neurocritical care society Neurocrit Care (2014) 21:S38–S63
DOI 10.1007/s12028-014-0033-5

REVIEW ARTICLE

Advanced Monitoring of Systemic Hemodynamics in Critically Ill Patients with Acute Brain Injury

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Published online: 11 September 2014

Abstract Hemodynamic monitoring is widely used in critical care; however, the impact of such intervention in patients with acute brain injury (ABI) remains unclear. Using PubMed, a systematic review was performed (1966–August 2013), and 118 studies were included. Data were extracted using the PICO approach. The evidence was classified, and recommendations were developed according to the GRADE system. Electrocardiography and invasive monitoring of arterial blood pressure should be the minimal hemodynamic monitoring required in unstable or at-risk patients in the intensive care unit. Advanced hemodynamic monitoring (i.e., assessment of preload, afterload, cardiac output, and global systemic perfusion) could help establish goals that take into account cerebral blood flow and oxygenation, which vary depending on diagnosis and disease stage. Choice of techniques for assessing preload, afterload, cardiac output, and global systemic perfusion should be guided by specific evidence and local expertise. Hemodynamic monitoring is important and has specific indications among ABI patients. Further data are necessary to understand its potential for therapeutic interventions and prognostication.

Keywords Hemodynamic · Monitoring · Brain injury · Critical care · Cardiac function · Prognosis · Outcome · Recommendations · Consensus

Abbreviations

ABI	Acute brain injury
CA	Cardiac arrest
CBF	Cerebral blood flow
CO	Cardiac output
CPP	Cerebral perfusion pressure
CVP	Central venous pressure
DCI	Delayed cerebral ischemia
ΔCO_2	Venous-arterial difference in carbon dioxide
dICV	Inferior vena cava distensibility
dP/dt	Rate of rise of left ventricular pressure
Ea	Arterial elastance
EGHO	Early goal-directed hemodynamic optimization
EVLW	Extravascular lung water
FR	Fluid responsiveness
FTC	Flow time corrected
GEF	Global ejection fraction
GEDVI	Global end-diastolic volume index
ICH	Intracranial hemorrhage
ICU	Intensive care unit
LVEF	Left ventricular ejection fraction
MAP	Mean arterial pressure
NPE	Neurogenic pulmonary edema
OHCA	Out-of-hospital cardiac arrest
PAC	Pulmonary artery catheter
PAOP	Pulmonary artery occlusion pressure
PE	Pulmonary edema
PLR	Passive leg raising
PWCA	Pulse contour wave analysis
RCT	Randomized clinical trial

The Participants in the International Multi-disciplinary Consensus Conference on Multimodality Monitoring (see "Appendix").

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RWMA	Regional wall motion abnormalities
SAH	Subarachnoid hemorrhage
SPV	Systolic pressure variation
SVR	Systemic vascular resistances
SVV	Stroke volume variation
TBI	Traumatic brain injury
TCD	Transcranial Doppler
TH	Therapeutic hypothermia
TT	Trans-pulmonary thermodilution

Introduction

The management of patients with acute brain injury (ABI) includes the diagnosis and management of several medical disorders and complications [1]. Cardiovascular impairment is frequent, e.g., after subarachnoid hemorrhage (SAH), and is associated with increased morbidity and mortality [2, 3]. The complex pathophysiology of these cardiovascular alterations has been directly linked to the neurological injury, with data suggesting involvement of hypothalamic stimulation and/or failure of the autonomic system [4–6]. Different therapeutic interventions to improve cerebral perfusion pressure (CPP), such as hypervolemia or induced hypertension, also can result in cardiac arrhythmias, pulmonary edema, or left ventricular dysfunction [7, 8], which may exacerbate brain injury. In this setting, monitoring systemic hemodynamics can play an important role in avoiding such complications and optimizing cerebral blood flow (CBF) and oxygen delivery [9, 10].

Methods

This systematic review was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [11].

Search Criteria

Studies were considered eligible based on the PICO approach, which includes (a) Patient population, i.e., critically ill patients with at least one of the following ABI: traumatic brain injury (TBI), SAH, intracranial hemorrhage (ICH), ischemic stroke, coma after cardiac arrest (CA), central nervous system infection, encephalitis, seizures; neurosurgery; (b) Intervention provided, i.e., monitoring of systemic hemodynamics; (c) Controls, i.e., patients with ABI without hemodynamic monitoring or patients without ABI but undergoing hemodynamic monitoring or patients with ABI monitored with two different devices; (d) Outcome endpoints, i.e., mortality, survival with intact neurological function, complications (cardiac, pulmonary, infection), or modification in therapy (including intensity or drug choice). For this review, arterial and central venous lines were considered the minimal monitoring needed in patients with ABI, and mean arterial pressure (MAP) and central venous pressure (CVP) monitoring were used as the "control" group when compared to more advanced monitoring. "Systemic hemodynamic" parameters were divided into the following categories: (a) assessment of systolic and diastolic function [left ventricular ejection fraction (LVEF); rate of rise of left ventricular pressure (dP/dt); continuous ejection fraction; global ejection fraction on trans-pulmonary thermodilution (GEF); esophageal Doppler]; (b) measurement of cardiac output (CO) [i.e., PAC; trans-pulmonary thermodilution (TT); pulse wave contour analysis (PWCA); echocardiography, bioimpedence, etc.]; (c) assessment of preload (pulmonary artery occlusion pressure (PAOP); extravascular lung water (EVLW); global end-diastolic volume (GEV); esophageal flow time corrected (FTC-Doppler)]; (d) assessment of afterload [(systemic vascular resistances (SVR); arterial elastance (Ea)]; (e) assessment of fluid responsiveness [systolic pressure variation (SPV); pulse pressure variation (ΔPP); stroke volume variation (SVV); passive leg raising (PLR); pleth variability index)]; (f) adequacy of global perfusion [lactate levels; mixed or central venous saturation; venous-arterial difference in carbon dioxide (ΔCO_2)]. Evaluation of heart-rate variability or other parameters of autonomic function, and studies using echocardiography to diagnose the etiology of ischemic stroke if no specific hemodynamic parameters were reported were not included in this review.

Information Sources and Search Strategy

Using PubMed, a systematic review of English articles was performed from 1966 through August 15, 2013. The search strategy included the terms "brain injury," "traumatic brain injury," "subarachnoid hemorrhage," "stroke," "intracranial hemorrhage," "cardiac arrest," "seizures," "epilepsy," "neurosurgery," "encephalitis," "meningitis"—used with one of the following: "hemodynamics," "hemodynamic monitoring," "cardiac output," "ventricular function," "ejection fraction," "preload," "extravascular lung water," "end-diastolic volume," "filling pressure," "venous saturation," "mixed venous saturation," "central venous saturation," "venous-arterial carbon dioxide," "arterio-venous carbon dioxide difference," "delta CO_2," "CO_2 gap," "venous CO_2," "lactate," "fluid responsiveness," "stroke volume variation," "systolic pressure variation," "pulse pressure variation," "passive leg raising," "pleth variability index," "afterload," "vascular resistances," and "elastance." Additional

references for relevant studies were also searched from review articles (i.e., defined as "other sources").

Study Selection and Data Collection

One author independently reviewed all citations, abstracts, and full-text articles to select eligible studies. Excluded were (a) review articles; (b) case reports or case-series with $\leq$5 patients; (c) experimental studies; (d) studies on pediatric ICU populations (<18 years); (e) studies that were not conducted on ICU patients; (f) studies dealing with brain dead patients. Data were abstracted using a predefined abstraction spreadsheet, according to the PICO system. No attempt was made to re-analyze the data and no meta-analysis was performed since there are insufficient randomized (RCT) or case–control studies.

