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Hunt-Hess 5 Subarachnoid Haemorrhage Presenting with Cardiac Arrest is Associated with Larger Volume Bleeds

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Abstract

Aims—The mechanism, effects, and outcomes of cardiac arrest (CA) caused by subarachnoid haemorrhage (SAH) remain unclear. We compared SAH patients presenting with CA to other high-grade SAH patients presenting without CA in order to better understand (1) the cause of CA, (2) cerebral pathophysiology following CA, and (3) outcomes of CA in patients with SAH.

Methods—We performed a retrospective analysis of a prospectively collected observational cohort. 31 Hunt-Hess 5 patients that presented with CA were compared to 146 Hunt-Hess 5 patients that presented without CA. Clinical and imaging findings were predefined and adjudicated. Cerebral physiology measures were available for a subset of patients, matched 1:1 by age.

Results—Twenty-two (71%) CA patients had pulseless electrical activity/asystole compared to 2 (6%) with a shockable rhythm. The CA patients were younger (OR 0.96, 95% CI 0.93-0.99, $p = .009$), had more SAH on CT (OR 1.07, 95% CI 1.01-1.13, $p = .02$), and had higher in-hospital mortality (87% vs. 58%, OR 6.2 (2.1-26.6), $p = .004$). There were no differences in aneurysm location, cerebral herniation, or ictal seizures. Despite similar cerebral perfusion pressure, CA patients had pathologically lower brain tissue oxygenation, lower glucose, and higher lactate to pyruvate ratios.

Conclusions—CA in SAH is associated with larger volume bleeds. Despite normal cerebral perfusion pressures, CA patients show compromised cerebral physiology.

Keywords

Cardiac Arrest; Subarachnoid Haemorrhage; Multimodality Monitoring; Cerebral Perfusion Pressure; Mortality

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Introduction

Subarachnoid haemorrhage (SAH) is the most common neurological cause of out of hospital cardiac arrest (CA).¹ 4-18% of patients with out of hospital CA have SAH as the cause.²⁻⁴ Conversely, at least 4-7% of SAH patients that are admitted alive to the hospital have suffered from a CA.^{5,6} The mechanism of CA in SAH patients remains uncertain.

Prognosis varies considerably among studies, ranging from 45-100% mortality with the majority suggesting a dismal outcome.^{2-4,7-9} Most studies preceded the widespread use of therapeutic hypothermia for CA patients. These prior reports do not elucidate how many patients had care withdrawn and reported poor outcomes may therefore be a reflection of a self- fulfilling prophecy.

In order to better understand CA in this population, we compared comatose Hunt-Hess 5 SAH patients presenting with and without CA. The Hunt-Hess grade is a well-established scale of clinical severity in SAH and is widely used for case matching in the SAH literature; Hunt-Hess 5 SAH patients are by definition comatose on admission with mortality reaching 71%.¹⁰ By comparing Hunt-Hess matched patients with and without CA, three hypotheses were tested: (1) clinical phenotypes between the groups differ and may suggest the cause of arrest, (2) brain physiology measures would demonstrate cerebrovascular decompensation in the CA patients despite identical cerebral perfusion pressure (CPP) and management targets, (3) outcomes of aggressively treated CA patients should not significantly differ from those of non-CA patients.

Methods

Study Population

We retrospectively analyzed patients 18 years or older admitted to the Neurological Intensive Care Unit at Columbia University Medical Center between July 1996 and June 2016 with spontaneous SAH who were prospectively enrolled in the Columbia University SAH Outcomes Project. Our centre has a policy of accepting all admissions. Patients who received cardiopulmonary resuscitation within 6 hours of onset (based on information obtained by interview of family members or first responders) or who had documented CA within 24 hours of onset of symptoms and were comatose (defined as unarousable and unresponsive), i.e. Hunt-Hess grade 5, were compared to comatose SAH patients that did not present with CA. The 24 hour cut-off for inclusion was chosen to account for inconsistent times to ICU admission due to delays in presentation, diagnosis, and transfer.

