Published in final edited form as:

Clin Neurol Neurosurg. 2014 October ; 0: 109–113. doi:10.1016/j.clineuro.2014.07.022.

Prediction of Ventriculoperitoneal Shunt Placement Based on Type of Failure During External Ventricular Drain Wean

Ariane Lewis, MD^a and W. Taylor Kimberly, MD, PhD^b

Division of Neurocritical Care and Emergency Neurology, Department of Neurology
Massachusetts General Hospital, Boston, MA

Abstract

Objective—There are multiple etiologies for failure while weaning an external ventricular drain (EVD) after subarachnoid hemorrhage (SAH), but there is little data on the relationship between etiology of wean failure and ventriculoperitoneal shunt (VPS) placement.

Methods—We performed a retrospective analysis of SAH patients who had an EVD placed between January 2008 and June 2012 at our institution. For each wean step (defined as raising or clamping the EVD), we recorded success or failure. We categorized failure as lowering or opening the EVD due to elevated intracranial pressure (ICP), clinical failure (due to headache or vomiting or altered mental status), leakage from the EVD site, or development of radiographic hydrocephalus. We evaluated the relationship between etiology of wean failure and subsequent need for VPS.

Results—Of 116 patients with an EVD placed, 35 required VPS placement (30%). Patients who required VPS placement had a median of 2 (interquartile range (IQR) 1-4) wean failures and those who did not require VPS placement had a median of 1 (IQR 0-1) wean failure ($p=0.001$). There was no significant relationship between age, sex, Hunt Hess score, Fisher score, Glasgow Coma Scale, aneurysm location, aneurysm size, aneurysm treatment method, vasospasm and need for VPS. There was a significant relationship between patients with at least one wean failure due to clinical changes or radiographic hydrocephalus and need for VPS ($p=0.007$ and $p=0.029$, respectively). After multivariate analysis, there was only a significant relationship between clinical changes and need for VPS (OR 2.76, CI 1.03-7.36, $p=0.04$).

Conclusion—There is a significant association between wean failure due to clinical changes

Keywords

extraventricular drain; hydrocephalus; subarachnoid hemorrhage; ventriculoperitoneal shunt

© 2014 Elsevier B.V. All rights reserved.

^a Ariane Lewis, MD 55 Fruit Street Boston, MA 02114 United States Ariane.kansas.lewis@gmail.com. ^b (corresponding author): W. Taylor Kimberly, MD, PhD Lunder 644 55 Fruit Street Boston, MA 02114 United States Tel 857-238-5644 Fax 857-238-5601 wtkimberly@partners.org.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

1. Introduction

7-65% of patients with subarachnoid hemorrhage (SAH) require external ventricular drain (EVD) placement [1,2] to facilitate cerebrospinal fluid (CSF) drainage to treat symptomatic hydrocephalus or to promote brain relaxation during aneurysm clipping [3,4].

The management of CSF drainage from an EVD varies. At some institutions, the EVD is kept clamped, to force CSF along normal pathways and prevent formation of occlusive membranes and clots, and is only opened if intracranial pressure (ICP) is greater than 20 mm Hg [4]. At others, the EVD remains open and is then clamped there is no evidence of vasospasm [5] or after day four or five if or after the CSF red blood cells (RBC) are less than 10,000 cells per cubic millimeter and there is no evidence of hydrocephalus, leakage from the EVD, or pseudomeningocele [6]. Many other institutions employ a progressive weaning strategy whereby the EVD is gradually raised over multiple days then ultimately clamped, a process that is extrapolated from evidence about chest tube and endotracheal tube management [3,7,8]. While at some institutions, patients have multiple EVD challenges [9], at others, failure of a single EVD clamp trial results in placement of a ventriculoperitoneal shunt (VPS) [6], and at still others there are radiologic [10] or clinical criteria [3,6] for VPS placement without a clamp trial.

