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Acute Operative Intervention for Intracranial Hemorrhage in Adult Patients with Ventricular Assist Device Therapy Associated with Fatal Outcomes

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Abstract

Objective: Ventricular assist devices (VADs) are utilized more frequently in the management of patients with advanced heart failure due to benefits in survival and quality of life. Intracranial hemorrhagic injuries (HI) are a significant source of morbidity and mortality in VAD patients. The aim of this investigation was to describe prognostic variables in VAD patients with HI, describe the outcomes after neurosurgical intervention, and find indications for surgery.

Methods: The records of 160 patients who underwent VAD implantation from 2007 to 2011 were retrospectively analyzed. Data was abstracted to identify risk factors for the development of HI. Premorbid patient data and demographics were considered. Risk factors for increased mortality were investigated within the HI group. Detailed investigations were performed on patients undergoing operative intervention.

Results: Twenty-three HIs were identified in 23 of the 156 VAD patients. A prior history of stroke and increased length of VAD therapy were found to be risks for HI. The 30-day mortality after HI was 69.6%. A presenting GCS < 13, hemorrhage > 30mL, and increasing cerebral midline shift were significant risk factors for increased 30-day mortality. Six of 7 patients undergoing surgery died within the immediate post-operative period. The lone surviving patient underwent delayed operative intervention 11 days after HI.

Conclusion: Despite improvements in morbidity with VAD implantation and survival after implant, HI remains a significant source of morbidity. This study has identified several potential risks for the development of HI and for increased mortality after HI. Acute operative interventions for HI were found to have limited success.

Keywords: Heart failure, Intracranial hemorrhage, Left ventricular assist device, Mortality, Neurosurgery, Vascular disorders

Abbreviations

CA: Cerebral Autoregulation, CI: Confidence Interval, GCS: Glasgow Coma Score, HF: Heart Failure, HI: Hemorrhagic Injury, ICH: Intra Cerebral Hemorrhage, INR: International Normalized Ration, IVH: Intra Ventricular Hemorrhage, MAP: Mean Arterial Pressure, SAH: Subarachnoid Hemorrhage, SD: Standard Deviation, SDH: Subdural Hematoma, VAD: Ventricular Assist Device

Introduction

Heart failure (HF) affects more than 6.6 million Americans with a 3% prevalence estimated in adults by 2015 [1,2]. In general patients with heart failure have a poor prognosis and while cardiac transplantation is an effective long-term therapy for a select group of patients, the number of transplants have plateaued [3]. Compared with optimal medical management, ventricular assist device (VAD) therapy significantly improves survival and quality of life [4]. Due to the severe organ shortage and marginal improvements in outcomes with medical management, alternate therapies such as VAD have developed.

One of the most frequent adverse events of VAD therapy includes intracranial hemorrhage injury (HI) with as many as 11% affected in large, multicenter trials [5-9]. Systemic anticoagulation and antiplatelet therapies are used in the majority of VAD patients to prevent ischemic complications, but predispose them to increased risk of spontaneous HIand HI after mild trauma. Other suggested risk factors for spontaneous HI in VAD patients include elevated mean arterial pressure (MAP), higher VAD speeds, and blood stream infections [10,11]. The reported mortality after such injuries has been reported to range from 33-100% [11,12].

Balancing the risk of ischemic complications by withholding anticoagulation and antiplatelet therapy, while treating a patient with HI can be challenging. A recent publication demonstrated success in the medical management of HI in patients with VADs by withholding antiplatelet therapy for one week and anticoagulation for 10 days without patients suffering from expansion of the hemorrhagic region, ischemic stroke, or pump thrombosis [12]. There is minimal information regarding the acute surgical management of HI in patients with VAD, as experience has been limited to case reports and a small case series of two patients [13-16].

The aim of this single-institution investigation was to examine the risk factors for the development of HI in patients undergoing VAD therapy and to identify risk factors for increased mortality.

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Additionally, patients who underwent operative intervention for treatment of an HI while on VAD therapy were described in greater detail. The authors hope this information will help physicians in the management of these critically ill patients.

Methods

Study design

The authors performed a retrospective review of a prospectively maintained database of adult patients who underwent VAD placement from 2007 to 2011. The study was approved by the Wexner Medical Center of The Ohio State University's Institutional Review Board. Demographic data, implant date, pump type and outcomes were collected for all 160 patients.

