Hydrocephalus After Aneurysmal Subarachnoid Hemorrhage

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KEYWORDS

- Hydrocephalus • Fenestration
- Subarachnoid hemorrhage • Aneurysm

Hydrocephalus often complicates the initial injurious effects of subarachnoid hemorrhage (SAH) (**Fig. 1**). In 1928, Bagley[1] was the first to suggest that ventricular dilatation could be a consequence of SAH. Most studies report an overall 20% to 30% incidence of hydrocephalus after SAH.[2–4] Although debate still exists over its pathophysiology, this condition typically presents acutely but can also occur in a delayed fashion, rarely even months after the initial hemorrhage. Its clinical sequelae can be devastating and lead to further neurologic deterioration and longer hospital stays. Early recognition and treatment, however, can lead to improved patient outcomes. Several strategies have been developed to minimize the need for placement of either temporary intraventricular catheters (IVCs) or permanent shunts. Intraoperative techniques used to reestablish normal cerebrospinal fluid (CSF) flow and resorption include fenestration of the lamina terminalis and thorough irrigation of blood out of the arachnoid cisterns. Postoperative techniques used to encourage CSF reabsorption in patients with IVCs or lumbar drains involve a steady, daily increase in the pop-off pressures, which is guided by recorded intracranial or thecal pressures, CSF output volume, and the patient's neurologic status. In patients without IVCs or lumbar drains but with persistent symptoms, serial lumbar punctures are necessary. Endovascular treatment of aneurysms may be associated with a higher rate of shunt-dependent hydrocephalus. In some institutions permanent shunting rates have been reduced to approximately 7%.

ETIOLOGY

The exact mechanism by which hydrocephalus develops after SAH remains poorly understood, although altered CSF dynamics in the acute and chronic states have been extensively studied. Although it is generally accepted that hydrocephalus after SAH is of the ''communicating'' type,[5] it is likely that this condition has communicating and noncommunicating components. Decreased absorption of CSF at the arachnoid granulations is defined as communicating hydrocephalus and an anatomic obstruction, as noncommunicating. Traditionally, if all 4 ventricles are equally dilated on CT scan, then hydrocephalus is presumed to be of the communicating type. This interpretation does not take into account that if the obstruction occurs at the foramina of Luschka and Magendie (ie, the outflow of the fourth ventricle), a noncommunicating or obstructive type of hydrocephalus may be misinterpreted radiologically as being communicating, given that all the ventricles are dilated. It is generally assumed that fibrosis of

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Neurosurg Clin N Am 21 (2010) 263–270
doi:10.1016/j.nec.2009.10.013
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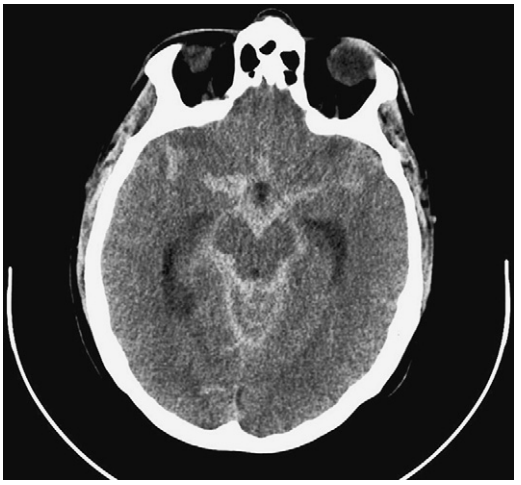

Fig. 1. Axial CT scan demonstrating acute SAH and enlarged third ventricle and temporal horns.

the leptomeninges and arachnoid granulations from blood product deposition causes impaired CSF flow and decreased absorption.[6,7]

