistent complaints.

In babies chronic hyperdrainage causes changes in the cranial volume and shape: microcephaly in 33 cases, with scaphocephaly in 18.

The skull shows chronic changes like microcrania, thickening of the lamina interna, hyperpneumatization, and cranial synostosis. These changes indicate an adaption to the reduced intracranial volume.

More important, however, is the danger of ventricular collapse and slit ventricles. The approximation of ventricular walls and the close contact of these with the ventricular catheter favour catheter occlusions. In some cases it leads to repeated ventricular catheter occlusions (12 patients).

In some cases there is a remarkable sensitivity to minimal intracranial pressure elevations. Shunt occlusions may cause in such cases threatening pressure symptoms, although the CSF pressure is still in the normal range (nine patients).

Subdural haematoma, effusions, or callus are in most instances not space-occupying, but only space-filling. We have discovered among the 400 cases 17 patients with such complications. Only five were operated on.

Low pressure valves seem to favour hyperdrainage phenomena. With high pressure systems they cannot be prevented. The antisiphon device of Portnoy is now the only method which allows prevention of hyperdrainage in some cases.

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