Review End-Points

The end-points of this review (in patients with ABI) were to answer the following questions:

1. What is the proportion of patients who have altered systemic hemodynamics and how many will develop circulatory failure, inadequate perfusion or organ dysfunction?
2. Can monitoring of systemic hemodynamics help understand the mechanisms of circulatory failure, inadequate perfusion, or organ dysfunction?
3. Does hemodynamic monitoring have a specific role in optimizing brain perfusion and oxygenation or brain-specific therapy?
4. What is the impact of systemic hemodynamic monitoring and related therapies on morbidity, mortality, and neurological outcome?
5. How can fluid responsiveness be assessed in ABI patients?
6. What hemodynamic monitoring is indicated in ABI patients, in particular to diagnose and support the management of unstable or at-risk patients?

Grading of Evidence

The quality of evidence was judged based on the Grades of Recommendation, Assessment, Development, and Evaluation (GRADE) system approach [12].

Literature Summary

The search retrieved a total of 25,801 citations (Fig. 1), and 118 article met inclusion criteria. The numbers of articles for each disease were 68 for SAH, 12 for TBI, 8 for stroke or ICH, 23 for CA, and 7 for neurosurgery. The search found 4 randomized clinical trials (RCTs), 5 case-controlled studies (all "before-after" studies), 81 prospective/observational studies, and 28 retrospective studies.

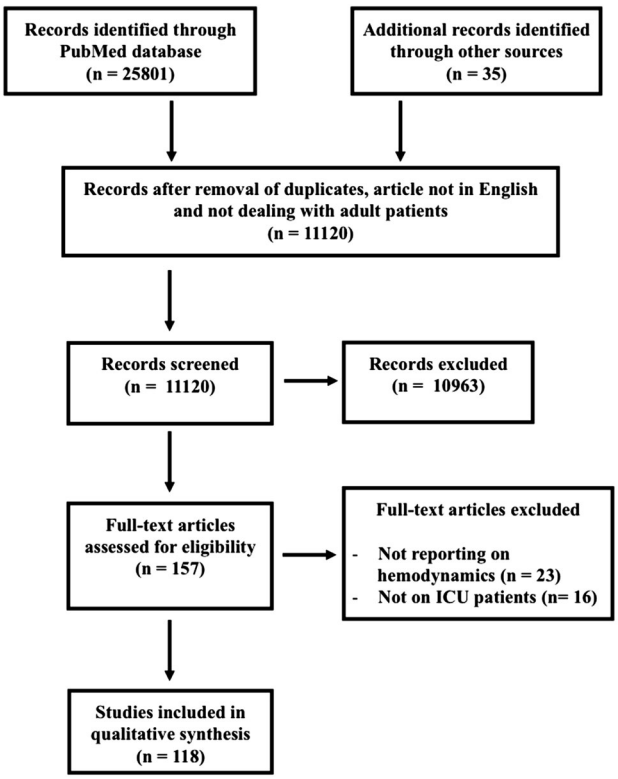

Fig. 1 Flow-chart of the research process according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement

Forty-six studies (n = 5,022) included data on cardiac function. Most (38/46) were prospective and focused on SAH; all except three used echocardiography (Table 1). Forty-two studies (n = 4,224) reported data on CO. Most (28/42) were prospective and 27/42 focused on SAH; 3 studies were RCTs (Table 2). Twelve studies (n = 969) reported data on preload assessment. Most were performed in SAH patients (11/12; Tables 3, 4). Five studies (n = 198) reported data on afterload. All evaluated SVR and were mostly performed in SAH patients (4/5). No study evaluated arterial elastance (Table 5). Six studies (n = 250) reported data on fluid responsiveness (Table 6). Twenty studies (n = 3,870) reported data on the adequacy of global perfusion; 3 used $ScvO_2$, 16 measured lactate levels, and only 1 focused on ΔCO_2. Most of studies were retrospective (11/20) and evaluated patients after CA (12/20) (Table 6).

Table 1 Studies that evaluate cardiac function in acute brain injury patients

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Incidence of altered cardiac function							
Sandvei et al. [21]	18	P	SAH	Echography	To assess the incidence of LV dysfunction	Systolic function and SV were higher in patients than in controls Diastolic function was altered in the early phase when compared to controls	Very low
Banki et al. [14]	173	P	SAH	Echography	To assess the incidence and time course of LV dysfunction	15 % had low LVEF 13 % of patients had RWMA with normal LVEF Recovery of LV function observed in 66 % of patients	Low
Mayer et al. [16]	57	P	SAH	Echography	To assess the incidence of LV dysfunction	8 % of RWMA, which were associated with hypotension and PE	Low
Jung et al. [15]	42	P	SAH	Echography	To assess the incidence of LV dysfunction	Only 1/42 patients had LV dysfunction	Low
Lee et al. [83]	24	P	SAH	Echography	To assess the incidence of Tako-Tsubo cardiopathy among patients with SAH-induced LV dysfunction	8/24 patients had Tako-Tsubo pattern All patients recovered LVEF >40 %	Very low
Khush et al. [19]	225	P	SAH	Echography	To assess the incidence and predictors of SAH-induced LV dysfunction	RWMA were found in 27 % of patients Apical sparing pattern was found in 49 % of patients Younger age and anterior aneurysm position were independent predictors of this AS pattern	Low
Jacobshagen et al. [84]	200	R	CA	Echography	To assess the incidence of LV dyfunction	Significant reduction of LVEF (32 ± 6 %) on admission	Very low
Ruiz-Bailen et al. [85]	29	P	CA	Echography	To assess the incidence and time course of LV dysfunction	LV dysfunction occurred in 20/29 patients in the early phase after CA LVEF slowly improved among survivors	Very low
Role of cardiac function monitoring to explain the mechanisms of brain injury-related cardiopulmonary complications							
Miss et al. [86]	172	P	SAH	Echography	To evaluate the correlation of LV dysfunction with the type of aneurysm therapy	No difference in the occurrence of RWMA or LV dysfunction with regard of coiling or clipping	Low
Frangiskasis et al. [56]	117	P	SAH	Echography	To evaluate the correlation of LV dysfunction with ECG abnormalities	Low LVEF associated with VA	Low
Pollick et al. [17]	13	P	SAH	Echography	To evaluate the correlation of LV dysfunction with ECG abnormalities	RWMA in 4/13 patients RWMA was associated with inverted T waves	Very low

Table 1 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Kono et al. [87]	12	P	SAH	Echography	To evaluate the correlation of LV dysfunction with ECG and coronary angiography abnormalities	Apical LV hypokinesia was not associated with coronary stenosis despite ST elevation on ECG	Low
Davies et al. [18]	41	P	SAH	Echography	To evaluate the correlation of LV dysfunction with ECG abnormalities	RWMA in 10 % of patients RWMA not associated with ECG alterations	Low
Bulsara et al. [13]	350	R	SAH	Echography	To evaluate the correlation of LV dysfunction with ECG abnormalities and markers of heart injury	LVEF <40 % in 3 % of patients No association of LV dysfunction with ECG abnormalities Peak of cTnI in SAH patients was lower than matched patients with MI	Very low
Deibert et al. [88]	43	P	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	RWMA associated with high cTnI RWMA resolved over time in all patients	Low
Hravnak et al. [89]	125	P	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	High cTnI was associated with RWMA and lower LVEF Only 26 % of patients returned to normal cardiac function over time	Low
Naidech et al. [29]	253	R	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	High cTnI was associated with RWMA and low LVEF	Very low
Parekh et al. [30]	41	P	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	High cTnI was associated with low LVEF	Low
Tung et al. [31]	223	P	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	Low LVEF predicted high cTnI	Low
Kothavale et al. [90]	300	P	SAH	Echography	To assess the relationship between LV dysfunction and markers of heart injury	RWMA in 18 % patients RWMA associated with poor neurological status and high cTnI levels	Low
Apak et al. [91]	62	P	Stroke	Echography	To assess the relationship between LV dysfunction and markers of heart injury	Serum levels of cTnT were inversely correlated with LVEF	Low
Zaroff et al. [92]	30	P	SAH	Echography	To assess the relationship between RWMA and patterns of coronary artery disease	RWMA did not correlate with typical patterns of coronary artery disease RWMA resolved in all patients	Very low
Banki et al. [6]	42	P	SAH	Echography	To assess the relationship between LV dysfunction of myocardial perfusion and innervation	LV systolic dysfunction was associated with normal myocardial perfusion and abnormal sympathetic innervation	Low
Abdelmoneim et al. [32]	10	P	SAH	RTP-CE	To assess microvascular perfusion and echographic abnormalities after SAH	RWMA was not associated with altered myocardial perfusion	Very low
Sugimoto et al. [28]	77	R	SAH	Echography	To assess the relationship between LV dysfunction and estradiol (ES) or norepinephrine (NE)	The incidence of RWMA in the high-NE/low-ES group was significantly higher than the low-NE/high-ES group	Very low