8-63% of all SAH patients require permanent VPS placement [1,4,7,8,10-13]. Previous studies that evaluated predictors of VPS placement explored a wide variety of variables, including age [8,10-12,14,15], sex [8,10-12], presence of comorbidities [15], intubation status on admission, initial Glasgow Coma Scale (GCS) [12,14], initial Hunt Hess [6,8-11,16] and Fisher scores [5,10,11,14,16,17], admission glucose [17], aneurysm location [8,10-12] aneurysm size [8,12] treatment method (clip versus coil) [9,10,12,15,16], third ventricle diameter at admission [6], intraventricular hemorrhage (IVH) [2,5,8,10,11,16], vasospasm [8], admission CSF red blood cells (RBC) and protein [6,9], ventriculitis [11,17], and number of EVD days [9]. Of these, the most reproducible factors include age, sex, Hunt Hess and Fisher scores, and the existence of many others suggests that local practice may influence the decision to place a VPS.

The use of a stepwise weaning strategy at our institution provides the opportunity to assess whether types of EVD failure predict VPS placement and whether some types of failure do not necessitate a VPS. Clamp trials are frequently repeated with the goal of removing the EVD. Accordingly, our institution's VPS placement rate is lower than some reported rates. In this context, we hypothesized that there would be a relationship between failure etiology during an EVD wean and need for VPS placement. We also sought to identify low risk causes of failure that would not necessitate VPS placement.

2.1 Study Population

We used the Research Patient Data Registry to generate a list of all neurosurgery inpatients at our institution from 1/1/08 to 6/30/12 with the diagnosis of SAH (International Classification of Diseases (ICD) code 430). Through review of daily progress notes, we excluded patients whose primary diagnosis was not SAH, received greater than 48 hours of care elsewhere, had a history of SAH, had traumatic brain injury precipitating SAH, did not

have an EVD at any point during their hospitalization, or who had care withdrawn during the acute hospitalization.

2.2 EVD Management

At Massachusetts General Hospital (MGH), EVDs placed prior to securing an aneurysm are clamped at 20 centimeters to decrease risk of rebleeding [18] then opened and lowered once the aneurysm is secured to facilitate drainage of CSF. EVD wean is not initiated if there is evidence of active vasospasm. If there is no evidence of vasospasm, the EVD is slowly raised then clamped. If a patient develops clinical changes (headache, altered mental status, nausea/vomiting), ICP elevation above 20 mm Hg for five minutes, leakage from the EVD site, or worsening radiographic hydrocephalus after raising/clamping the EVD, it is lowered and/or unclamped. At the discretion of the treating neurosurgeon, there may be further attempts to wean the EVD with the goal of removing it after a successful clamp trial, but if a patient develops clinical changes or has elevated ICP after one or more clamp trials, a VPS is placed.

2.3 Data Ascertainment

We recorded age, sex, admission Hunt Hess/Fisher/GCS (interpreted through review of clinical and radiographic data if not stated in the notes), presence of an aneurysm felt to be responsible for the bleed, and aneurysm size (larger if there were two aneurysms), location (as reported from the formal angiogram or the computed tomography angiogram if no formal angiogram was done), treatment method (coil/clip/onyx), and presence of vasospasm (defined as peak velocities greater than 200 centimeters/second).

Using the data in procedure notes, we recorded the day of EVD placement relative to day of admission and location of EVD placement (operating room or emergency room/ICU). We reviewed progress notes to ascertain the number of EVD days, the EVD day the drain was first weaned (excluding clamping prior to securing the aneurysm), the number of EVD wean trials (defined as raising/clamping the EVD including clamping the EVD after initial placement), the number of EVD clamp trials prior to EVD removal or VPS placement, the CSF output prior to the final clamp trial, and the success/failure of each trial (failure defined as reopening or lowering the EVD, except when it was reopened/lowered after the aneurysm was secured) and the reason for failure. This study was approved by the local institutional review board.