There is increasing evidence, however, that hydrocephalus after SAH may be caused primarily by fibrosis and partial obstruction of the fourth ventricular outflow and secondarily by impaired CSF absorption. Based on this understanding, creation of an anterior third ventriculostomy has been proposed to facilitate CSF fluid dynamics with increased blood clearance, decreased leptomeningeal fibrosis, and better balance between CSF production and resorption.[5] In the authors' experience, the overall shunt rate in patients undergoing fenestration of the lamina terminalis can be reduced to 2.3%, whenever possible. The efficacy of lamina terminalis fenestration, however, has yielded conflicting results in other series, and a multi-center, randomized, controlled trial will most likely be necessary to determine the overall effectiveness of this technique.[8,9]

The type of hydrocephalus may be a function of the site of hemorrhage and not of the temporal breakdown of blood in the subarachnoid space.[10,11] This hypothesis may explain why ruptured posterior circulation aneurysms are associated with higher rates of hydrocephalus as compared with ruptured anterior circulation aneurysms.[12] Posterior circulation aneurysmal rupture may be more likely to cause impaired CSF egress from the fourth ventricle and an obstructive pattern of hydrocephalus. Alternatively, anterior circulation aneurysmal rupture may cause hydrocephalus primarily by fibrosis of the leptomeninges and arachnoid granulations and result in a communicating pattern in the acute and delayed states. It is evident that the pathophysiology of chronic hydrocephalus remains poorly understood and that several hypotheses exist regarding its cause.[11]

DIAGNOSIS

Acute hydrocephalus, which develops 48 to 72 hours after SAH, occurs in approximately 20% of patients.[13] Most patients with aneurysmal SAH present with headache, nausea, and vomiting, which are symptoms attributable to the presence of acute blood in the subarachnoid space but are also compounded by hydrocephalus. Subacute hydrocephalus, which develops 3 to 7 days after the hemorrhage, is rare and has a frequency of 2% to 3%.[3] Because the clinical diagnosis of hydrocephalus after SAH is difficult, its recognition is based primarily on radiographic findings, specifically CT scans.

Although several ventricular measurements based on CT studies have been used to establish the diagnosis of hydrocephalus, currently, the preferred marker for this condition is the bicaudate index. Historically, there has been an evolution of radiological markers for hydrocephalus. In 1979, Vassilouthis and Richardson[14] measured the ratio between the width of the lateral ventricles at the foramen of Monro and the inner diameter of the skull at the same level. A ratio less than 1:6.4 was considered normal and a ratio more than 1:4 represented marked ventricular dilatation, suggestive of hydrocephalus. In 1970, Galera and Greitz[15] compared the maximum width of the frontal horns to that of the skull at the same axial level. Other studies have focused on volumetric measurements, suggesting that linear measurements are less accurate.[16] Zatz and colleagues[17] reported that the best correlation between ventricular volume and linear measurements existed with the width of the third ventricle. However, they concluded that the empiric radiographic evaluation by a radiologist is more accurate than any linear ratio in diagnosing hydrocephalus. Currently, the preferred system for the objective diagnosis of hydrocephalus is based on the bicaudate index (**Fig. 2**).

Using data from 2 separate control groups showing the distribution of bicaudate values in patients without neurologic disease,[18,19] Gijn and colleagues[20] proposed that hydrocephalus should be diagnosed when the bicaudate index was more than the age-corrected 95th percentile (**Table 1**).

In this manner, atrophic changes that result in ventriculomegaly and are not the result of increased ventricular CSF pressures are taken into account. They then prospectively studied 174 consecutive patients with SAH and found