Table 1 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Sugimoto et al. [27]	48	R	SAH	Echography	To assess the relationship between LV dysfunction and norepinephrine (NE) levels	Plasma NE levels were significantly higher in patients with RWMA and inversely correlated with LVEF	Very low
Tanabe et al. [22]	103	P	SAH	Echography	To assess the relationship between LV dysfunction and PE	Higher incidence of systolic or diastolic dysfunction in patients with elevated cTnI	Low
Kopelnik et al. [23]	207	P	SAH	Echography	To assess the incidence of diastolic dysfunction and its relationship with PE	Diastolic dysfunction was observed in 71 % of subjects Diastolic dysfunction was an independent predictor of PE	Low
Tung et al. [93]	57	R	SAH	Echography	To assess the relationship between LV dysfunction and elevated BNP	High BNP in patients with systolic or diastolic dysfunction	Very low
Meaudre et al. [94]	31	P	SAH	Echography	To assess the relationship between LV dysfunction and elevated BNP	No correlation between diastolic dysfunction and BNP	Very low
Naidech et al. [34]	171	P	SAH	Echography	To assess the relationship between LV dysfunction and PE	No association of LV dysfunction and PE	Low
McLaughin et al. [95]	178	R	SAH	Echography	To assess the relationship between LV systolic or diastolic dysfunction and PE	Occurrence of PE was associated with both systolic or diastolic dysfunction	Very low
Sato et al. [36]	49	P	SAH	TT	To assess variables related to the development of PE	Patients with PE had lower cardiac function than others	Low
Junttila et al. [35]	108	P	ICH	Echography	To evaluate the predictive value of echographic abnormalities for NPE	LVEF <50 % and E/A >2 more frequent in NPE patients No predictive value of such abnormalities for NPE	Low
Kuwagata et al. [60]	8	P	TBI	Echography	To assess the effects of TH on cardiac function	TH did not affect stroke volume and diastolic function	Very low
Cardiac function monitoring findings and outcome							
Yousef et al. [46]	149	P	SAH	Echography	To evaluate which hemodynamic variable was associated with DCI	No influence of LVEF or RWMA on DCI	Low
Jyotsna et al. [96]	56	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	LV dysfunction was associated with poor outcome	Low
Sugimoto et al. [58]	47	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	RWMA independent risk factor of mortality	Low
Papanikolaou et al. [97]	37	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	LV dysfunction associated with DCI and poor outcome	Low
Temes et al. [3]	119	P	SAH	Echography	To assess the impact of LV dysfunction on cerebral infarction and neurological outcome	LV dysfunction independent predictor of DCI but not of neurologic outcome	Low
Vannemreddy et al. [57]	42	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	RWMA was associated with poor GCS on admission and increased hospital stay but not with increased mortality	Low

Table 1 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Urbaniak et al. [61]	266	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	LV dysfunction not associated with outcome	Low
Yarlagadda et al. [62]	300	P	SAH	Echography	To evaluate the prognostic value of myocardial dysfunction after SAH	LVEF not associated with outcome	Low
Front et al. [98]	64	R	Stroke	Radionuclide	To evaluate the prognostic value of LVEF after stroke	Non-survivors had lowe LVEF (52 ± 18 %) than survivors (64 ± 10 %)	Very low
Chang et al. [59]	165	P	CA	Echography	To assess the LV function and its relationship with outcome	Lower LVEF associated with previous cardiac disease and epinephrine doses LVEF <40 % had higher mortality than normal LVEF	Low
Khan et al. [99]	138	P	CA	Echography	To assess the LV function and its relationship with outcome	LVEF <40 % had higher mortality than normal LVEF	Low

P prospective, *R* retrospective, *SAH* subarachnoid hemorrhage, *TBI* traumatic brain injury, *TT* transpulmonary thermodilution, *PE* pulmonary edema, *CO* cardiac output, *PCWA* pulse contour wave analysis, *LVEF* left ventricular ejection fraction, *NPE* neurogenic pulmonary edema, *CV* cerebral vasospasm, *CI* cardiac index, *PE* pulmonary edema, *IABP* intra-aortic balloon counterpulsation, *LVEF* low ventricular ejection fraction, *cTnI* troponin I, *GEDVI* global end-diastolic volume index, *GEF* global ejection fraction, *DCI* delayed cerebral infarction, *BNP* brain natriuretic peptide, *SV* stroke volume, *ECG* electrocardiogram, *VA* ventricular arrhythmias, *NPE* neurogenic pulmonary edema, *RTP-CE* real-time contrast echocardiography

What is the Proportion of Patients Who Have Altered Systemic Hemodynamics and How Many Will Develop Circulatory Failure, Inadequate Perfusion or Organ Dysfunction?

Several studies reported altered LVEF after SAH in 2–15 % of patients. [13–15] Similarly, regional wall motion abnormalities (RWMA) were described in 5–45 % of patients [16–19], although RWMA may be present together with normal LVEF [20]. Diastolic dysfunction occurred in 46–89 % patients after SAH [21–23]. Few studies evaluated the incidence of altered CO in patients with ABI. In SAH patients, high CI values were present on admission and progressively diminished on day 5 (from 5.3 ± 0.4 to 3.5 ± 0.2 L/min/m^2); higher CI and EVLWI were found in those patients admitted with a poor neurological status (WFNS 4-5) [24]. After ischemic stroke, similar CI values were reported when compared to matched-control subjects; however, patients admitted with poor neurological status had higher CI than others [25]. Finally, low CI was common in the early phase after CA and progressively normalized over time, except in those patients who eventually died in cardiogenic shock [26]. Hypovolemia, suggested by low GEDVI on admission, is frequent after SAH, especially in those patients admitted with a poor neurological status [24].

Can Monitoring of Systemic Hemodynamics Help Understand the Mechanisms of Circulatory Failure, Inadequate Perfusion, or Organ Dysfunction?

Cardiac Function

Among several hypotheses, the main mechanism of cardiac injury following SAH is thought to be associated with sympathetic stimulation and catecholamine release. In one study in 48 SAH patients [27], those with RWMA had significantly higher plasma norepinephrine levels than did those with normal echocardiography (2,098 ± 1,773 vs. 963 ± 839 pg/mL, $p = 0.02$). Plasma norepinephrine levels also were inversely correlated with LVEF. Multivariate logistic regression analysis revealed that increased plasma norepinephrine levels were predictive of RWMA. Similarly, Sugimoto et al. [28] showed those patients with RWMA on admission had decreased estradiol and elevated norepinephrine levels, and the combination of both significantly increased the risk for RWMA development.