Clinical Management

All patients were admitted to a dedicated neurological intensive care unit and treated according to a standardized protocol for management of SAH that has been described in detail previously.¹⁰ External ventricular drains (EVDs) were placed in patients with symptomatic hydrocephalus or intraventricular haemorrhage with reduced level of consciousness. We administered epsilon aminocaproic acid (4g bolus followed by 1g/hr until angiography) at the time of diagnosis to prevent rebleeding. Multimodality brain monitoring (MMM) including intracranial pressure (ICP) (ICP Express, Codman Neuro, Raynham,

MA), brain tissue oxygenation (PbtO₂) (Licox System, Integra LifeSciences, Tullamore, Ireland), microdialysis (CMA Microdialysis, Kista, Sweden), and regional cerebral blood flow (rCBF) (QFlow catheter, Hemedex, Cambridge, MA) was utilized according to previously published protocols.¹¹

Since 2002, all patients with CA were cooled to 32-34 °C unless absolute contraindication such as severe hemodynamic instability or sepsis (excluding intracranial haemorrhage) by either external (Arctic Sun Temperature Management System Medivance, Inc. Louisville, CO) or intravascular means (Celsius Control System, Innercool Therapies, Inc, San Diego, CA; Cool Guard, Zoll Therapeutics, San Francisco, CA) per previously described protocols.¹² Patients in whom brain death was suspected based on absent cranial nerve reflexes, motor responses, and spontaneously triggered breaths did not undergo hypothermia. The decision for the placement of invasive brain monitoring catheters during hypothermia was reached by consensus between the treating neurointensivist and neurosurgeon.

Clinical Assessment

Demographic data, social history, medical history, and clinical features (including initial cardiac rhythm following documented arrest, time to return of spontaneous circulation, and presence of clinical seizure at ictus) were prospectively obtained as soon as possible after admission via interview with family members and first responders. Both the Glasgow Coma Scale (GCS) and the Hunt-Hess Scale were used to evaluate neurological status. Physiologic stress was assessed using the Acute Physiology and Chronic Health Evaluation II (APACHE II) scale by calculating a physiological subscore after subtracting the GCS, age, and chronic health conditions from the total score.¹³ All variables were adjudicated at a weekly meeting of the neurointensivists.

Imaging, EEG, Multimodality Monitoring, and Laboratory Assessment

Admission CT scans were evaluated using the Modified Fisher Scale,¹⁴ Intraventricular Haemorrhage Score,¹⁵ Hijdra SAH sum score,¹⁶ bicaudate index for hydrocephalus,¹⁷ and for the presence of global cerebral edema.¹⁸ Continuous electroencephalography (cEEG) reports were assessed for the presence of reactivity, severe attenuation, and seizures. MMM was recorded including ICP, CPP, PbtO₂, rCBF, and microdialysis substrates (glucose, lactate, pyruvate, and the lactate to pyruvate ratio (LPR)). The levels of each microdialysis substrate were averaged for each patient over the first 12 hours following probe insertion and compared to age matched (± 2 years) Hunt-Hess 5 patients who did not have CA. The same was done for PbtO₂ and rCBF, with the exception that data was used from hours four through sixteen after probe insertion (to allow for probe stabilization/calibration).^{19,20} Echocardiographic findings, ECG abnormalities, chest radiograph findings, and initial serum studies (including basic metabolic panel, liver function tests, and cardiac troponin levels) were recorded.

Outcome Assessment

Good functional outcome was assessed by structured telephone interview and defined as a modified Rankin scale < 4 (no symptoms through moderate disability but able to walk without assistance) at 3 months.