A detailed chart review was performed to evaluate patients who suffered a HI during VAD therapy. Data abstracted included sex, race, mortality, age at implantation of VAD therapy, type of implant, date of implantation, date of death or implant removal, last follow-up date, premorbid medical history, antiplatelet therapy, anticoagulation therapy, and occurrence of HI. One diagnosis of HI was made based on records from an outside hospital. Four patients were lost to follow-up. None of the four excluded patients suffered a HI at last follow-up.

Specific data for HI included type of hemorrhage (subarachnoid hemorrhage (SAH), subdural hematoma (SDH), and intracerebral hemorrhage (ICH)), date of the hemorrhage, presenting Glasgow Coma Score (GCS), presenting international normalized ratio (INR), need for neurosurgical intervention, and type of neurosurgical intervention and outcome. Data abstracted about HI patients were used to identify risks of increased 30-day mortality. Midline shift was measured in patients with ICH and SDH. If a patient had an ICH, an ICH score was tabulated from presenting GCS, age, infratentorial or supratentorial location, volume, and presence or absence of intraventricular hemorrhage (IVH). Multiplying the length by width by height and dividing by 2 on CT of the head calculated the volume of hemorrhage. The length and width of the hemorrhage were measured on the axial CT slice where the hemorrhage was largest, and height was determined by counting the number of CT slices containing hemorrhage and multiplying by slice thickness. The ICH score is an externally validated prognostic clinical scale with an increasing score predicting greater mortality [17,18].

Statistical analysis

Statistical analysis was performed with commercially available software (SPSS version 22.0, IBM Corp). All categorical data were reported as number and percentage and analyzed using chi-square (χ^2). Univariate analyses of continuous data were reported as mean ± standard deviation (SD) and assessed with t-tests. For all analyses, $p < 0.05$ was considered significant. Confidence intervals (CI) are reported as 95% confidence limits.

Results

Patient characteristics

During the 5-year study period, 160 patients underwent VAD placement. One hundred and fifty-six were included for analysis as four patients had incomplete follow-up. Twenty-three HIs were found in 23 patients during the study period. Table 1 outlines the baseline characteristics of the HI patients compared to all other VAD patients. There was no significant difference in age, sex, race, or pump type (continuous versus pulsatile) between HI and no intracranial hemorrhagic injury (NHI) groups. The Heart Mate II VAD (Thoratec, Pleasonton, CA, USA) was used in 122 (78.2%) of 156 patients. Females were found more frequently in the HI group (34.8%) then the NHI group (19.5%), but, again, this was not significant (p=0.102). Similarly, premorbid diabetes mellitus, hypertension, dyslipidemia, and smoking status were not statistically significant. Prior stoke was found to be significantly more common in post implant HI patients (39.1%) as compared to the NHI group(19.1%; p=0.010).

Table 1: Baseline demographics of patients with no hemorrhagic injury (NHI) and those with hemorrhagic injury (HI).

Factor	NHI n=133 (%)	HI n=23 (%)	p-value	Test
Demographics				
Age at implant (years)[Ŧ]	53.7 ± 2.4	49.7 ± 6.4	0.198	t-test
Sex (male)	107 (80.5)	15 (65.2)	0.102	χ^2
Ethnicity				
Caucasian	103 (77.4)	19 (82.6)	0.810	χ^2
African American	29 (21.8)	4 (17.4)		
Asian	1 (0.8)	0 (0)		
Pump Type				
Continuous	116 (87.2)	20 (87.0)	0.972	χ^2
Pulsatile	17 (12.8)	3 (13.0)		
Premorbid medical history				
Stroke	21 (15.8)	9 (39.1)	0.009	χ^2
Diabetes Mellitus	56 (42.1)	8 (34.8)	0.510	χ^2
Hypertension	97 (72.9)	17 (73.9)	0.922	χ^2
Dyslipidemia	95 (71.4)	12 (52.2)	0.066	χ^2
Premorbid or current tobacco	88 (66.2)	12 (52.2)	0.197	χ^2
Antiplatelet and anticoagulation				
Aspirin	107 (80.5)	18 (78.3)	0.887	χ^2
Clopidogrel	11 (8.3)	3 (13.0)	0.460	χ^2
Anticoagulation	100 (75.2)	20 (87.0)	0.216	χ^2
Duration of VAD support				
Mean (days)[Ŧ]	699.3 ± 100.5	898.0 ± 245.7	0.134	t-test
>2 years	54 (40.6)	15 (65.2)	0.021	χ^2
All cause mortality	66 (49.6)	21 (91.3)	< 0.001	χ^2

Ŧ No percentage was applicable for factors evaluated by t-test.