Another important issue was the concomitant presence of altered cardiac function and altered ECG or increased markers of heart injury. SAH patients with cTnI ≥0.3 ng/mL had significantly lower LVEF (52 vs. 63 %, $p < 0.001$) than others [29]. In addition, 44 % of them had

Table 2 Studies evaluating cardiac output (CO) in acute brain injury patients

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Incidence of altered CO							
Mutoh et al. [24]	46	P	SAH	TT	To evaluate the time course of cardiac function	High CI on admission which diminished on day 5	Low
						Higher CI in patients with poor neurological status	
Trieb et al. [25]	30	P	Stroke	PAC	To evaluate CO after ischemic stroke	Patients with stroke had significantly higher CO than comparable controls	Low
Laurent et al. [26]	165	R	CA	PAC	To evaluate hemodynamics after CA	Low CI is common in the early phase after CA, which normalizes thereafter, except in those dying with cardiogenic shock and MOF	Very low
Rzheutskaya et al. [100]	13	P	TBI	TT	To assess hemodynamic alterations after TBI	Four different hemodynamic response according to CI, SVR, SVV and response to fluid administration	Very low
Schulte Esch et al. [39]	12	P	TBI	PAC	To assess hemodynamic alterations after TBI	Elevated CI with high PAOP and low SVRI were reported	Very low
Role of CO monitoring to explain the mechanisms of brain injury-related cardiopulmonary complications							
Sato et al. [36]	49	P	SAH	TT	To assess variables related to the development of PE	Patients with PE had lower CI than others	Low
Deehan et al. [37]	24	R	SAH	PAC	To evaluate hemodynamics in patients with PE	Variable hemodynamic variables	Very low
					To assess effects of dobutamine	Increased CI and decreased PAOP in patients with PE	
Vespa et al. [38]	56	R	SAH	PAC	To evaluate the mechanisms of poor oxygenation after SAH	Similar hemodynamics between patients with and without poor oxygenation	Very low
Tamaki et al. [101]	15	P	TBI	PAC	To assess hemodynamic alterations after TBI	All patients had high PAOP and PVR	Very low
						Hypotensive patients had low CI and elevated SVRI	
						Normotensive patients had high SVRI	
Nicholls et al. [102]	60	P	TBI		To assess hemodynamic alterations after TBI	High CI and MAO with reduced tissue oxygenation were found	Low
						Survivors had higher CI and tissue oxygenation than non-survivors	
Bergman et al. [40]	50	P	OHCA	PAC	To evaluate the effects of TH on hemodynamics	TH lowered heart rate, filling pressures, CO and MAP without deleterious effects on SvO_2	Low
Zobel et al. [41]	40	P	CA	PAC	To evaluate the effects of TH on hemodynamics during cardiogenic shock	TH improved hemodynamics during cardiogenic shock following CA	Low

Table 2 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Sato et al. [42]	60	P	SAH	PAC	To evaluate the effects of TH on systemic and cerebral hemodynamics during surgery	TH was associated with decreased CI and increased arterio-jugular difference in oxygen	Low
Association between CO and brain perfusion, neurological complications, or outcome							
Tone et al. [47]	42	P	SAH	PAC	To evaluate the correlation between hemodynamic variables and CBF	CBF was correlated with CI	Very low
Hashimoto et al. [103]	20	P	BS	TT	To evaluate the correlation between hemodynamic variables and CBF	CBF was not correlated with CI after BAVM resection	Very low
Watanabe et al. [33]	34	P	SAH	TT	To evaluate which hemodynamic variable was associated with the occurrence of DCI	DCI was associated with lower CI	Low
Mayer et al. [44]	72	R	SAH	Echography	To evaluate which hemodynamic variable was associated with the occurrence of DCI	DCI was associated with lower CI	Very low
Yousef et al. [46]	149	P	SAH	Echography	To evaluate which hemodynamic variable was associated with the occurrence of DCI	DCI was associated with lower CI	Low
Torgesen et al. [104]	153	R	CA	PAC	To evaluate the impact of hemodynamic variables on outcome during NT	No association of hemodynamic variables with outcome	Very low
Torgesen et al. [105]	134	R	CA	PAC	To evaluate the impact of hemodynamic variables on outcome during TH	Elevated CI was associated with poor outcome	Very low
Yamada et al. [106]	34	P	TBI	Dye Dilution	To evaluate the impact of hemodynamic variables on outcome after severe TBI	Low CI was associated with poor outcome	Very low
Effects of therapies modifying CO on neurological status							
Chatterjee et al. [107]	15	P	BS	Echography	To evaluate the effects of mannitol on systemic hemodynamics	Mannitol increased CI during 15 min after administration	Low
Stoll et al. [108]	20	P	Stroke	BioImp	To evaluate the effects of HES on systemic hemodynamics	HES administration avoided nocturnal decrease in CO and MAP No effects on neurological status were reported	Very low
Finn et al. [51]	32	P	SAH	PAC	To evaluate the effects of hemodynamic optimization on neurological status	Maintaining PAOP between 14 and 16 mmHg reversed neurological deficit; all patients had CI >4.5 L/min m^2	Very low

Table 2 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Mori et al. [52]	98	P	SAH	PAC HHH	To evaluate the effects of HHH therapy on CBF and neurological status	HHH increased PAOP and CI Increased MAP and CI was associated with increased CBF	Low
Otsubo et al. [50]	41	P	SAH	PAC NV-HT	To evaluate the effects of NV-HT on neurological status	NV-HT increased also CI and improved neurological status in 71 % of symptomatic vasospasm	Low
Muench et al. [53]	47	P	SAH	PAC HHH (NE)	To evaluate the effects of different components of HHH therapy on brain perfusion and oxygenation	Increased MAP but unchanged CI Increase in rCBF/PbO_2 only with HTN	Low
Mutoh et al. [109]	7	P	SAH	TT	To evaluate the effects of hyperdynamic therapy on brain oxygenation during symptomatic vasospasm	TT-guided therapy Increased rSO_2 during VSP	Very low
Levy et al. [54]	23	P	SAH	PAC Dobu	To evaluate the effects of dobutamine on neurological status	Increased CI improved neurological status during CV in 78 % of patients who failed to respond to NE	Low
Tanabe et al. [49]	10	R	SAH	PAC	To evaluate the effects of IV albumin on systemic hemodynamics	Increased CI improved neurological status during CV	Very low
Hadeishi et al. [48]	8	R	SAH	PAC Dobu	To evaluate the effects of dobutamine on neurological status	Increased CI improved neurological status during CV	Very low
Kim et al. [110]	16	P	SAH	PAC Dobu/Phenyl	To evaluate the effects of dobutamine and phenylephrine on neurological status	Both drugs increased CBF in patients with vasospasm	Very low
Miller et al. [111]	24	P	SAH	PAC Phenylephr	To evaluate the effects of phenylephrine on neurological status	Increased MAP did not result in CI changes—88 % of patients improved neurological status	Low
Naidech et al. [112]	11	R	SAH	PAC Dobu/Milri	To evaluate the effects of different inotropes on systemic hemodynamics	Milrinone was more effective to increase CI but was also associated with lower MAP	Very low
Impact of specific therapies dealing with optimization of CO on outcomes							
Tagami et al. [63]	1,482	R (b/a)	OHCA	TT-guided therapy	To assess the impact of TT-guided therapy on outcome of CA patients	Improved good neurological outcome	Low
Kim et al. [65]	453	P (b/a)	SAH	PAC	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	Reduced incidence of sepsis and pulmonary edema Reduced mortality (29 vs. 34 %, $p = 0.04$)	Moderate

Table 2 continued

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Mutoh et al. [76]	45	P	SAH	TT	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	4/8 DCI in patients with VSP No pulmonary edema or heart failure	Low
Vermeij et al. [113]	348	R (b/a)	SAH	PAC (VSP) HHH	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	Reduced mortality among patients with DCI	Low
Medlock et al. [67]	47	P	SAH	PAC Proph. HHH	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	Proph HHH did not prevent DNID 26 % incidence of PE	Low
Rondeau et al. [64]	41	RCT	SAH	TT	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	Dobu versus NE: similar VSP and DCI but lower MV duration and ICU stay	Moderate
Mutoh et al. [66]	116	RCT	SAH	PAC (late) TT	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	Reduced VSP, DCI, VSP-related infarctions, CV complications—Improved mRS	Moderate
Lennihan et al. [114]	82	RCT	SAH	PAC HV vs. NV	To evaluate the effects of hemodynamic monitoring on the occurrence of complications	HV did not increase CBF but raised filling pressures No differences in occurrence of VSP and DCI	Moderate