2.4 Statistical Analysis

Statistical analysis was performed using JMP Pro 10. Categorical variables were recorded as percentages and evaluated using a Fisher's two-tailed exact test. Parametric continuous variables were recorded as mean $\pm$ standard deviation (SD) and evaluated using a Student's two-tailed t-test. Nonparametric continuous variables were recorded as median and interquartile range (IQR) and evaluated using a Wilcoxon ranked sum test. Multivariate analysis was performed using nominal logistic regression. A p-value of <0.05 was considered statistically significant.

3. Results

3.1 Patient Selection

Our query generated a list of 390 neurosurgical inpatients at MGH with the diagnosis of SAH between 1/1/08 to 6/30/12. Of these, 75 were eliminated because their primary diagnosis was not SAH (31 arteriovenous malformations/fistulas, 12 intraparenchymal hemorrhages, 11 ischemic strokes, 10 subdural hematomas, 3 elective aneurysm clippings, 3 cavernous malformations, 2 traumatic brain injuries, 1 epidural hematoma, 1 idiopathic intracranial hypertension, and 1 angiogram to check on a prior coil). Five patients with the diagnosis of SAH were eliminated because they received greater than 48 hours of care elsewhere and one patient with the diagnosis of SAH was eliminated because of a history of a prior SAH. Of the remaining 309 patients, 150 were excluded because they did not have EVDs and 43 patients were excluded because care was withdrawn. The final study population consisted of 116 patients.

3.2 Demographic Data

Table 1 shows the clinical characteristics of the study population. Thirty-five patients (30%) required VPS placement. There was no significant difference between patients with a VPS and those without a VPS and age, sex, Hunt Hess or Fisher scores, GCS, aneurysm location/size/treatment method, vasospasm, day of initial wean, or CSF output prior to final clamp trial. Patients who were shunted had an EVD for significantly longer than those who were not shunted ($p=0.004$). The number of wean trials was higher in the group that had a VPS placed (mean of 5 with SD 2 compared with mean of 4 with SD 2, $p=0.03$) as was the number of clamp trials (median of 2 with IQR of 1-3 compared with median of 1 with IQR of 1-1.5, $p=0.01$). Patients who were shunted had significantly more wean failures than those who were not shunted (median of 2 with IQR of 1-4 compared with median of 1 with IQR of 0-1, $p=0.001$).

3.3 External Ventricular Drain Analysis

There were 469 total weaning trials for the 116 patients (mean of 4 trials per patient with SD of 2) and 174 of these (37%) were failures (median of 1 failure per patient with IQR of 0-2) and resulted in lowering or opening the EVD. The most common cause of wean failure was ICP elevation (68.9%), with other factors comprising the remainder (Table 2). We evaluated the association of wean failure type with VPS placement. On univariate analysis, there was a significant association between clinical changes ($p=0.007$) or radiographic changes ($p=0.029$) and VPS requirement (see Figure 1). There was no association between wean failure due to elevated ICP or EVD leakage and need for VPS. After multivariate analysis, wean failure due to clinical changes was the only independent predictor of VPS (OR 2.76, CI 1.03-7.36, $p=0.04$) (see Table 3).

4. Discussion

While previous studies showed that 8-63% of patients required VPS placement after SAH [1,4,7,8,10-13], only 30% of our patients had a VPS placed. These studies have reported conflicting and inconsistent predictors for VPS placement. For example, some studies found

age [10] and sex [10-12] had no correlation with need for VPS, whereas others found associations between shunt placement and age [8,11,12,14,15] and female sex [8]. The relationship between aneurysm location [8,10-12], aneurysm size [8,12], and treatment method [8-10,12,15] and VPS requirement remains similarly controversial. While the variant findings may be due to limited sample sizes, an important factor may be the variation in EVD management [3-8] and decision making for VPS placement [3,6,9,10].