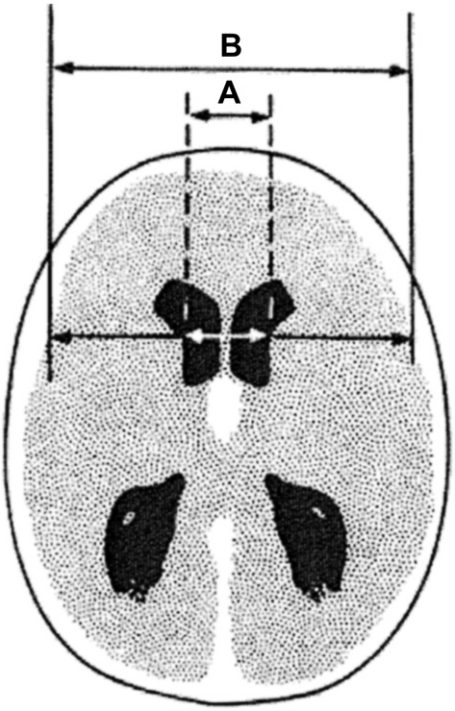

Fig. 2. Axial CT scan demonstrating method for determining bicaudate index (A, B). (A) is the width of the frontal horns at the level of the caudate nuclei; (B) is the diameter of the brain at the same level. (*Reproduced from* Van Gijn J, Hijdra A, Wijdicks EF et al. Acute hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg 1985;63(3):355–62; with permission.)

that 20% (34 of 174) had bicaudate indices greater than the 95th percentile for their age. Using similar criteria, Hasan and colleagues[3] reported a consecutive series of 473 patients with SAH and found an incidence of acute hydrocephalus in 19% of patients (91 of 473). Several large retrospective series have confirmed these findings.

Table 1
Upper 95% confidence value for ventriculocranial ratio stratified by age, as proposed by Gijn and colleagues

Age (Years)	Upper 95% Confidence Value
<30	0.16
<50	0.18
<60	0.19
<80	0.21
<100	0.25

Van Gijn J, Hijdra A, Wijdicks EF, et al. Acute hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg 1985;63(3):355–62.

Chronic hydrocephalus (presenting later than one week after SAH) develops in an additional 10% to 20% of patients.[21] Although the cause may be different, this diagnosis must be entertained in the setting of progressive neurologic decline. As a general rule, SAH patients who regress clinically weeks to months after discharge should have a follow-up CT scan and clinical evaluation to rule out delayed hydrocephalus.

PREDICTIVE FACTORS

Experimental studies have shown that the injection of blood into the subarachnoid space results in intracranial pressure (ICP) elevation higher than that caused by infusion of an equivalent volume of saline. This effect is generally attributed to increased CSF outflow resistance at the level of the subarachnoid space or the arachnoid granulations caused by blood components, such as erythrocytes and proteins.[2] The infusion of heparinized blood causes only a transient rise in ICP, implying that fibrin formation and deposition in the subarachnoid space and arachnoid granulations play an important role in this process.[2,22] Such studies have partially elucidated the complex changes resulting from the presence of blood in the subarachnoid space, and have supported clinical and radiographic variables associated with hydrocephalus.

Clinically, the most important prognostic feature associated with the development of hydrocephalus is the neurologic condition of the patient at the time of presentation. Data from 3521 aneurysmal SAH cases in the Cooperative Aneurysm Study show that several factors were related to symptomatic hydrocephalus, which are listed in **Table 2**.[12] Among these, a poor level of

Table 2
Admission variables predicting clinical hydrocephalus

Variable	*P* Value
CT Hydrocephalus	<.001
CT intraventricular hemorrhage	<.001
Consciousness level	<.001
Pre-SAH hypertension	<.001
Age	<.001
CT SAH	=.005
Posterior circulation aneurysm	=.012
Postoperative hypertension	=.024

Reproduced from Graff-Radford NR, Torner J, Adams Jr HP, et al. Factors associated with hydrocephalus after subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. Arch Neurol 1989;46(7):744–52; with permission.

consciousness and the presence of an intraventricular hemorrhage had a high statistical correlation. In a separate cohort study of 3120 patients, radiographic ventriculomegaly, ventilation on admission, aneurysms in the posterior circulation, and giant aneurysms were all predictors of shunt-dependent hydrocephalus.[23] Higher Fisher grades, and angiographic vasospasm on SAH day 7 have also been associated with hydrocephalus after SAH.[24]