P prospective, *R* retrospective, *SAH* subarachnoid hemorrhage, *TBI* traumatic brain injury, *TT* transpulmonary thermodilution, *PE* pulmonary edema, *CO* cardiac output, *PCWA* pulse contour wave analysis, *LVEF* left ventricular ejection fraction, *NPE* neurogenic pulmonary edema, *NR* not reported, *CV* cerebral vasospasm, *CI* cardiac index, *PE* pulmonary edema, *IABP* intra-aortic balloon counterpulsation, *LVEF* low ventricular ejection fraction, *cTnI* troponin I, *GEDVI* global end-diastolic volume index, *GEF* global ejection fraction, *DCI* delayed cerebral infarction, *BNP* brain natriuretic peptide, *SV* stroke volume, *ECG* electrocardiogram, *VA* ventricular arrhythmias

Table 3 Studies evaluating preload in acute brain injury patients

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Role of preload monitoring to explain the mechanisms of brain injury-related cardiopulmonary complications							
Deehan et al. [37]	24	R	SAH	PAC	To assess effects of dobutamine	High variable PAOP in patients with PE	Very low
Watanabe et al. [33]	34	P	SAH	TT	To evaluate which hemodynamic variable was associated with the occurrence of PE	PE was associated with higher GEDVI DCI was associated with lower GEDVI	Very low
Mayer et al. [44]	72	R	SAH	Echography	To evaluate the impact of hemodynamic alterations on cerebral complications	PAOP was not associated with the development of DCI	Very low
Vespa et al. [38]	56	R	SAH	PAC	To evaluate the mechanisms of poor oxygenation after SAH	Increased ELVWI in patients with poor oxygenation	Very low
Touho et al. [43]	25	R	SAH	TT	To evaluate the mechanisms of poor oxygenation after SAH	Increased intrapulmonary shunt and ELWI were found in patients with poor oxygenation	Very low
Sato et al. [36]	49	P	SAH	TT	To assess variables related to the development of PE	Patients with PE had higher ELWI than others	Low
Verein et al. [115]	17	P	Stroke	TT	To assess the relationship between ELVWI and ICP or brainstem function	ELVWI was correlated with latency of auditory potentials	Very low
Role of preload monitoring to optimize therapy							
Bulters et al. [55]	71	RCT	SAH	PAC	To assess hemodynamic changes with IABP	PAOP-guided therapy resulted in increased CBF and CPP during IABP	Moderate
Mutoh et al. [109]	7	P	SAH	TT	To assess the effects of hyperdynamic therapy on cerebral oxygenation during s-VSP	Increased CO was associated with improved cerebral oxygenation	Very low
Preload monitoring findings and outcome							
Mutoh et al. [76]*	45	P	SAH	TT	To evaluate the effects of TT-guided therapy on DCI occurrence during VSP	4/8 DCI in patients with VSP No pulmonary edema or heart failure	Low
Kim et al. [65]	453	P (b/a)	SAH	PAC HHH vs. HD	To compare the effects of two therapeutic strategies on neurological outcomes	Reduced incidence of sepsis and pulmonary edema Reduced mortality	Moderate
Mutoh et al. [66]*	116	RCT	SAH	PAC (late) TT	To compare the effects of two therapeutic strategies on neurological outcomes	Reduced VSP, DCI, VSP-related infarctions, CV complications – Improved mRS	Moderate

P prospective, *R* retrospective, *SAH* subarachnoid hemorrhage, *TT* transpulmonary thermodilution, *PE* pulmonary edema, *LVEF = NPE* neurogenic pulmonary edema, *NR* not reported, *HHH* triple-H therapy, *PAC* pulmonary artery catheter, *HD* hyperdynamic therapy, *CI* cardiac index, *GEDV* global end-diastolic volume, *ELVWI* extravascular lung water index, *DCI* delayed cerebral ischemia, *VSP* vasospasm, *s-VSP* symptomatic vasospasm, *CV* cardiovascular, *PAOP* pulmonary artery occlusive pressure, *IABP* intra-aortic balloon counterpulsation

* TT-guided therapy consisted in optimizing CI, GEDV and reducing EVLWI

Table 4 Studies evaluating afterload in acute brain injury patients

Reference	Patient number	Study design	Group	Technique assessment	End-point	Findings	Quality of evidence
Hadeishi et al. [48]	8	R	SAH	PAC	To assess the effects of dobutamine to treat CV	Decreased SVR	Very low
Bulters et al. [55]	71	RCT	SAH	PAC	To assess hemodynamic changes with IABP	Higher SVR during IABP	Moderate
Watanabe et al. [33]	34	P	SAH	TT	To evaluate which hemodynamic variable was associated with the occurrence of DCI	DCI was associated with increased SVR	Low
Rzheutskaya et al. [100]	13	P	TBI	TT	To evaluate hemodynamic alterations after TBI	SVRI were used to identify four different patterns of hemodynamic status	Very low
Mayer et al. [44]	72	R	SAH	Echography	To assess the impact of cardiac injury on hemodynamic and cerebral complications after SAH	Higher SVRI were found in patients developing s-VSP	Very low

P prospective, *SAH* subarachnoid hemorrhage, *TT* transpulmonary thermodilution, *IABP* intra-aortic balloon counterpulsation, *DCI* delayed cerebral infarction, *SVR* systemic vascular resistances, *s-VSP* symptomatic vasospasm

Table 5 Studies evaluating fluid responsiveness (FR) in acute brain injury patients

Reference	Patient number	Study design	Group	Preload assessment	End-point	Findings	Quality of evidence
Berkenstadt et al. [20]	15	P	BS	SVV	To assess the accuracy of SVV to predict FR	SVV was a strong predictor of FR	Low
Li et al. [74]	48	P	BS	SVV	To assess the accuracy of SVV when compared to commonly used variables to predict FR	SVV was a strong predictor of FR	Low
Mutoh et al. [77]	16	P	SAH	SVV	To compare SVV with GEDVI to predict FR	SVV was a better predictor than GEDVI for FR	Moderate
Mutoh et al. [66]	116	RCT	SAH	GEDVI changes	To evaluate the changes in GEDVI vs. PAOP/CVP to predict FR	Only changes in GEDVI after fluid loading was associated with SV changes	Moderate
Moretti et al. [75]	29	P	SAH	dICV	To evaluate the changes in SVV vs. dICV to predict FR	SVV and dICV were both strong predictor of FR	Moderate
Deflandre et al. [82]	26	P	BS	ΔPP	To evaluate the changes in ΔPP vs. DD to predict FR	ΔPP and DD were both strong predictor of FR	Moderate

P prospective, *RCT* randomized clinical trial, *BS* brain surgery, *SAH* subarachnoid hemorrhage, *SVV* stroke volume variation, *GEDVI* global end-diastolic volume index, *dICV* distensibility of inferior vena cava, *ΔPP* pulse pressure variation

Table 6 Studies evaluating parameters of global perfusion in acute brain injury patients