Antiplatelet therapy did not vary significantly between the two groups for aspirin (p=0.887) or aspirin and clopidogrel (Plavix, Bristol-Myers Squibb, New York City, NY, USA; p = 0.460). Only one patient was on clopidogrel monotherapy. More patients in the HI group (87%) were systemically anticoagulated with heparin or warfarin then in the NHI group (75.2%), but this was not significantly different (p=0.216).

The duration of device therapy (days) was not significantly different between the HI group (898 ± 245.7) and the NHI group (699.3 ± 100.5; p=0.134). However, patients with HI were more frequently found to have had VAD therapy for at least two years (65.2%) when compared to the NHI group (39.4%; p=0.021). The NHI group had 30 patients in whom VAD therapy was terminated prior to latest follow-up or death. Two patients underwent device explanation, and 28 patients underwent cardiac transplantation. No patient with HI went on to undergo cardiac transplantation. The mean duration of VAD therapy for these 30 patients was 355.9 ± 104.0 days (95% CI). All because mortality at latest follow-up was found to be different with 8.7% of HI patients alive compared to their NHI counterparts (50.4%; $p < 0.001$). The total follow-up time for all patients was 311.4 years or approximately 2.0 years per patient.

Intracranial hemorrhagic injuries

Twenty-three HIs were identified in 23 (14.7%) of 156 patients during the study period. One HI was found for every 4942 days of VAD therapy for a 7.4% event rate per patient year. The mean time (days) to HI after VAD implant was 671.7 ± 228.2 (95% CI=443.4-899.9). The mean age (years) of HI patients at the time of their injury was 49.7 ± 6.4 (95% CI=43.3-56.0). Age was not found to be a risk for increased mortality after HI (p=0.986). Table 2 summarizes the data abstracted to identify risks for increased 30-day mortality. Sixteen of 23 (69.6%) HI patients died within 30 days. Patients who underwent a neurosurgical intervention for the hemorrhagic complication were not found to have a statistically significant increase in mortality (p=0.266).

Although HI patients suffering an ICH were found to have the highest 30-day mortality (80%) among all hemorrhagic complications, there was no statistically significant difference between subgroups (p=0.241). The 3 patients with SDHs greater than 1cm in their largest

Table 2: Risk factors for increased 30-day mortality after hemorrhagic injury (HI).

Factor	All N (%)	30-day mortality n (%)	p-value	Test
Hemorrhagic injury patients	23 (100)	16 (69.6)		
Sex				
Male	15 (65.2)	11 (73.3)	0.591	χ^2
Female	8 (34.8)	5 (62.5)		
Age (>50 years old)	13 (56.5)	9 (69.2)	0.968	χ^2
Type of Hemorrhage				
ICH	15 (65.2)	12 (80.0)	0.241	χ^2
SAH	3 (13.1)	1 (33.3)		
SDH	5 (21.7)	3 (60.0)		
Presenting GCS (≥13)				
Yes	11 (47.8)	4 (36.4)	<0.001	χ^2
No	12 (52.2)	12 (100)		
Presenting INR (>2.5)				
Yes	13 (56.5)	10 (76.9)	0.382	χ^2
No	10 (43.5)	6 (60)		
Traumatic versus Spontaneous HS				
Spontaneous	18 (78.3)	14 (77.8)	0.104	χ^2
Traumatic	5 (21.7)	2 (40.0)		
Neurosurgical intervention				
Yes	7 (30.4)	6 (85.7)	0.266	χ^2
No	16 (69.6)	10 (62.5)		
ICH Volume (n=15)				
>30 mL	11 (73.3)	11 (100)	0.001	χ^2
<30 mL	4 (26.7)	1 (25.0)		
ICH Score (Mean; 95% confidence)[Ŧ]	2.3 ± 0.8	2.8 ± 0.5	0.003	t-test

Ŧ No percentage was applicable for factors evaluated by t-test.

dimension died within 30 days, whereas the 2 patients with smaller SDHs survived (p=0.025). Midline shift (mm) was found to be significantly different in 30-day mortality between those that lived (0.8 ± 2.2) and died (11.1 ± 4.8) in ICH and SDH patients (p=0.012). The average midline shift was 7 and 11 mm in the ICH and SDH groups, respectively. The one SAH patient that died suffered from severe systemic disease and died from non-neurological complications.