Statistical Analysis

Data was assessed using univariable logistic regression. Significant univariable correlations ($p < .05$) were then explored in multivariable logistic regression analysis via backward elimination with CA as the dependent variable. Neurophysiologic data was compared between the two cohorts via an unpaired t-test to assess for significance. Box plots were developed based on medians, quartiles, and max/min in the usual fashion. Statistical analysis was performed with R version 3.3.1.

Results

Of 1626 patients with SAH, 1618 had data regarding CA on presentation. 36 patients (2%) presented with CA, 31 of which were included for study as they were comatose following the arrest and thus rated as Hunt-Hess 5. Comparison was made to 146 patients that presented with Hunt-Hess 5 SAH without CA.

Admission characteristics of CA patients and non-CA patients are provided (Tables 1 and 2). CA patients were younger than non-CA patients; otherwise, there were no significant differences in premorbid characteristics between the groups. There was no difference in the seizure rate between the two groups at ictus. In multivariable logistic regression, age and presence of ICH were protective for CA while SAH sum score increased the risk of CA (Table 3).

Of the CA patients, 22 (71%) had PEA/asystole, 2 (6%) had VFib/Vtach arrests and 7 (23%) had unknown initial rhythms. Twenty-two had data regarding estimated time to ROSC with a mean of 18 minutes. Treatment of CA patients included therapeutic hypothermia in 17 (55%), osmotherapy in 21 (68%), vasopressors in 25 (81%), ICP monitoring in 27 (87%) including EVD placement in 20 (65%), and additional MMM devices placed in 11 (35%), without significant differences in their use when compared to non-CA patients. Fourteen CA patients were not offered hypothermia on the presumption of brain death – all 14 were confirmed brain dead. Rebleeding rates were similar between CA patients and non-CA patients despite therapeutic hypothermia use in the majority of CA patients (19% vs. 17%, OR 1.2, 95% CI 0.4-3, $p = .78$). Aneurysm repair was attempted in 6 (19%) CA patients compared to 72 (50%) of non-CA patients (OR 0.3, 95% CI 0.1-0.6, $p = .006$).

Aminocaproic acid was given to 11 (36%) CA patients and 54 (37%) non-CA patients. Only two patients received aminocaproic acid prior to CA, neither of which was judged to have arrested due to aminocaproic acid. None of the CA patients were adjudicated to have suffered myocardial infarction (as evaluated by a combination of ECG, serum troponins, echocardiogram, and – when there was still question – cardiac consultation), but severe cardiomyopathy on echocardiogram was significantly more likely to be found in CA patients (see table 4). QTc prolongation was common in both groups.

Eleven CA patients and 32 non-CA had a component of MMM available for analysis (Figure 1). The mean CPP targets were not different between the groups (74 mm Hg vs. 68 mm Hg, 95% CI difference -6-18, $p < .32$). MMM revealed that ICP was significantly higher (18.2 mm Hg vs. 7.1 mm Hg, 95% CI difference 9.7-12.5, $p < .001$) and CPP was significantly lower (65.8 mm Hg vs. 79.9 mm Hg, 95% CI difference 11.3-16.9, $p < .001$) in CA patients.

Microdialysis revealed lower brain glucose (1.2 mM vs. 4.5 mM, 95% CI difference 2.5-4.1, $p < .001$), higher brain lactate (4.2 mM vs. 3.2 mM, 95% CI difference 0.6-1.48, $p < .001$), lower brain pyruvate (84.2 mM vs. 102.5 mM, 95% CI difference 7.7-29.1, $p = .002$), and higher LPR (50.7 vs. 36.2, 95% CI difference 10.5-18.4, $p < .001$) in CA patients. PbtO₂ was significantly lower (19.4 mm Hg vs. 26.6 mm Hg, 95% CI difference 7.7-29.1, $p = .002$) in CA patients. rCBF was not significantly lower in CA patients (20.3 vs. 22.5, 95% CI difference -0.5-4.9, $p = .1$). One patient (3%) had an asymptomatic haemorrhage due to MMM probe placement. In all patients studied, there were 2 cases of ventriculitis attributed to EVDs (2%) and no cases of infection attributed to other intracranial monitors.