In this study, we looked at the relationship between etiology for wean failure and requirement for VPS in an institution where the goal is to minimize VPS placement. This provided an opportunity to also identify patients that may not need VPS, based on the type of wean failure. We found that the majority (68.9%) of wean failures were due to elevated ICP and 52% of patients had at least one failure due to elevated ICP. Despite this, there was no significant relationship between weaning failure due to elevated ICP ever and need for VPS, though there was a trend towards significance on univariate ($p=0.068$) and multivariate analysis ($p=0.07$). This type of failure may indicate temporary hydrocephalus due to accumulation of blood products that block CSF circulation [8,19], which resolves when the EVD is lowered/opened allowing blood products to be drained. We found that wean failure due to clinical change was the only independent predictor of VPS placement. While this type of failure may be representative of a more permanent type of hydrocephalus due to impaired arachnoid granulations [8], it is also possible that it may reflect our institution's threshold for VPS placement. Given the retrospective nature of our study, we cannot distinguish between these possibilities.

Other limitations of our study include the retrospective data collection from daily progress notes, which may have omitted additional wean trials or causes for wean failure. However, the standardized format of our progress notes likely reduces the chance of omission.

Furthermore, there would be no reason to suspect exclusion of EVD trials biased toward a specific type of wean failure etiology. The decision to repeat a clamp trial or place a VPS after a failed clamp trial was made by the treating neurosurgeon and was not standardized. However, patients who were shunted had significantly more wean and clamp trials, as the goal was to have a successful trial so that the EVD could be removed and VPS placement could be avoided. In a future prospective study, the number of failed clamp trials prior to VPS placement should be defined. In addition, the timing for the EVD wean was not standardized, but this was not statistically different between patients who were shunted and those who were not.

Conclusions

There is a significant association between wean failure due to clinical changes and requirement for VPS placement after SAH. Radiographic hydrocephalus alone is not a good predictor for the need for VPS placement. Future prospective studies are necessary to determine the optimum management of EVDs and patients most likely to benefit from shunt placement. VPS placement is associated with risk of obstruction, malpositioning, and infection [5,10,12], but untreated hydrocephalus after SAH is associated with delayed cognitive and motor recovery and transition to independent activities of daily living [20,21] so risks and benefits of VPS placement must be balanced for each patient.

Acknowledgments

This work was supported by the Andrew David Heitman Neurovascular Research Foundation (W.T.K.). The Foundation had no role in study design, collection, analysis, interpretation of data, writing of the manuscript, or decision to submit the manuscript for publication.

Abbreviations

CSF	cerebrospinal fluid
CT	computed tomography
EVD	external ventricular drain
GCS	Glasgow Coma Scale
ICD9	International Classification of Diseases 9
IQR	interquartile range
ICP	intracranial pressure
IVH	intraventricular hemorrhage
RBC	red blood cell
SD	standard deviation
SAH	subarachnoid hemorrhage
VPS	ventriculoperitoneal shunt