Spontaneous HI was found to be more lethal (77.8%) then traumatic injuries (40.0%), although this was not found to be significant (p=0.104). HI patients with a GCS of 12 or less at the time of diagnosis were found to have 100% 30-day mortality, whereas 36.4% of those with a GCS of 13 or greater died within 30 days (p<0.001). Although there was a trend toward a higher presenting INR at the time of HI diagnosis in patients who died (3.4 ± 2.1) compared to those who lived (2.3 ± 0.9), this was not significant (p=0.498).

Subgroup analysis was performed in HI patients suffering from ICH (n=15). Hemorrhages found to be larger than 30mL were found to have a 100% 30-day mortality rate, while only 1 of 4 patients with smaller hemorrhages died (p=0.001). The average presenting ICH score of patients dead at 30 days was 2.8 ± 0.5, with all of those surviving the hemorrhage had a score of 0 (p<0.001).

Neurosurgical interventions

Seven patients (3 women, 4 men) of the total 23 HIs underwent operative intervention with 6 (85.7%) of these patients dying within 30 days. This was not significantly different from the HIs that were not intervened upon surgically (62.5%; p=0.266). Patients who underwent intervention tended to be younger (42.3 ± 12.8 years) compared to their nonsurgical counterparts (52.9 ± 7.6 years), although this was not significant statistically (p=0.113). The average presenting GCS was 7.4 ± 4.3 (95% CI=3.10-11.76). The average midline shift for the 3 SDH patients operated on was 13 mm. The average midline shift for the 4 ICH patients operated on was 11.5mm. In all but one case, operative intervention was performed within 24 hours of presentation. The patient (case 5) who underwent delayed operative intervention (burr hole drainage 11 days after presentation) is the only patient alive at 30 days and remains at latest follow-up. Table 3 summarizes the characteristics of the 7 patients whom underwent operative intervention.

In total, 4 craniectomies, 2 craniotomies, and 1 burr hole drainage were performed. Five patients were in the hospital at the time of the development of the HI and had spontaneous mental status decline (4 ICHs, 1 SDH). Two patients with SDHs presented when emergency services brought them to the hospital after a fall. Five of the seven patients were being managed with warfarin at the time of their HI and were found to have a mean INR of 2.94±1.28 (95% CI=1.66-4.22), while the other 2 patients were systemically anticoagulated with heparin at the time of injury. All patients' anticoagulation was reversed with fresh frozen plasma (FFP), Factor IX Complex (Profilnine® SD, Grifols International, Barcelona, Spain), protamine, vitamin k, or a combination of these. The average INR obtained after attempted correction was 1.3 ± 0.1 (95% CI=1.2-1.4). Three of 7 patients were given donor platelets perioperatively.

Failure to improve or progression to a moribund neurological examination after surgery was the cause of death in 5 of 6 patients. In case 1, despite decompression of the posterior fossa from an enlarging cerebellar hematoma, follow-up CT of the head showed progressive bilateral, hemispheric ICH and her neurological exam continued to deteriorate. The family withdrew care and the patient expired. In case 2, the patient became hemodynamically unstable immediately after surgery, and the patient died from cardiopulmonary arrest despite aggressive attempts at resuscitation. In cases 3, 4, 6, and 7, the patients underwent surgery for large HI and moribund exams. Their exams showed no sign of improvement postoperatively, and the patients' families elected to withdraw care. In case 5, the patient's stable neurological exam allowed for a delayed intervention 11 days after initial presentation. Excluding the lone surviving patient, the average survival for HI patients who underwent neurosurgical intervention was 3.3 days.

Discussion

In 2001, the results of REMATCH (Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure) study demonstrated significant benefits of VAD therapy in survival and quality of life in patients with advanced HF when compared to optical medical management [4]. However, with limited cardiac donors and increasing number of patients receiving the VAD for destination therapy, HI will continue to remain prevalent within this population. Since cerebrovascular injury, including HI and ischemic stroke, is a significant source of morbidity and mortality in such patients, it is necessary to investigate outcomes and potential causes of such injuries [10,12,19]. There is limited information from case reports and a small case series documenting successful surgical intervention in managing HI with mechanical circulatory support, including VAD [13-16]. The aim of this retrospective, cohort study was to identify patients that may benefit from operative intervention and identify risk factors for increased mortality.