TREATMENT

Reports of the proportion of patients with aneurysmal SAH who require permanent shunting are highly variable. In part, this variability is probably a result of treating neurosurgeons basing their decision to pursue permanent shunting on the presence of radiographic hydrocephalus alone or pursuing shunting only when symptomatic hydrocephalus is evident. Another important determining feature is when this decision is made during the course after the initial hemorrhage. For instance, according to the report of 473 patients with aneurysmal SAH by Hasan and colleagues,[3] 19% of their patients had radiographic hydrocephalus, but only two-thirds approximately were symptomatic, resulting in a rate of symptomatic hydrocephalus of only 13%. Furthermore, nearly half of their patients with symptomatic hydrocephalus improved spontaneously in the early stages, resulting in a final rate of persistent, symptomatic hydrocephalus of approximately 7%. Such findings are similar to those reported 10 years earlier by Vassilouthis and Richardson.[14] Hasan and colleagues noted a similar incidence of radiographic hydrocephalus in a group of 46 patients with SAH and negative arteriograms, but only one of these 46 patients (2%) developed symptomatic hydrocephalus.

Nevertheless, there is a minority of aneurysmal SAH patients in whom either temporary or permanent CSF diversion is of benefit. Hasan and colleagues noted improvement in 78% of the 32 patients treated with external ventricular drainage or a shunt. Raimondi and colleagues[25] saw improvement in 86% of 21 patients. Although initial studies noted high rates of infection with external ventricular drainage (50% in the Hasan series), precautions such as subcutaneous tunneling of the catheter, administering prophylactic antibiotics at the time of IVC placement, minimizing the duration of drainage to 5 days or less, and maintaining a closed drainage system have reduced infection rates to well below 10%. In a large study focusing on drainage days (DD) with external ventricular or lumbar drains, an overall infection rate was 6.3 per 1000 DD for ventricular drains and 19.9 per 1000 DD for lumbar drains, with an overall device-associated meningitis rate of 8.6 infections per 1000 DD.[26] Patients with poor grades (Hunt and Hess IV or V) typically require temporary CSF diversion.

Repeat rupture of an unsecured aneurysm is a major concern associated with placement of an IVC and continuous external ventricular drainage. Early studies reported a 43% incidence of rebleeding associated with CSF diversion in patients with unsecured aneurysms.[3] In another series in which ventricular drainage was pursued only when ICP was greater than 25 mmHg, a 17% rate of rebleeding was noted.[27] Patients with poor grades (III-V) have a higher incidence of rebleeding (25% vs 9.2% in patients with good grades).[28,29] Recent studies, however, have questioned whether CSF diversion via ventriculostomy or lumbar drainage increases the risk of rebleeding.[30] Perhaps this corresponds to improved control of ICP after ventriculostomy and lumbar drain placement and the natural history of unsecured ruptured aneurysms.

Temporary CSF diversion can be accomplished by insertion of an IVC (ventriculostomy), insertion of a lumbar drain, or with serial lumbar punctures. The need for permanent shunting can be reduced using several intraoperative maneuvers, such as fenestration of the lamina terminalis, opening the arachnoid cisterns, and thorough removal of subarachnoid clots. There is controversy over prolonged temporary CSF diversion increasing the incidence of shunt dependency.[31,32] Permanent CSF diversion procedures include ventriculoperitoneal shunting (VPS) (and its ventriculopleural and ventriculoatrial variants) and lumboperitoneal shunting (LPS). Currently the criteria for permanent CSF diversion are as varied as the ways of achieving it. Although the decision for permanent shunt placement is highly physician-dependent, a prolonged course of CSF diversion, persistent elevated ICPs, high drainage volumes, and persistent poor neurologic condition are criteria that justify permanent CSF diversion (**Fig. 3**).