References

1. Hasan D, Vermeulen M, Wijdevicks EF, Hijdra A, van Gijn J. Management problems in acute hydrocephalus after subarachnoid hemorrhage. *Stroke*. 1989; 20:747–53. [PubMed: 2728040]
2. Wang YM, Lin YJ, Chuang MJ, Lee TH, Tsai NW, Cheng BC, et al. Predictors and outcomes of shunt-dependent hydrocephalus in patients with aneurysmal subarachnoid hemorrhage. *BMC Surgery*. 2012; 12:12–9. [PubMed: 22765765]
3. Vale F, Bradley E, Fisher W. The relationship of subarachnoid hemorrhage and the need for postoperative shunting. *J Neurosurg*. 1997; 86:462–6. [PubMed: 9046303]
4. Auer L, Mokry M. Disturbed cerebrospinal fluid circulation after subarachnoid hemorrhage and acute aneurysm surgery. *Neurosurgery*. 1990; 26(5):804–9. [PubMed: 2352599]
5. Kang DH, Park J, Park SH, Kim YS, Hwang SK, Hamm IS. Early ventriculoperitoneal shunt placement after severe aneurysmal subarachnoid hemorrhage: role of intraventricular hemorrhage and shunt function. *Neurosurgery*. 2010; 66(5):904–9. [PubMed: 20404694]
6. Chan M, Alaraj A, Calderon M. Prediction of ventriculoperitoneal shunt dependency in patients with aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 2009; 110:44–9. [PubMed: 18950263]
7. Klopfenstein JD, Kim LJ, Feiz-Erfan I, Hott JS, Goslar P, Zabramski JM, Spetzler RF. Comparison of rapid and gradual weaning from external ventricular drainage in patients with aneurysmal subarachnoid hemorrhage: a prospective randomized trial. *J Neurosurg*. 2004; 100:225–9. [PubMed: 15086228]
8. Dorai Z, Hynan L, Kopinik T, Samson D. Factors related to hydrocephalus after aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2003; 52(4):763–9. [PubMed: 12657171]
9. Esposito DP, Goldenberg F, Frank J, Ardeli A, Roitberg B. Permanent cerebrospinal fluid diversion in subarachnoid hemorrhage: influence of physician practice style. *Surg Neurol Int*. 2011; 2:117–22. [PubMed: 21918732]

10. Little A, Zabramski JM, Peterson M, Goslar PW, Wait SD, Albuquerque FC, et al. Ventriculoperitoneal shunting after aneurysmal subarachnoid hemorrhage: analysis of the indications, complications, and outcome with a focus on patients with borderline ventriculomegaly. *Neurosurgery*. 2008; 62(3):618–26. [PubMed: 18425009]
11. Kwon JH, Sung SK, Song YJ, Choi HJ, Huh JT, Kim HD. Predisposing factors related to shunt-dependent chronic hydrocephalus after aneurysmal subarachnoid hemorrhage. *J Korean Neurosurg Soc*. 2008; 43:177–81. [PubMed: 19096639]
12. O'Kelly CJ, Kulkarni AV, Austin PC, Urbach D, Wallace MC. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage: incidence, predictors, and revision rates. *J Neurosurg*. 2009; 111:1029–35. [PubMed: 19361256]
13. Brandner S, Xu Y, Schmidt C, Emtmann I, Buchfelder M, Kleindienst A. Shunt-dependent hydrocephalus following subarachnoid hemorrhage correlates with increased S100B levels in cerebrospinal fluid and serum. *Acta Neurochir Suppl*. 2012; 114:217–20. [PubMed: 22327696]
14. Yamada S, Nakase H, Park YS, Nishimura F, Nakagawa I. Discriminant analysis prediction of the need for ventriculoperitoneal shunt after subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis*. 2012; 21(6):493–7. [PubMed: 21236700]
15. Hoh B, Kleinhenz DT, Chi YY, Mocco J, Barker FG. Incidence of ventricular shunt placement for hydrocephalus with clipping versus coiling for ruptured and unruptured cerebral aneurysms in the nationwide inpatient sample database: 2002 to 2007. *World Neurosurg*. 2011; 76(6):548–54. [PubMed: 22251503]
16. Nam KH, Hamm IS, Kang DH, Park J, Kim YS. Risk of shunt dependent hydrocephalus after treatment of ruptured intracranial aneurysms: surgical clipping versus endovascular coiling according to fisher grading system. *J Korean Neurosurg Soc*. 2010; 48:313–8. [PubMed: 21113357]
17. Rincon F, Gordon E, Starke RM, Buitrago MM, Fernandez A, Schmidt JM, et al. Predictors of long-term shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 2010; 113(4):774–80. [PubMed: 20367072]
18. McIver J, Friedman J, Wijdicks E, Piegras D, Pichelmann M, Toussaint G, et al. Preoperative ventriculostomy and rebleeding after aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 2002; 97:1042–4. [PubMed: 12452133]
19. Blasberg R, Johnson D, Fenstermacher J. Absorption resistance of cerebrospinal fluid after subarachnoid hemorrhage in the monkey; effects of heparin. *Neurosurgery*. 1981; 9(6):686–91. [PubMed: 7322334]
20. Chen Z, Chen G, Song W, Liu L, Yang Y, Ling F. Rehabilitation combined with ventriculoperitoneal shunt for patients with chronic normal pressure hydrocephalus due to aneurysm subarachnoid hemorrhage: a preliminary study. *J Rehabil Med*. 2009; 41:1096–9. [PubMed: 19894008]
21. Chen Z, Song W, Du J, Li G, Yang Y, Ling F. Rehabilitation of patients with chronic normal-pressure hydrocephalus after aneurysmal subarachnoid hemorrhage benefits from ventriculoperitoneal shunt. *Top Stroke Rehabil*. 2009; 16(5):330–8. [PubMed: 19903651]