Ten CA and 64 non-CA patients had cEEG data. None of the CA patients had seizures compared to 17 (26%) non-CA patients ($p = .9$). CA patients were not less likely to show reactivity (70% CA vs. 89% non-CA, OR 0.53, 95% CI 0.07-10.95, $p = .6$) or severe attenuation (50% CA vs. 48% non-CA, OR 0.9, 95% CI 0.2-3.7, $p = .9$).

CA patients had higher mortality and higher rates of brain death than non-CA patients despite no difference in code status / withdrawal of care (Table 5). 27 (87%) CA patients died compared to 84 (58%) non-CA patients (OR 6.2, 95% CI 2.1-26.6, $p = .004$). Of note, all 5 non-comatose SAH-CA patients survived. 19 (61%) CA deaths were categorized as brain death. Sixteen (84%) brain death diagnoses were made on the day of admission or the following hospital day. Nine (29%) CA patients received a DNR/DNI order (median post-bleed day 1, IQR 0) and 11 (36%) had withdrawal of life sustaining treatment (median post-bleed day 1, IQR 1). Nine CA patients donated organs (1 following cardiac death). Two (6%) CA patients achieved a mRS less than 4 compared to 16 (11%) non-CA (OR 0.5, 95% CI 0.1-2, $p = .4$).

Discussion

We attempted to elucidate cardiac arrest in patients with subarachnoid hemorrhage by testing three main hypotheses. First, we expected different clinical phenotypes between Hunt Hess 5 patients with and without cardiac arrest. Compared with non-CA patients, in our cohort Hunt-Hess 5 SAH with CA patients were younger and had more SAH on CT scan. Second, we hypothesized that brain physiology measures would demonstrate decompensation in CA patients despite identical CPP targets to non-CA patients. Using invasive neuromonitoring, we found more evidence of cerebrovascular compromise including lower CPP, lower PbtO₂, lower glucose, and higher LPR despite similar CPP targets. Third, we hypothesized that with aggressive care Hunt Hess 5 patients who presented with CA might have similar outcomes to those who presented without CA. However, despite aggressive care in a dedicated neuro-ICU, 87% of CA patients died compared to 58% of the non-CA patients. Of note, all SAH patients who were not comatose following CA survived.

While recent work has focused on distinguishing SAH patients with CA from patients with cardiac etiologies for their arrest, we instead compared Hunt-Hess 5 patients presenting with and without CA in hopes of revealing insight into the mechanism of CA in SAH patients. Seizure at ictus was not more likely in the CA group, making seizure an unlikely proximate cause of CA. Prior work has found that right-sided aneurysms weakly correlate with

measures of cardiac dysfunction, including ECG changes, troponinemia, and left ventricular dysfunction on echocardiogram.^{21,22} Posterior circulation aneurysms have been linked to sudden death in SAH.⁸ Nonetheless, we did not find any significant association between aneurysm location and CA among Hunt-Hess 5 patients. While perisylvian blood was associated with CA in the univariable analysis, only the SAH sum score remained significant in multivariable logistic regression.

Previously, aneurysmal rupture has been associated with transient spikes of ICP with decrements in CPP and CBF.²³ These perturbations are exacerbated by increasing volume of SAH.²⁴ While temporary cerebral circulatory failure may promote hemostasis, it may also precipitate respiratory center failure. The preponderance of non-shockable rhythms in our cohort and others^{2,3,9,25} likewise points to primary respiratory arrest leading to CA; although we cannot rule out that shockable rhythms may have devolved into pulseless electrical activity. QTc prolongation, while common in other cohorts and ours during the acute phase of SAH,²⁶ does not seem to commonly devolve into CA.