Endovascular embolization for the treatment of intracranial aneurysms has become increasingly common, and it may be associated with a higher rate of shunt-dependent hydrocephalus. A large single-institutional review and meta-analysis comparing the risk of shunt dependence in patients with ruptured intracranial aneurysms treated by surgical clipping or endovascular coiling concluded that clipping of ruptured aneurysms may be associated with a lower incidence of shunt dependency.[24] In 385 patients treated at a single institution, those undergoing endovascular coiling had a higher shunting rate of 19.6% versus that of

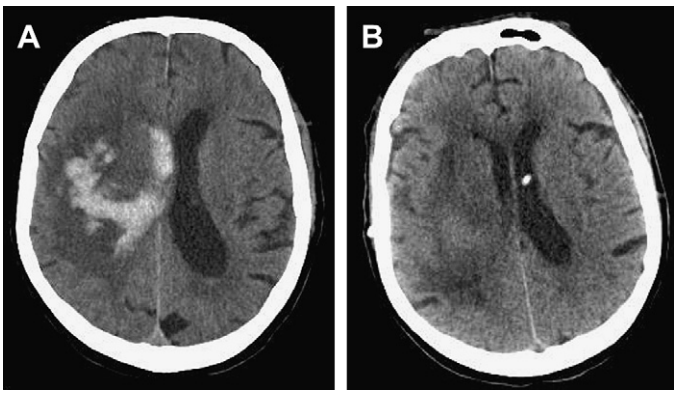

Fig. 3. (*A*) and (*B*) Initial CT scan revealing acute intraparenchymal and intraventricular hemorrhage (*left*). The patient was found to have a ruptured right internal carotid artery terminus aneurysm and underwent endovascular embolization. Follow-up CT scans showed persistent ventricular dilatation. A follow-up CT scan reveals the ventriculostomy catheter tip, decreased ventriculomegaly, and blood resolution (*right*). The patient required permanent CSF diversion with a ventriculoperitoneal shunt.

the surgical group, which was 17.4%. The overall shunt-dependent rate in this study was 18.4%. Although this difference was not statistically significant, combining this data with that of 4 other large series, for a total of 1718 patients, revealed a statistically significant higher rate of shunt dependence of 20.9% in endovascular patients, as opposed to 17.4% in surgical patients. Perhaps, clearing the subarachnoid clot and opening of the cisterns may lead to better CSF circulation and a reduced incidence of shunt dependence. A summary of the characteristics of the studies included in the meta-analysis is listed in **Table 3**.

MANAGEMENT STRATEGIES

The authors have summarized their strategy for the management of hydrocephalus after aneurysmal SAH (**Fig. 4**). At the authors' institutions, the rate of VPS placement after aneurysmal SAH has decreased to about 7%. They attribute this low rate to intraoperative fenestration of the lamina terminalis and a strict postoperative protocol in which postoperative external CSF drainage is minimized and CSF absorption is encouraged. Patients taken to the operating room with a preoperatively placed IVC typically do not leave the operating room with the IVC. During surgery, the authors create an anterior third ventriculostomy by fenestration of the lamina terminalis whenever this structure is accessible, open the arachnoid cisterns, and irrigate out the subarachnoid clot. Fenestration of the lamina terminalis is always accomplished during frontosphenotemporal (pterional) approaches, but is obviously not possible during suboccipital, interhemispheric, or subtemporal approaches. Although, on occasion, an intraoperative IVC is placed for additional brain relaxation, this is removed after intraoperative fenestration of the lamina terminalis.

In the intensive care unit, patients are observed for evidence of symptomatic hydrocephalus. The intraoperative creation of a third ventriculostomy transforms any postoperative hydrocephalus into the communicating type. If progressive hydrocephalus is observed clinically or radiographically, serial lumbar punctures or placement of a lumbar drain catheter is performed. In rare instances, an IVC is placed in the unit postoperatively. In such cases, the pop-off is raised by 5mmHg every

Table 3
Summary characteristics of studies included in the meta-analysis, as described by de Oliveira and colleagues