Reference	Patient number	Study design	Group	GP assessment	End-point	Findings	Quality of evidence
Venous saturation							
Di Filippo et al. [68]	121	P	TBI	$ScvO_2$	To assess the prognostic value of $ScvO_2$ after TBI	$ScvO_2$ values were lower in non-survivors than in survivors ($p = 0.04$) but not independently predictor of mortality	Very low
Gaieski et al. [69]	38	R (b/a)	CA	$ScvO_2$ CTRL	To assess the impact of $ScvO_2$-guided therapy on outcome after CA	$ScvO_2$-guided therapy tended to a reduction in mortality	Low
Walters et al. [70]	55	P (b/a)	CA	$ScvO_2$ CTRL	To assess the impact of $ScvO_2$-guided therapy on outcome after CA	$ScvO_2$-guided therapy tended to an improved neurological outcome	Moderate
Lactate							
Donnino et al. [73]	79	R	CA	Lactate	To assess the prognostic value of lactate clearance after CA	Higher lactate clearance at 6, 12 and 24-h in survivors than non-survivors	Very low
Karagiannis et al. [116]	28	R	IHCA	Lactate	To assess the prognostic value of lactate clearance after CA	Lactate clearance was significantly lower in survivors than non-survivors	Very low
Kliegel et al. [72]	394	R	CA	Lactate	To assess the prognostic value of lactate levels and lactate clearance after CA	Lactate levels at 48 h were independently associated with poor neurological outcome	Very low
Lemiale et al. [45]	1,152	R	OHCA	Lactate	To assess the prognostic value of lactate after CA	Admission lactate was an independent predictor of ICU mortality	Very low
Starodub et al. [71]	199	R	OHCA IHCA	Lactate	To assess the prognostic value of lactate levels and lactate clearance after CA	Initial serum lactate and lactate clearance were not predictive of survival	Very low
Cocchi et al. [117]	128	R	OHCA	Lactate	To assess the prognostic value of lactate levels and vasopressors after CA	Vasopressor need and lactate levels could predict mortality	Very low
Oddo et al. [118]	88	P	CA	Lactate	To assess the prognostic value of several hospital variables after CA	Lactate on admission was an independent predictor of poor outcome	Low
Shinozaki et al. [119]	98	P	OHCA	Lactate	To assess the prognostic value of lactate after CA	Initial lactate level was independently associated with poor outcome Level >12 mmol/L predicted poor outcome (Sens. 90 % and Sp. 52 %)	Low
Mullner et al. [120]	167	R	OHCA	Lactate	To assess the prognostic value of lactate after CA	Initial lactate values were correlated with the duration of arrest and associated with poor outcome	Very low

Table 6 continued

Reference	Patient number	Study design	Group	GP assessment	End-point	Findings	Quality of evidence
Adrie et al. [121]	130	P	OHCA	Lactate	To identify clinical and laboratory variables that predict outcome after CA	Lactate on admission was an independent predictor of poor outcome	Low
Zhao et al. [122]	81	P	TBI	Lactate	To assess the effect of TH on lactate and glucose levels after TBI	TH reduced more rapidly lactate levels than normothermia	Low
Yatsushige et al. [123]	12	P	TBI	Lactate	To assess predictors of poor outcome after decompressive craniectomy	Lactate levels were independently associated with poor outcome	Very low
Meierhans et al. [124]	20	R	TBI	Lactate	To assess the effects of arterial lactate on brain metabolism	Blood lactate >2 mmol/L increased brain lactate and decreased brain glucose	Very low
Cureton et al. [125]	555	R	TBI	Lactate	The impact of lactate on neurological outcome	Increased lactate was associated with more severe head injury Patients with lactate >5 mmol/L had better outcome	Very low
Brouns et al. [126]	182	P	Stroke	Lactate	The impact of lactate on neurological outcome	Blood lactate was not associated with outcome	Low
Jo et al. [127]	292	R	Stroke	Lactate	The impact of initial lactate on neurological outcome	Initial lactate levels >2 mmol/L associated with poor outcome	Very low
ΔCO_2							
Tsaousi et al. [128]	51	P	BS	ΔCO_2	To assess the relationship between CI and ΔCO_2	Good correlation ($R^2 = 0.830$) between the two variables	Very low

P prospective, *R* retrospective, *CA* cardiac arrest, *OHCA* out-of-hospital CA, *IHCA* in-hospital CA, $ScvO_2$ central venous saturation, ΔCO_2 veno-arterial CO_2 difference, *BS* brain surgery, *CI* cardiac index, *TBI* traumatic brain injury, *TH* therapeutic hypothermia, *Sens.* sensitivity, *Spec.* specificity, *CTRL* control group

LVEF <50 vs. 5 % in others ($p < 0.001$). A higher incidence of RWMA among patients with cTnI ≥0.3 ng/mL was found in both early and late phases after SAH [30, 31]. Patients with high cTnI levels also had a higher incidence of diastolic dysfunction [22]. These alterations were associated with normal myocardial perfusion but altered sympathetic innervation [6, 32].

Since pulmonary complications are frequent after ABI, the role of altered cardiac function was evaluated in several studies. Patients who developed pulmonary edema (PE) after SAH had lower global ejection fraction (GEF) than did others [33]. Also, a higher incidence of systolic and diastolic dysfunction was found in patients with PE compared to others after SAH [22, 23]. Naidech et al. [34] reported no association between LV dysfunction and the occurrence of PE, while Junttila et al. [35] showed that the proportion of patients with LVEF <50 % was similar in patients developing neurogenic pulmonary edema (NPE) and those who did not (26 vs. 18 %, $p = 0.6$). Although patients with NPE often had more a restrictive profile on echography when compared to others (21 vs. 5 %, $p = 0.03$), filling pressures were similar between groups. Moreover, echocardiographic abnormalities could not predict development of NPE.

Cardiac Output

In patients with SAH, lower CI was reported in patients developing PE compared to others; [36] however, variable hemodynamics, including even high CI after SAH, were found in patients with PE in other studies [37–39].Three studies evaluated changes in CI during the use of therapeutic hypothermia (TH). Cooling resulted in lower heart rate, filling pressure, and CI without deleterious effects on global perfusion in OHCA [40]. TH improved systemic hemodynamics in CA survivors suffering from cardiogenic shock [41]. The use of hypothermia in patients with SAH decreased CI and increased artero-venous jugular difference in oxygen, suggesting a potential role for brain hypoperfusion in this setting [42].

Preload

In three studies, patients with PE or poor oxygenation had a higher ELWI than others [33, 36, 43]. Moreover, PE was associated with higher GEDVI after SAH in another study [33]. However, two other studies reported a poor predictive value of PAOP for PE development [37, 44].

Adequacy of Global Perfusion

Admission lactate levels were significantly higher in patients with shock after CA than the others and were an independent predictor of ICU mortality [45].

Does Hemodynamic Monitoring Have a Specific Role in Optimizing Brain Perfusion and Oxygenation or Brain-Specific Therapy?

Cardiac Output and Preload

Several studies have shown a relationship between the development of DCI and low CI in SAH patients [33, 44, 46], and different therapeutic interventions have been performed using systemic hemodynamic monitoring in these patients to optimize brain perfusion. Since CBF values are associated with CI [47], the use of a "hyperdynamic" approach (i.e., increase CI optimizing preload and inotropes to increase CBF, Fig. 2) has been observed to be associated with neurological improvement [48–52]. Alternatively, the more traditional "hypertensive" approach (i.e., increase MAP to increase CBF) is used to increase CPP in symptomatic vasospasm after SAH and can improve CBF and brain oxygenation in this setting [53]. Importantly, a "hyperdynamic" approach may still improve neurological status when patients with vasospasm fail to respond to norepinephrine [54].

Afterload

Hadeishi et al. [48] reported that in 8 SAH patients, in whom a PAC was used to optimize therapy for cerebral vasospasm, fluids, and inotropic agents induced an increase of CI (from 3.4 to 4.9 L/min m^2) with stable PAOP, while SVR decreased. Patients who develop DCI or symptomatic vasospasm have higher SVRI than others [33, 44]. Finally, the prophylactic use of IABP after SAH was associated with higher SVR than patients treated with a conventional approach but did not affect neurological outcome in this setting [55].

What is the Impact of Systemic Hemodynamic Monitoring and Related Therapies on Morbidity, Mortality, and Neurological Outcome?

Cardiac Function

Several studies have evaluated the impact of hemodynamic alterations on the morbidity and mortality after ABI. Patients with reduced LVEF are more likely to develop ventricular arrhythmias than others (29 vs. 13 %, $p = 0.12$) [56]. However, there is conflicting data exist on the association between LV dysfunction and survival or neurological outcome after SAH or CA [3, 14, 57–62].