An alternative mechanism of CA is that a large volume of SAH causes herniation leading to respiratory arrest, but we did not find more herniation in the CA patients on CT and we lacked early MRI to evaluate for more subtle brainstem injury from resolved herniation. A third option is that increased SAH volume may simply be a marker of injury severity, and as such be indirectly correlated with other complications of SAH such as dysautonomia. There was a significantly higher rate of severely reduced ejection fraction by echocardiography among the CA patients, but this could be a result of the arrest as opposed to a proximate cause.

Regarding management, 55% of CA patients underwent hypothermia. Institution of hypothermia was limited by the high number of patients that arrived with suspicion of brain death. We did not note a significant difference in the rebleeding rate following therapeutic hypothermia. Prior studies evaluating the role of hypothermia during the acute phase of SAH (prior to securing aneurysm) are limited to small retrospective series.^{27,28} The safety of this practice given the effects of cooling on coagulation²⁹ is unknown. It is reassuring then that we did not see an increased rebleeding rate following initiation of therapeutic hypothermia.

MMM of a limited number of CA patients represents a novel aspect of our study. Our cohort offers a unique opportunity to explore the post-CA brain physiology. CA patients demonstrated higher ICP, lower CPP, lower PbtO₂, lower brain glucose, and higher LPR than no-CA patients despite similar CPP targets. Mean values for PbtO₂, brain glucose, and LPR in the CA patients all fall within a range previously associated with cerebrovascular compromise,^{30–32} even though mean ICP and CPP were within normal limits. rCBF was similarly lower in CA patients but this result did not meet significance ($p < .1$) likely due to small sample size. In sum, the MMM data suggest that in CA patients conventional ICP and CPP goals are insufficient to restore brain physiology. As such, we would advocate that in the future clinicians may either attempt to target brain tissue oxygenation and cerebral microdialysis goals in lieu of CPP goals or pursue higher MAP/ CPP goals.

Other groups have advocated very high MAP goals following CA.³³ Studies show improvements in CBF and in outcomes for animals treated with induced hypertension following CA.^{34,35} In humans, there is a loss or right-shift of autoregulation following CA, implying that augmented MAP goals may be necessary to secure adequate blood supply to the brain.³⁶ Although one small trial of induced hypertension following CA failed to find an improvement in cerebral tissue oxygenation as measured by near infrared spectroscopy, MAP pushes in that trial may have been too late as they were instituted after a mean of 17 hours whereas prior data has shown that spontaneously higher blood pressures in the first 2 hours after arrest predict good outcome.^{37,38}

Rather than advocating for a specific MAP/CPP goal, it may instead be beneficial to target normalization of PbtO₂ and cerebral microdialysis values. Retrospective trials in brain resuscitation following TBI have shown a benefit to targeting PbtO₂ instead of traditional MAP/CPP goals.³⁹ We await the results of BOOST 3, the first phase 3 randomized prospective trial to assess this strategy which may offer further support for MMM targeted therapy after other forms of severe brain injury such as SAH-induced CA.

Our study has several limitations. We lacked important data regarding the arrest for many patients including the presence of a witness, initial rhythm, and receipt of bystander CPR. Outcomes were obtained in an unblinded fashion which may have biased the results. MMM was only available in a small subset of patients. Furthermore, we lacked direct measures of cerebral blood flow (such as Xenon-CT). Hypothermia, performed only in CA patients, may have affected MMM values; however, limited data suggest that hypothermia attenuates, not causes, metabolic crisis following CA.⁴⁰ We did not obtain early MRI which may have been more informative in terms of early brain injury and evidence of transient herniation. Finally, we cannot be sure how representative our sample was, as many patients with SAH-induced CA may escape diagnosis.

Conclusion

CA in SAH is associated with larger volume bleeds. More study is required to confirm the mechanism of arrest. Future study is likewise indicated to assess the effect of a strategy targeting invasive markers of brain physiology.