Series	Number of Patients	Treatment Ratio Number of Clip/Coil	Total Shunt Rate (%)	Shunt Rate (%) Clip/Coil (*P* Value)
Gruber and colleagues[33]	187	125/62	21.4	23/18 (0.45)
Dorai and colleagues[34]	718	684/34	21.0	20/47 (0.001)
Dehdashti and colleagues[35]	245	180/65	15.5	14/19 (0.53)
Varelas and colleagues[36]	183	135/48	6.6	4.4/12.5 (0.16)
De Oliveira and colleagues[24]	385	212/173	18.4	17.4/19.6 (0.59)

Data from de Oliveira JG, Beck J, Setzer M, et al. Risk of shunt-dependent hydrocephalus after occlusion of ruptured intracranial aneurysms by surgical clipping or endovascular coiling: a single-institution series and meta-analysis. Neurosurgery 2007;61(5):924–33 [discussion: 33–4].

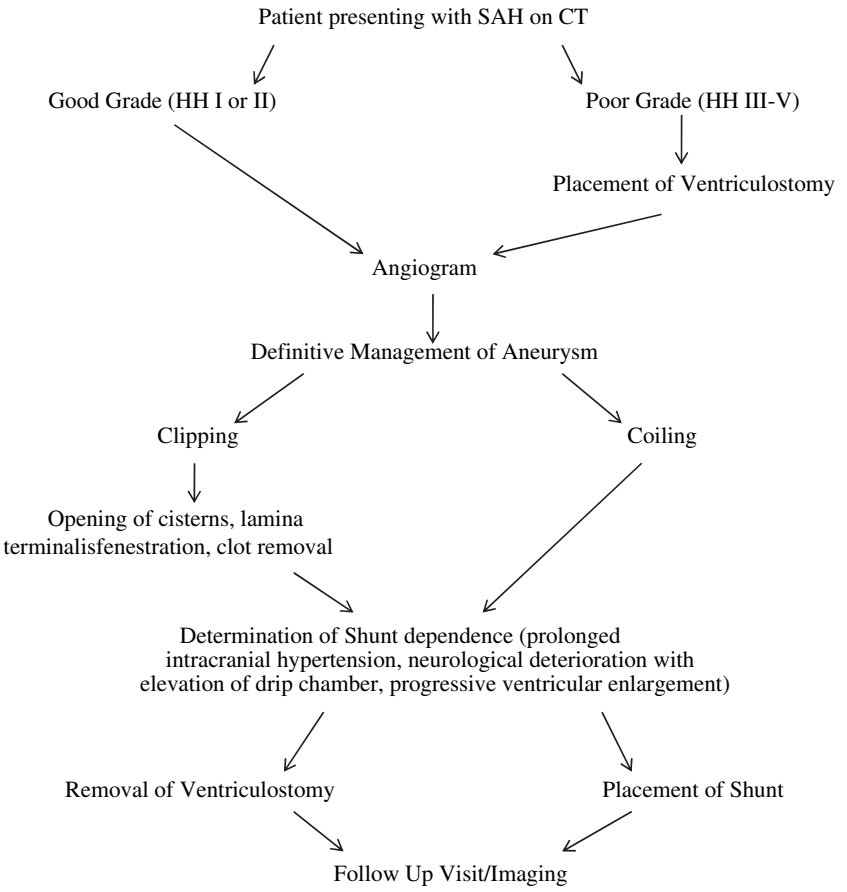

Fig. 4. Management strategies for patients with aneurysmal SAH.

24 hours, with close monitoring of ICP measurements, total CSF output, and the patient's neurologic condition. Once a pop-off of 20mmHg is reached and seems to be tolerated for 24 to 48 hours, the IVC is clamped. Provided that patients tolerate clamping for an additional 24 to 48 hours, a CT scan is obtained as a baseline and the IVC (or lumbar drain catheter) is removed. Further symptomatic hydrocephalus is managed with subsequent lumbar punctures. Either prolonged IVC dependence or the extended need for lumbar punctures then leads to insertion of a VPS or LPS.