In the present study, the HI group was found to have a longer average length of VAD therapy, and therapy greater than two years was more frequently found in the HI group. This would only be intuitive as the risk of hemorrhagic injury is likely cumulatively increased with longer time on VAD therapy. Age was not found to be a predictive risk of the development of HI, nor was it found to influence survival after such injuries. Although not statistically different, more women were found in the HI group. This trend was also found in the only other dedicated publications of HI in VAD patients [12]. Premorbid stroke was significantly increased in HI group, which is not unexpected as patients with prior stroke are at greater risk for suffering future spontaneous HI [20,21]. Pump type, continuous or pulsatile, was not found be different between groups, which has been previously found, although this could be owed to our

Table 3: Characteristics of patients undergoing operative intervention.

Case No.	Age	Sex	Type of Injury	Presenting GCS	Time from Implant (days)	Neurosurgical Intervention	Outcome	Survival after injury (days)
1	23	F	Bilateral cortical ICHs (>30 cm^3), right cerebellar hemorrhage (<30 cm^3), and IVH	13	1322	Craniotomy, evacuation of cerebellar hematoma, and EVD placement	The patient had continued evolution of ICHs and elevated ICP; Family withdrew care on POD 3	4
2	62	M	Right spontaneous SDH (20mm) with 12 mm of midline shift	11	172	Craniotomy and SDH evacuation	The patient decompensated hemodynamically after surgery and was unable to be resuscitated	1
3	30	F	Left frontal ICH (>30 cm^3) with 11 mm of midline shift	3	1072	Craniectomy and ICH evacuation	The patient continued to develop remote ICH and ischemic injury; her neurological exam was moribund; care was withdrawn on POD 2	3
4	52	F	Right frontal ICH (>30 cm^3) with 8 mm midline shift and IVH	4	11	Craniectomy, ICH evacuation, and EVD placement	The patient's neurological exam never improved, and she had considerable comorbidities including respiratory failure and acute renal failure; care was withdrawn	2
5	42	M	Left SDH (9mm) after fall with 4mm of midline shift	13	138	Burr hole evacuationof SDH 11 days after presentation	The patient tolerated the procedure well, had an uneventful post operative course, and was discharged home	>798
6	52	M	Left SDH (15 mm) with IVH and 23 mm of midline shift after fall	5	37	Craniectomy and SDH evacuation	The patient remained moribund on exam and the family decided to withdraw care	5
7	35	M	Right frontal ICH (>30 cm3) with 20 mm of midline shift	3	1	Craniectomy, evacuation of hematoma, and EVD placement	The patient progressed to brain death on POD 2	2

relatively small cohort size and the disproportionate use of the Heart Mate II VAD [9]. As expected, survival was significantly decreased in patients in the HI group.

Historically HI has been found to affect 2-11% of subjects in larger VAD therapy cohorts [5-9]. Lazar et al. [19] evaluated the risk of neurological injury in the original REMATCH study population with 16% of VAD patients suffering a stroke, although only 2 patients (3.3%) suffered HI [19]. In Wilson et al. [12] publication on the management of HI in VAD patients, they found an 11% risk of injury in their cohort. Although the absolute rate of injury is higher in the present study (14.7%) then what has been previously reported, the rate of HI for days of therapy (one HI per 4942 days) is comparable or lower than prior studies [9,12].

A lower presenting GCS, larger hemorrhage size, and increasing midline shift were associated with poorer outcomes. In Wilson et al. [12] study, the authors found similar results with GCS, but hemorrhage size and midline shift were not significantly different in predicting survival [12]. This could be explained, in part, by our cohort's comparatively increased average, midline shift in ICH and SDH patients. The subgroup analysis done on ICH patients demonstrates 100% mortality for any patient without a small supratentorial hemorrhage and with any other associated risks such as older age, IVH, or altered GCS upon presentation. The fact that any ICH score above 0 portends poor survival is further evidence that VAD patients have limited capacity to tolerate neurological injuries compared to their non-VAD, historical cohorts [17,18].