Cardiac Output and Preload

Using a hemodynamic-guided therapy in out-of-hospital cardiac arrest (OHCA), Tagami et al. showed a significant

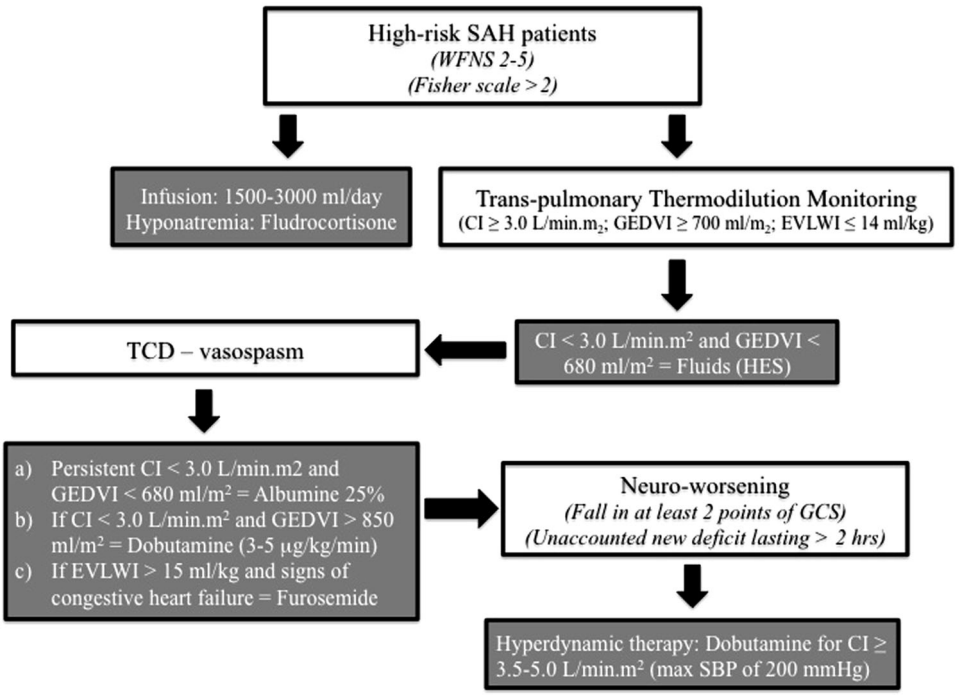

Fig. 2 Optimization of systemic hemodynamics in high-risk patients after subarachnoid hemorrhage (SAH). Adapted from [34, 35]

increase of favorable neurological outcome from 0.5 to 3.0 % when compared to an historical cohort, even after adjustment for confounders [63]. The use of such intervention to improve patients' management was an independent predictor of good outcome. In the sub-group of patients with witnessed ventricular fibrillation, the proportion of patients with good neurological outcome also significantly improved from 7.9 to 26.2 %. In SAH patients, "hyperdynamic" therapy does not always reduce the incidence of TCD-vasospasm or DCI, when compared to norepinephrine [64]. However, the duration of mechanical ventilation (median 8 vs. 19 days, $p = 0.01$) and ICU stay (11 vs. 21 days, $p = 0.01$) was less when using dobutamine than norepinephrine. ICU mortality was 18 % for dobutamine and 6 % for norepinephrine ($p = 0.33$). Kim et al. [65] showed that the occurrence of vasospasm, myocardial infarction, ARDS, and renal failure was similar when using invasive hemodynamic monitoring with PAC in SAH patients compared to non-monitored patients. However, the use of PAC was associated with a reduced incidence of pulmonary edema (6 vs. 14 %, $p = 0.003$) and sepsis (3 vs. 11 %, $p < 0.001$). Reduced 6-month mortality was observed using PAC (34 vs. 29 % $p = 0.04$). Others have observed that the use of TT was associated with significantly less TCD-vasospasm (50 vs. 66 %, $p = 0.03$), delayed neurological deficit (32 vs. 48 %, $p = 0.03$), and vasospasm-related infarctions (6 vs. 14 %, $p = 0.049$) than conventional therapy after SAH [66]. The use of TT also was associated with a reduced number of cardiopulmonary complications (from 12 to 2 %, $p = 0.01$) and reduced maximal daily fluid intake. Others have observed that the prophylactic use of hypertensive therapy does not reduce the incidence of delayed neurological deficit; instead it increases the occurrence of PE [67].

Adequacy of Global Perfusion

In TBI patients, $ScvO_2$ values are lower in non-survivors ($n = 22$; 67 ± 12 %) than in survivors ($n = 99$; 70 ± 9 %, $p = 0.04$). $ScvO_2 \leq 65$ % had a relative risk for increased mortality of 2.3 (95 % CI 1.1–4.8); however, $ScvO_2$ was not an independent predictor of mortality [68]. Gaieski et al. [69] showed that early goal-directed hemodynamic optimization (EGHO) of patients after CA using a target $ScvO_2$ of ≥ 65 % (Fig. 3) was associated with reduced mortality when compared with historical controls (10/20, 50 % vs.14/18, 78 %; $p = 0.15$). Similarly, Walters et al. [70] showed better neurological outcome in patients treated with EGHO and TH when compared to historical controls (31 vs. 12 %, $p = 0.08$).

Several studies have described lactate levels in CA patients with varied results. Starodub et al. [71] observed that initial serum lactate (divided in three groups; <5 mmol/L; 5–10 mmol/L; >10 mmol/L) was not associated with mortality. An association with serial lactate levels or lactate clearance and outcome is observed. For example, Kliegel et al. observed that lactate levels were

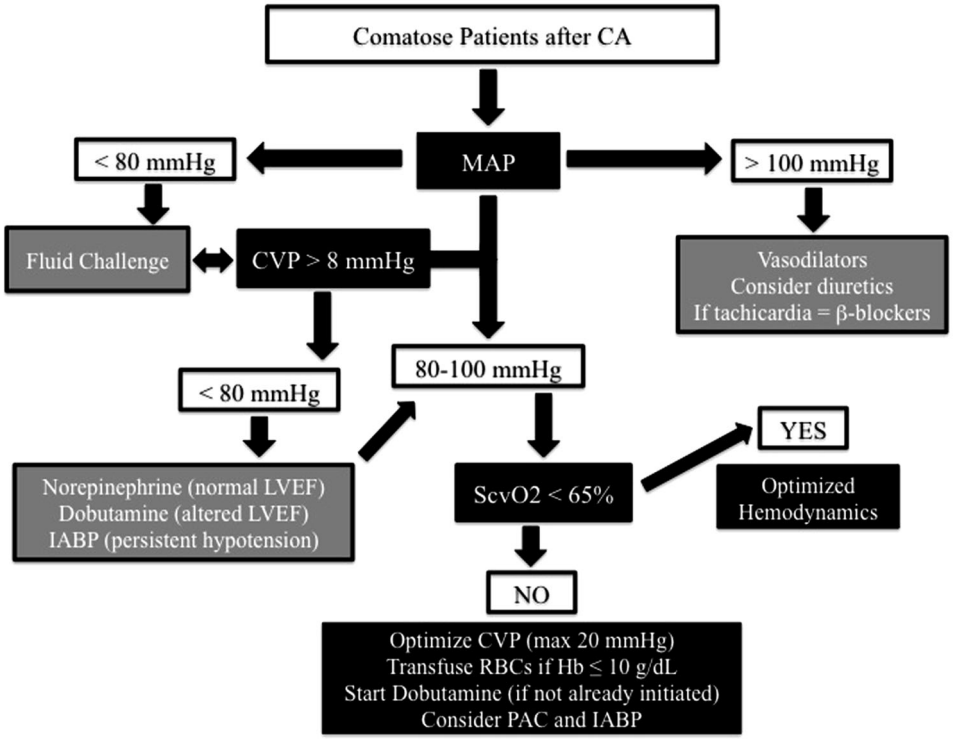

Fig. 3 Optimization of systemic hemodynamics in comatose patients after cardiac arrest. Adapted from [81]

significantly higher in non-survivors than survivors on admission, at 24 and 48 h in patients surviving OHCA [72]. However, lactate levels returned within normal ranges within 24 h in most of patients. A higher proportion of non-survivors had hyperlactatemia (defined as lactate >2 mmol/L) at 48 h than survivors (31 vs. 14 %, $p < 0.001$), and lactate levels at 48 h were independently associated with mortality. In the same study, similar results were reported when patients were categorized as good ($n = 161$) or poor ($n = 233$) neurological outcome. Some studies have shown that, rather than measuring admission lactate levels, the rate of decline in lactate concentration, the so-called "lactate clearance" reflects the improvement of global perfusion during therapy. A higher lactate clearance is independently associated with good outcome in this setting [73].