A review of the literature reveals a broad range of shunting rates for aneurysmal SAH. Placement of a shunt depends on the biologic manifestations of hydrocephalus and the neurosurgeon's approach to this problem. The authors' inclination to minimize the rate of shunt placement may require patients to spend a few more days in the intensive care unit or undergo serial lumbar punctures. Although temporarily inconvenient, such steps may help avoid lifelong shunt dependency, a state that most patients and neurosurgeons prefer to avoid.

SUMMARY

Hydrocephalus after SAH has been recognized for over 80 years. Although the cause of this problem is not fully understood, more is known about this alteration of CSF dynamics from acute subarachnoid blood since Bagley's initial description. Although further clinical and experimental work is necessary to grasp the complex pathophysiology of hydrocephalus after SAH, ongoing awareness of this delayed complication and rapid intervention in the form of either temporary or permanent CSF diversion are required to minimize the devastating effects that can result from this condition.

REFERENCES

1. Bagley C Jr. Blood in the cerebrospinal fluid. Resultant functional and organic alterations in the central nervous system. A. Experimental data. Arch Surg 1928;17:18–38.
2. Brinker T, Seifert V, Stolke D. Acute changes in the dynamics of the cerbrospinal fluid system during

experimental subarachnoid hemorrhage. Neurosurgery 1990;27:369–72.
3. Hasan D, Vermeulen M, Wijdicks EF, et al. Management problems in acute hydrocephalus after subarachnoid hemorrhage. Stroke 1989;20(6): 747–53.
4. Heros RC. Acute hydrocephalus after subarachnoid hemorrhage. Stroke 1989;20(6):715–7.
5. Komotar RJ, Olivi A, Rigamonti D, et al. Microsurgical fenestration of the lamina terminalis reduces the incidence of shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery 2002;51(6):1403–12 [discussion: 1412–3].
6. Kosteljanetz M. CSF dynamics in patients with subarachnoid and/or intraventricular hemorrhage. J Neurosurg 1984;60(5):940–6.
7. Torvik A, Bhatia R, Murthy VS. Transitory block of the arachnoid granulations following subarachnoid hemorrhage. A postmortem study. Acta Neurochir (Wien) 1978;41(1–3):137–46.
8. Komotar RJ, Hahn DK, Kim GH, et al. The impact of microsurgical fenestration of the lamina terminalis on shunt-dependent hydrocephalus and vasospasm after aneurysmal subarachnoid hemorrhage. Neurosurgery 2008;62(1):123–32 [discussion: 132–4].
9. Komotar RJ, Hahn DK, Kim GH, et al. Efficacy of lamina terminalis fenestration in reducing shunt-dependent hydrocephalus following aneurysmal subarachnoid hemorrhage: a systematic review. J Neurosurg 2009;111(1):147–54.
10. Greenberg M. Handbook of neurosurgery. 5th edition. New York (NY): Thieme Medical Publishers; 2001. p. 759.
11. McCormick P. Elevated intracranial pressure, ventricular drainage, and hydrocephalus after subarachnoid hemorrhage. Subarachnoid hemorrhage: pathophysiology and management. In: Neurosurgical Topics. Park Ridge (IL): AANS; 1997. p. 82.
12. Graff-Radford NR, Torner J, Adams HP Jr, et al. Factors associated with hydrocephalus after subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. Arch Neurol 1989;46(7):744–52.
13. MacDonald RL, Weir B. Perioperative management of subarachnoid hemorrhage. In: Youmans Neurological Surgery, vol. 2. 5th edition, 2004. p. 1823.
14. Vassilouthis J, Richardson AE. Ventricular dilatation and communicating hydrocephalus following spontaneous subarachnoid hemorrhage. J Neurosurg 1979;51(3):341–51.