The uniformly poor survivability of ICH in the VAD population with or without surgery may be associated with a failure of the physiologic response and decoupling of normal cerebral autoregulation (CA). In 2013, Gould, et al. [22] demonstrated stable cerebral blood flow on serial CT perfusion studies in the presence of ICH despite systemic changes in blood pressure, indicating preservation of CA and a potential protective mechanism during HI [22]. The relatively fixed cardiac flow in these patients coupled with the difficulty in measuring and managing systemic MAP may predispose these patients to spontaneous ICH. In 2009, Lietz et al. [23] published evidence of impaired CA associated with cerebral hyperperfusion in the immediate post-implant period of VAD patients associated with neurological dysfunction [23]. Although initially described for the immediate post-implant period, this same mechanism may result in a failure of CA after HI.

As stated, there is limited, published information regarding the surgical management of VAD patients. Prior publications have demonstrated successful surgical management of such patients, but have largely been limited to case reports. In two pediatric publications, 3 patients were successfully operated on for spontaneous SDH with all children surviving at least 286 days [13,15]. These successes may not necessarily be referred to the adult population, as the systemic premorbid condition in children may not be as severe as adults with long-standing advanced HF. In two case reports of adults with VAD therapy successfully operated on for ICH (one spontaneous, one mycotic), the authors report the patients were able to undergo delayed (greater than 24 hours) surgery [14,16]. Similar to our lone surviving patient (case 5), delayed operative intervention would suggest the initial injury is not severe enough to cause a significant neurological decline and allows for adequate time to reverse anticoagulation and wait for the effects of antiplatelet therapy to be diminished.

In the present study, acute surgical intervention (less than 24 hours) was found be associated with 100% mortality and an average survival of 3.3 days. Operative intervention for ICH was associated with poor results and no clinical evidence of patient improvement. Wilson et al. [12] found similar results for operative intervention of ICH as 4 of 5 of their patients died within short follow-up and the lone survivor severely debilitated [12]. Unfortunately, our results would reinforce the suggestion that acute operative intervention for HI is met with dismal results. With the limited results, the authors suggest that only patients with extra-axial hematomas or patients presenting with only moderate neurological dysfunction allowing for a delayed operative intervention should be considered operative candidates with an anticipated good survival or functional outcome.

Limitations

There are several limitations to the study. First, the retrospective nature of this cohort study has limited the authors' ability to collect data. For example, data regarding pump settings and alterations in those settings is limited as the study period includes an institutional change from paper charting to electronic medical record. Progress notes were largely used to capture data regarding neurological

condition such as GCS and confounding factors such as sedating or paralyzing medications could not be controlled for in all cases.

Second, no standardization of medical or surgical practice was established prior to the data collection for these patients. Treatment of antiplatelet and anticoagulation therapies varied between providers. Similarly, surgical practice and pre- and perioperative medical care was performed by several different surgeons and anesthesiologists as all but 1 of these cases was performed under emergent circumstances.

Finally, the cohort remains relatively small with only 156 patients completing last follow-up, 23 having HI, and only 7 patients undergoing operative intervention. Definitive conclusions are impossible to be demonstrated, although the evidence within this small cohort would suggest prospective data collection is warranted to delineate the operative role for these critically ill patients.

Conclusion

In this single-institution, retrospective cohort study, the authors found evidence that reaffirms HI as a devastating event in patients with VAD therapy. History of prior stroke, female sex, and longer duration of VAD therapy may be risk factors for the development of HI. A GCS less than 13 and larger hemorrhage portends a poor prognosis for these patients. The premorbid medical conditions along with intrinsic failures of CA in patients with VAD may contribute to poor survival, but further investigation is necessary to identify this as a true risk of VAD therapy. In this study, acute operative intervention was met with 100% mortality. Extra-axial hematoma such as SDH and patients with relatively good neurological function at presentation may be the only patients expected to have good surgical outcome. It is not the authors' intention to suggest withholding a potential life-saving measure from these critically ill patients, but expectations of poor outcomes must be understood with physicians and for the patients' families prior to an emergent operative intervention is undertaken.