Figure 1.
 Wean Failure Etiology By Ventriculoperitoneal Shunt (VPS) Placement

Table 1**Demographics**

Demographic Data:	NO VPS (n=81)	VPS (n=35)	P-Value
Age, mean (SD)	55 (13)	58 (14)	0.18
Female, n (%)	55 (68%)	27 (77%)	0.37
Hunt Hess 3/4/5	37 (46%)	19 (54%)	0.42
Fisher 3/4	73 (90%)	31 (89%)	0.75
Glasgow Coma Scale <9	13 (16%)	9 (26%)	0.30
Glasgow Coma Scale <15	45 (56%)	19 (54%)	1.00
Treated Aneurysm, n (%)	71 (88%)	32 (91%)	0.75
Clip, n (%)	40 (56%)	14 (44%)	
Coil, n (%)	31 (44%)*	18 (56%)	0.29
Aneurysm Size in mm, median [IQR]	5 [4, 7]	6 [4, 8]	0.30
Posterior Aneurysm, n (%)	15 (19%)	6 (17%)	1.00
Vasospasm, n (%)	40 (50%)	20 (59%)	0.42
Hospital Day EVD Placed, median [IQR]	0 [0, 1]	0 [0, 1]	0.30
EVD Placed in ICU/Emergency Room, n (%)	63 (78%)	30 (86%)	0.45
EVD Days, mean (SD)	12 (4)	15 (5)	0.004
EVD Day of Initial Wean, median [IQR]	6.5 [4, 10]	6 [5, 10]	0.49
CSF Output (ml) Before Final Clamp Trial, median [IQR]**	135 [81, 173]	153 [92, 226]	0.10
Wean Trials ^a , mean (SD)	4 (2)	5 (2)	0.03
Clamp Trials ^b , median [IQR]	1 [1, 1.5]	2 [1, 3]	0.01
Wean Failures, median [IQR]	1 [0-1]	2 [1-4]	0.001

SD=standard deviation; IQR=interquartile range; EVD= external ventricular drain; ICU=intensive care unit; ml=milliliter

* includes one patient who was treated endovascularly with onyx

** CSF output data not available for all patients (n=71 for no VPS, n=27 for VPS)

^a number of attempts to clamp or raise the EVD at any time

^b number of attempts to clamp the EVD prior to EVD removal or VPS placement (excludes attempts to clamp the EVD prior to securing the aneurysm)

Table 2

Etiology of Wean Failure

Etiology	N (%)
Clinical Failure	32 (18.4%)
ICP Failure	120 (68.9%)
EVD Leakage Failure	9 (5.2%)
Radiographic Failure	4 (2.3%)
Combination of Factors	9 (5.2%)

ICP= intracranial pressure, EVD= external ventricular drain

Table 3

Multivariate Analysis of Wean Failure Etiology

	OR (95% CI)	P-Value
Clinical Failure Ever	2.76 (1.03-7.36)	0.04
ICP Failure Ever	2.27 (0.953-5.67)	0.07
Radiographic Failure Ever	7.65 (0.891-167)	0.10

ICP= intracranial pressure