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Figure 1. Invasive Multimodality Monitoring Parameters in Hunt-Hess 5 SAH Patients, Stratified by Presentation with Cardiac Arrest

CA patients show evidence of cerebral compromise compared to no-CA patients despite similar cerebral perfusion pressure targets, including lower cerebral perfusion pressure (A), higher intracranial pressure (B), lower brain tissue oxygenation (C), lower cerebral glucose concentrations (D), and higher cerebral lactate- pyruvate ratio (E). All comparisons are significant with $p < .001$.

Table 1

Admission Characteristics of Hunt-Hess 5 patients, Stratified by Presentation with Cardiac Arrest

Characteristic	Hunt-Hess 5 with CA (N = 31)	Hunt-Hess 5 without CA (N = 146)	OR (95% CI)	p-value
Age, mean (SD), y	51.7 (14.6)	60.0 (16.4)	0.97 (0.94-0.99)	.009
Female	19 (61)	105 (72)	0.60 (0.28-1.42)	.24
Race				
White	11 (35)	53 (36)	1 (0.3-3.50)	.95
Black	5 (16)	25 (17)	1 (0.3-2.96)	.95
Hispanic	14 (45)	50 (34)	1.4 (0.60-3.30)	.36
Asian	1 (3)	13 (9)	0.37 (0.02-2.20)	.5
Other	0 (0)	5 (3)	–	.99
Past Medical History				
Hypertension	14 (45)	79(54)	0.7 (0.3-1.6)	.4
Diabetes	2 (6)	79(54)	0.5 (0.1-2.0)	.4
Angina	1 (3)	4 (2.7)	1.21 (0.06-8.6)	.9
Myocardial Infarction	0	3 (2.0)	–	.99
Arrhythmia	0	8 (5)	–	.99
Congestive Heart Failure	0	4 (3)	–	.99
Alcohol Use	10 (32)	46(32)	11.6 (0.47-2.82)	.9
Tobacco use	13 (42)	55 (38)	1.16 (0.5-2.69)	.9
Clinical Features				
Sentinel Headache	13 (42)	29 (19.7)	1.7 (0.5-5.2)	.4
Seizure at Onset	5 (16)	25 (17.0)	1 (0.3-2.6)	.9
GCS, mean (SD)	3.4 (1.1)	4.8 (2.0)	0.4 (0.3-0.7)	<.001
Apache-GCS, mean (SD)	16.7 (5.1)	11.8 (4.7)	1.2 (1.1-1.3)	<.001
Abnormal Electrocardiogram	22 (70)	61 (42)	4.5 (1.6-16.2)	<.001
Prolonged QTc	11 (44)	41 (39)	1.2 (0.5-2.9)	.7
Hospital Complications				
Arrhythmia	15 (48)	33 (23)	3.2 (1.4-7.1)	.007
Hypotension requiring vasopressors	25 (81)	86 (59)	2.9 (1.2-8.1)	.04
Myocardial Infarction	6 (19)	20 (14)	1.5 (0.5-3.9)	.6
Hyperglycemia	28 (90)	123 (84)	1.8 (0.6-7.7)	.6

Data are presented as number (%) unless otherwise specified. Abbreviations: CA, cardiac arrest; OR, odds ratio; CI, confidence interval; SD, standard deviation; GCS, Glasgow Coma Scale

Table 2

Initial CT/Angiography Characteristics of Hunt-Hess 5 patients, Stratified by Presentation with Cardiac Arrest