References

1. Heidenreich PA, Trogdon JG, Khavjou OA, Butler J, Dracup K, et al. (2011) Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. Circulation 123: 933-944.
2. Yancy CW, Jessup M, Bozkurt B, Masoudi FA, Butler J, et al. (2013) 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 62: 147-239.
3. Long JW, Kfoury AG, Slaughter MS, Silver M, Milano C, et al. (2005) Long-term destination therapy with the HeartMate XVE left ventricular assist device: improved outcomes since the REMATCH study. Congest Heart Fail 11: 133-138.
4. Rose EA, Gelijns AC, Moskowitz AJ, Heitjan DF, Stevenson LW, et al. (2001) Long-term use of a left ventricular assist device for end-stage heart failure. N Engl J Med 345: 1435-1443.
5. Aaronson KD, Slaughter MS, Miller LW, McGee EC, Cotts WG, et al. (2012) Use of an intrapericardial, continuous-flow, centrifugal pump in patients awaiting heart transplantation. Circulation 125: 3191-3200.
6. John R, Naka Y, Smedira NG, Starling R, Jorde U, et al. (2011) Continuous flow left ventricular assist device outcomes in commercial use compared with the prior clinical trial. Ann Thorac Surg 92: 1406-1413.
7. Pagani FD, Miller LW, Russell SD, Aaronson KD, John R, et al. (2009) Extended mechanical circulatory support with a continuous-flow rotary left ventricular assist device. J Am Coll Cardiol 54: 312-321.
8. Park SJ, Milano CA, Tatooles AJ, Rogers JG, Adamson RM, et al. (2012) Outcomes in advanced heart failure patients with left ventricular assist devices for destination therapy. Circ Heart Fail 5: 241-248.
9. Slaughter MS, Rogers JG, Milano CA, Russell SD, Conte JV, et al. (2009) Advanced heart failure treated with continuous-flow left ventricular assist device. N Engl J Med 361: 2241-2251.
10. Adzic A, Patel SR, Maybaum S (2013) Impact of adverse events on ventricular assist device outcomes. Curr Heart Fail Rep 10: 89-100.
11. Aggarwal A, Gupta A, Kumar S, Baumblatt JA, Pauwaa S, et al. (2012) Are blood stream infections associated with an increased risk of hemorrhagic stroke in patients with a left ventricular assist device?. ASAIO J 58: 509-513.
12. Wilson TJ, Stetler WR Jr, Al-Holou WN, Sullivan SE, Fletcher JJ (2013) Management of intracranial hemorrhage in patients with left ventricular assist devices. J Neurosurg 118: 1063-1068.
13. Haque R, Wojtasiewicz T, Gerrah R, Gilmore L, Saiki Y, et al. (2012) Management of intracranial hemorrhage in a child with a left ventricular assist device. Pediatr Transplant 16: E135-139.
14. Hoy FB, Clemson BS, Geiss DM, Bond LM, Linett CE, et al. (2001) Intracranial bleed during bridge to transplant may not preclude a successful result. J Heart Lung Transplant 20: 901-903.
15. Mayer RR, Hwang SW, Reddy GD, Morales DL, Whitehead WE, et al. (2012) Neurosurgical complications of left ventricular assist devices in children. J Neurosurg Pediatr 10: 370-375.
16. Morito H, Nishimura T, Ando M, Kinoshita O, Hisagi M, et al. (2012) Successful treatment of cerebral hemorrhage using computed tomography angiography in a patient with left-ventricular-assist device. J Artif Organs 15: 90-93.
17. Clarke JL, Johnston SC, Farrant M, Bernstein R, Tong D, et al. (2004) External validation of the ICH score. Neurocrit Care 1: 53-60.
18. Hemphill JC 3rd, Bonovich DC, Besmertis L, Manley GT, Johnston SC (2001) The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. Stroke 32: 891-897.
19. Lazar RM, Shapiro PA, Jaski BE, Parides MK, Bourge RC, et al. (2004) Neurological events during long-term mechanical circulatory support for heart failure: the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) experience. Circulation 109: 2423–2427.
20. Charidimou A, Baron JC, Werring DJ (2013) Transient focal neurological episodes, cerebral amyloid angiopathy, and intracerebral hemorrhage risk: looking beyond TIAs. Int J Stroke 8: 105-108.
21. Hankey GJ, Jamrozik K, Broadhurst RJ, Forbes S, Burvill PW, et al. (1998) Long-term risk of first recurrent stroke in the Perth Community Stroke Study. Stroke 29: 2491-2500.
22. Gould B, McCourt R, Asdaghi N, Dowlatshahi D, Jeerakathil T, et al. (2013) Autoregulation of cerebral blood flow is preserved in primary intracerebral hemorrhage. Stroke 44: 1726-1728.
23. Lietz K, Brown K, Ali SS, Colvin-Adams M, Boyle AJ, et al. (2009) The role of cerebral hyperperfusion in postoperative neurologic dysfunction after left ventricular assist device implantation for end-stage heart failure. J Thorac Cardiovasc Surg 137: 1012-1019.