15. Galera R, Greitz T. Hydrocephalus in the adult secondary to the rupture of intracranial arterial aneurysms. J Neurosurg 1970;32(6):634–41.
16. Penn RD, Belanger MG, Yasnoff WA. Ventricular volume in man computed from CAT scans. Ann Neurol 1978;3:216–23.
17. Zatz LM, Jernigan TL, Ahumada AJ. Changes on computed cranial tomography with aging: intracranial fluid volume. Am J Neuroradiol 1982;3: 1–11.
18. Earnest MP, Heaton RK, Wilkinson WE, et al. Cortical atrophy, ventricular enlargement and intellectual impairment in the aged. Neurology 1979;29(8): 1138–43.
19. Meese W, Kluge W, Grumme T, et al. CT evaluation of the CSF spaces of healthy persons. Neuroradiology 1980;19(3):131–6.
20. Van Gijn J, Hijdra A, Wijdicks EF, et al. Acute hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg 1985;63(3):355–62.
21. Vale FL, Bradley EL, Fisher WS. The relationship of subarachnoid hemorrhage and the need for postoperative shunting. J Neurosurg 1997;86:462–6.
22. Blasberg R, Johnson D, Fenstermacher J. Absorption resistance of cerebrospinal fluid after subarachnoid hemorrhage in the monkey; effects of heparin. Neurosurgery 1981;9:686–91.
23. O'Kelly CJ, Kulkarni AV, Austin PC, et al. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage: incidence, predictors, and revision rates. J Neurosurg 2009;111(5):1029–35.
24. de Oliveira JG, Beck J, Setzer M, et al. Risk of shunt-dependent hydrocephalus after occlusion of ruptured intracranial aneurysms by surgical clipping or endovascular coiling: a single-institution series and meta-analysis. Neurosurgery 2007;61(5): 924–33 [discussion: 933–4].
25. Raimondi AJ, Torres H. Acute hydrocephalus as a complication of subarachnoid hemorrhage. Surg Neurol 1973;1:23–6.
26. Scheithauer S, Bürgel U, Ryang YM, et al. Prospective surveillance of drain-associated meningitis/ventriculitis in a neurosurgery and a neurologic intensive care unit. J Neurol Neurosurg Psychiatr 2009;80(12):1381–5.
27. Voldby B, Enevoldsen EM. Intracranial pressure changes following aneurysmal rupture. J Neurosurg 1982;56:784–9.
28. Richardson AE, Jane JA, Payne PM. Assessment of the natural history of anterior communicating aneurysms. J Neurosurg 1964;21:266–74.
29. Adams HP Jr, Kassell NF, Torner JC. Early management of aneurysmal subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. J Neurosurg 1981;54:141–5.
30. Hellingman CA, van den Bergh WM, Beijer IS, et al. Risk of rebleeding after treatment of acute hydrocephalus in patients with aneurysmal subarachnoid hemorrhage. Stroke 2007;38(1):96–9.
31. Connolly ES Jr, Kader AA, Frazzini VI, et al. The safety of intraoperative lumbar subarachnoid drainage for acutely ruptured intracranial aneurysm: technical note. Surg Neurol 1997;48(4):338–42.

32. Auer LM, Mokry M. Disturbed cerebrospinal fluid circulation after subarachnoid hemorrhage and acute aneurysm surgery. Neurosurgery 1990;26(5):804–8.
33. Gruber A, Reinprecht A, Bavinzski G, et al. Chronic shunt-dependent hydrocephalus after early surgical and early endovascular treatment of ruptured intracranial aneurysms. Neurosurgery 1999;44(3):503–9.
34. Dorai Z, Hynan LS, Kopitnik TA, et al. Factors related to hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery 2003; 52(4):763–9.
35. Dehdashti AR, Rilliet B, Rufenacht DA, et al. Shunt-dependent hydrocephalus after rupture of intracranial aneurysms: a prospective study of the influence of treatment modality. J Neurosurg 2004;101(3): 402–7.
36. Varelas P, Helms A, Sinson G, et al. Clipping or coiling of ruptured cerebral aneurysms and shunt-dependent hydrocephalus. Neurocrit Care 2006;4(3):223–8.