Characteristic	Hunt-Hess 5 with CA (N = 31)	Hunt-Hess 5 without CA (N = 146)	OR (95% CI)	p-value
Modified Fisher, mean (SD)	3.2 (0.68)	3 (0.99)	1.4 (0.9-2.2)	.2
Hijdra SAH Sum score, mean (SD)	23.7 (8.9)	18.7 (9.3)	1.07 (1.02-1.13)	.01
IVH	22 (71)	119 (82)	0.5 (0.2-1.2)	.1
ICH	4 (13)	51 (35)	0.28 (0.08-0.75)	.02
Global Cerebral Edema	28 (90)	128 (88)	1.31 (0.33-8.73)	1
Evidence of Herniation	12 (39)	51 (35)	1.6 (0.1-10.7)	.7
Hydrocephalus	5 (16)	51 (35)	0.32 (0.1-0.85)	.046
Early Infarction	4 (13)	20 (14)	0.97 (0.3-3)	.97
Early Vasospasm	1 (6)	5 (4)	1.6 (0.1-10.7)	.7
Aneurysm Size, mean (SD), mm	10.3 (7.9)	9 (8.0)	1 (0.9-1.1)	.63
Aneurysm Location *	(N=13)	(N=113)		
Anterior	7 (54)	83 (73)	0.54 (0.18-1.74)	.28
Posterior	6 (46)	30 (27)	–	–
Right	7 (54)	45 (40)	1.2 (0.4-4.3)	.8
Left	5 (38)	38 (34)	–	–

Data are presented as number (%) unless otherwise specified.

Abbreviations: CA, cardiac arrest; CT, computed tomography; OR, odds ratio; CI, confidence interval; SD, standard deviation; GCS, Glasgow Coma Scale; IVH, intraventricular haemorrhage; ICH, intracerebral haemorrhage.

* Anterior compared to posterior location and right compared to left.

Table 3

Multivariate Analysis of Demographic and Imaging Characteristics for Hunt-Hess 5 Patients Presenting with Cardiac Arrest

Characteristic	Adjusted OR (95% CI)	p-value
Age	0.96 (0.93-0.99)	.009
Hijdra SAH Sum score	1.07 (1.01-1.13)	.02
ICH	0.23 (0.05-0.73)	.03

Abbreviations: OR, odds ratio; CI, confidence interval; SAH, subarachnoid haemorrhage; ICH, intracerebral haemorrhage

Table 4

Echocardiogram Abnormalities of Hunt-Hess 5 SAH patients, Stratified by Presentation with Cardiac Arrest

	Hunt-Hess 5 with CA (n=17)	Hunt-Hess 5 without CA (n=115)	OR (95% CI)	p-value
Mildly reduced EF (41-49%)	1 (6)	8 (7)	2.4 (0.1-19.1)	.99
Moderately reduced EF (31-40%)	1 (6)	8 (7)	2.4 (0.1-19.1)	.99
Severely reduced EF (<30%)	8 (47)	9 (8)	17.3 (4.6-77)	<.001

Data presented as number (%).

Abbreviations: CA, cardiac arrest; SAH, subarachnoid haemorrhage; OR, odds ratio; CI, confidence interval; EF, ejection fraction

Table 5

Outcomes of Hunt-Hess 5 SAH patients, Stratified by Presentation with Cardiac Arrest

	Hunt-Hess 5 with CA (n = 31)	Hunt-Hess 5 without CA (n=146)	OR (95% CI)	p-value
Rebleed	6 (19)	25 (17)	1.2 (0.4-3)	.78
DNR	9 (29)	57 (39)	0.6 (0.3-1.4)	.2
Withdrawal of care	11 (35)	55 (38)	0.9 (0.4-1.9)	.7
In-hospital death	27 (87)	84 (58)	6.2 (2.1-26.6)	.004
Brain Death	19 (61)	42 (29)	4.3 (1.9-9.8)	<.001
Cardiac death	8 (26)	42 (29)	0.8 (0.1-16)	.8
mRS < 4 at 3 months	2 (6)	16 (11)	0.5 (0.1-2)	.4

Data presented as number (%).

Abbreviations: CA, cardiac arrest; SAH, subarachnoid haemorrhage; OR, odds ratio; CI, confidence interval; DNR, do not resuscitate order placed; mRS, modified Rankin scale