How Can Fluid Responsiveness be Assessed in ABI Patients?

Berkenstadt et al. [20] studied stroke volume variation in 15 patients undergoing elective neurosurgery; 140 fluid loadings were performed. Half of them were associated with fluid responsiveness (FR). Li et al. [74] examined FR in 48 patients undergoing brain surgery, and SVV was the best predictor of FR (defined as an increase of stroke volume >10 %), while other common hemodynamic variables, including MAP, CVP, and CO, did not discriminate between fluid responders and non-responders. Moretti et al. [75] showed that the inferior vena cava distensibility (dIVC) assessed on echocardiography was a strong predictor of FR in patients with SAH, with an AUC of 0.902 (95 % CI 0.73–0.98). A dIVC >16 % yielded a sensitivity of 71 % and a specificity of 100 % to predict FR. After SAH, changes in GEDVI after fluid loading are associated with changes in SV, while changes in PAOP and CVP were not [66]. The GEDVI had an AUC of 0.73 to predict FR (defined as an increase of stroke volume >10 %) in this setting.

What Hemodynamic Monitoring is Indicated in ABI Patients, in Particular to Diagnose and Support the Management of Unstable or At-Risk Patients?

Eight studies ($n = 458$) reported data on a specific technique/device to monitor CO and 3 ($n = 71$) to monitor FR in ABI patients. No study compared different parameters of preload, afterload, or global perfusion in patients with ABI. The main advantages and disadvantages of different monitoring devices are listed in Table 7.

In SAH patients receiving hyperdynamic therapy, the PCWA technique has good accuracy to measure CI when TT was used as reference; [76] the coefficient of agreement was 0.77, and the bias was 0.33 L/min m^2 with a percentage of error of 15 %. The incidence of side effects, cerebral infarction, maximal dobutamine doses, and neurological outcome was similar when patients were

Table 7 Available techniques used for hemodynamic monitoring in patients with acute brain injury and their potential advantages and disadvantages

Techniques	Cardiac output	LV function	Preload	Fluid responsiveness	Afterload	Advantages	Disadvantages
PAC	+	−(LV) +(RV)	+	−	+	Measure of PAP, PAOP Measure of SvO_2	Invasiveness not beat-by-beat analysis
Trans-pulmonary thermodilution[a]	+	+	+	+	+	Less invasive No need for PAC positioning	Requires a specific femoral arterial catheter not beat-by-beat analysis
External+internal calibrated PCM[a]	+	+	−	+	+	Continuous CO monitoring continuous $ScvO_2$ (optional)	Recalibration every 4-6 h requires a specific femoral arterial catheter
Internal-calibrated PCM[b]	+	−	−	+	+	Continuous CO monitoring continuous $ScvO_2$ (optional) mini-invasive	Less accuracy for CO sensitive to SVR requires specific catheter
Non-calibrated PCM[c]	+	+	−	+	+	Continuous CO monitoring No need for dedicated catheter Mini-invasive	Few data available Less accuracy for CO (?) requires optimal arterial pressure tracing
Echocardiography	+	+	+	+	−	Visualization of the heart	Intermittent use
						estimate for filling pressure	requires adequate training

PAC pulmonary artery catheter, *PCM* pulse contour method, *$ScvO_2$* central venous oxygen saturation, *SvO2* mixed venous oxygen saturation, *SVR* systemic vascular resistances, *CO* cardiac output, *PAP* pulmonary artery pressure, *PAOP* pulmonary artery occlusive pressure, *LV* left ventricle, *RV* right ventricle

+ Possible; − not feasible

[a] PiCCO device (Pulsion Medical Systems, Irving, TX, USA)

[b] FloTrac Vigileo device (Edwards, Irvine, CA, USA)

[c] MostCare-PRAM device (Vygon, Padova, Italy)

Table 8 Differences among different monitoring techniques for cardiac output (CO) in acute brain injury patients

Reference	Patient number	Study design	Group	Technique assessment	Findings	Quality of evidence
Franchi et al. [129]	121	P	TBI	PCA	CO: correlation 0.94; bias 0.06 L/min:	Moderate
				PCWA	PE 18 %	
Mutoh et al. [76]	45	P	SAH	PCWA TT	CI: correlation 0.77; bias 0.33 L/min m^2; PE 15 %.	Moderate
Mutoh et al. [66]	116	RCT	SAH	PAC TT	CI: correlation 0.78; bias 0.05 L/min m^2; PE 14 %	High
Mutoh et al. [77]	16	P	SAH	PCWA TT	CI: correlation 0.82; bias was 0.57 L/min m^2;	Moderate
					PE 25 % and higher during MV	
Junttila et al. [78]	16	P	BS	PCWA	CO: bias 1.7 L/min; PE 45 %.	Moderate
				PAC	Larger bias during NE and NIMO therapy	
					Significant correlation SVR/bias	
Haenggi et al. [80]	8	P	OHCA	PCWA	CO: bias 0.23 L/min, PE 34 %	Moderate
				PAC	No differences between TH and NT	
Tagami et al. [81]	88	P	CA	TT	Coefficient of error <10 % (3 injections)	Moderate
Mayer et al. [79]	48	P	SAH	Echography	CO: correlation 0.67; bias 0.75 L/min; precision 1.34 L/min;	Moderate
				PAC	Echography underestimated PAC-derived CO	

P prospective, *R* retrospective, *RCT* randomized clinical trial, *TBI* traumatic brain injury, *SAH* subarachnoid hemorrhage, *OHCA* out-of-hospital cardiac arrest, *TT* transpulmonary thermodilution, *PCWA* pulse contour wave analysis, *PAC* pulmonary artery catheter, *CO* cardiac output, *CI* cardiac index, *PE* percentage of error, *NE* norepinephrine, *NIMO* nimodipine, *SVR* systemic vascular resistances, *TH* therapeutic hypothermia, *NT* normothermia

managed with one of the two techniques. In patients with SAH treated with a hyperdynamic approach, CI measured by TT showed a good correlation ($r^2 = 0.78$) [66] with the PAC measurements, with a bias 0.05 L/min m^2, a precision of 0.11 L/min m^2 and a percentage of error of 14 %. In SAH patients undergoing brain surgery, PCWA-measured CI showed a good correlation with TT ($r = 0.82$); The percentage of error was higher when measurements were collected during mechanical ventilation than on spontaneous breathing [77]. In patients undergoing brain surgery, an important bias on CO measurement was reported (1.7 L/min), with limits of agreement of −2.4 to 5.4 L/min when a PCWA was compared to TT [78]. The percentage of error was 45 %. The bias was larger in those patients receiving norepinephrine and nimodipine, but not in those receiving dobutamine (Table 8). There was a significant negative correlation between SVR and bias. Others have observed a poor agreement between echocardiography, and PAC was reported to measure CO in SAH patients receiving aggressive fluid therapy [79]. After CA, TT, and not PCWA devices, appears to be a reliable technique to assess CO during TH [80, 81]. Finally other studies show that ΔPP or SVV was equivalent to assess FR in ABI patients [75, 82].

Acknowledgments We would like to thank Christos Lazaridis and Sabino Scoletta for their fruitful comments on this manuscript.

Conflict of interest Giuseppe Citerio receives speaker honoraria from Codman and has received research funding from Italian Governative agencies (AIFA, Ministero Salute, Regione Lombardia). Fabio Taccone none to declare.

Appendix: Participants in the International Multidisciplinary Consensus Conference on Multimodality Monitoring

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