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REPORTS OF ORIGINAL INVESTIGATIONS

Pulmonary artery catheterization in patients with cardiogenic shock: a systematic review and meta-analysis

Cathétérisme de l'artère pulmonaire chez des patients en choc cardiogénique : une revue systématique et une méta-analyse

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Abstract

Purpose *Cardiogenic shock carries high morbidity and mortality. The purpose of this review was to determine the safety and efficacy of pulmonary artery catheterization (PAC) in adult patients hospitalized with cardiogenic shock.*

Source *We performed a systematic review and meta-analysis of observational studies and randomized controlled trials comparing PAC vs no PAC in cardiogenic shock. We searched MEDLINE, EMBASE, Cochrane CENTRAL, and grey literature. We screened articles, abstracted data, and evaluated risk of bias in duplicate. We pooled data using a random-effects model and evaluated the quality of evidence using the GRADE framework. Outcomes of interest were mortality, length of stay, and procedural complications.*

Principal findings *We identified 19 eligible observational studies (≥ 2,716,287 patients) and no randomized controlled trials; 14 studies were at high risk of bias (lack of adjustment for prognostic variables and/or co-interventions). When pooling adjusted results, PAC was associated with improved survival to hospital discharge (relative risk [RR], 0.77; 95% confidence interval [CI], 0.64 to 0.91, I^2 = 98%; very low-quality evidence) and at longest available follow-up (RR, 0.72; 95% CI, 0.60 to 0.87; I^2 = 99%; very low-quality evidence). Unadjusted length of stay was 3.5 days longer (95% CI, 1.49 to 5.54; I^2 = 100%; very low-quality evidence) with PAC. Procedural complications were inconsistently reported.*

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Conclusions *Very low-quality observational evidence suggests PAC use in patients with cardiogenic shock is associated with lower mortality. Overall, these results support consideration of PAC for hemodynamic assessment in cardiogenic shock. Prospective randomized clinical trials are needed to further characterize the role of PAC in this population.*

Résumé

Objectif *Le choc cardiogénique entraîne une morbidité et une mortalité élevées. Le but de cette revue était de déterminer la sécurité et l'efficacité de l'utilisation d'un cathétérisme de l'artère pulmonaire (CAP) chez des patients adultes hospitalisés en choc cardiogénique.*

Sources *Nous avons réalisé une revue systématique et une méta-analyse d'études observationnelles et d'études randomisées contrôlées comparant l'utilisation vs la non-utilisation de CAP pour le traitement d'un choc cardiogénique. Nous avons effectué des recherches dans les bases de données MEDLINE, EMBASE, Cochrane CENTRAL et dans la littérature grise. Nous avons examiné les articles, résumé les données et évalué le risque de biais à deux reprises. Nous avons regroupé les données à l'aide d'un modèle à effets aléatoires et évalué la qualité des données probantes en nous appuyant sur le système GRADE. Les issues d'intérêt étaient la mortalité, la durée de séjour et les complications procédurales.*

Constatations principales *Nous avons identifié 19 études observationnelles admissibles (≥ 2 716 287 patients) et aucune étude randomisée contrôlée; 14 études comportaient un risque élevé de biais (absence d'ajustement sur les variables pronostiques et/ou les interventions concomitantes). En regroupant les résultats ajustés, le CAP a été associé à une meilleure survie jusqu'au congé de l'hôpital (risque relatif [RR], 0,77; intervalle de confiance [IC] à 95 %, 0,64 à 0,91, I2 = 98 %; données probantes de très faible qualité) et jusqu'au point de suivi disponible rapporté le plus lointain dans le temps (RR, 0,72; IC 95 %, 0,60 à 0,87; I2 = 99 %; données probantes de très faible qualité). La durée de séjour non ajustée était 3,5 jours plus longue (IC 95 %, 1,49 à 5,54; I2 = 100 %; données probantes de très faible qualité) avec un CAP. Les complications procédurales n'étaient par rapportées de manière uniforme.*

Conclusion *Des données observationnelles de très faible qualité suggèrent que l'utilisation d'un CAP chez des patients en choc cardiogénique est associée à une réduction de la mortalité. Dans l'ensemble, ces résultats suggèrent de considérer le CAP pour l'évaluation hémodynamique en cas de choc cardiogénique. Des études cliniques randomisées prospectives sont nécessaires pour mieux caractériser le rôle du CAP dans cette population.*

Keywords Cardiogenic shock · pulmonary artery catheterization · right heart catheterization · hemodynamic assessment · acute heart failure · meta-analysis

Cardiogenic shock is a feared complication of decompensated heart failure (HF). Despite advances in HF therapies, such as revascularization and mechanical circulatory support (MCS), in-hospital mortality for patients with cardiogenic shock remains high, approaching 50%.[1,2]

Invasive hemodynamic assessment with pulmonary artery catheterization (PAC) allows clinicians to quantify cardiac output, assess intracardiac pressures, differentiate between etiologies of shock, and titrate guideline-directed HF therapies.[3] Nevertheless, routine use of PAC in critically ill patients has declined considerably after randomized evidence from other populations—including results of the ESCAPE trial, which studied patients with decompensated HF without cardiogenic shock—failed to show improved outcomes with its use.[4–8] The availability of non-invasive modalities of hemodynamic assessment, such as echocardiography, has also contributed to decreasing PAC use.[9,10]

The role of PAC in cardiogenic, mixed, or undifferentiated shock remains uncertain; these groups may derive the most benefit from invasive hemodynamic assessment to guide therapy. Based on limited evidence, international societies continue to recommend PAC when patients with cardiogenic shock fail to respond to initial medical therapy (Electronic Supplementary Material [ESM] eTable 1).[11–14] We performed a systematic review and meta-analysis of studies evaluating the safety and efficacy—pertaining to mortality and length of stay—of PAC in patients with cardiogenic shock.

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Methods

We adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement.[15] We pre-registered the protocol with PROSPERO (CRD42019134025).

Study selection

Studies were eligible if: 1) the population consisted of hospitalized adults with a diagnosis of cardiogenic shock as defined by study authors in the spirit of the parameters used in the SHOCK and IABP-SHOCK II trials (see ESM eTable 2);[16,17] 2) the study design was a randomized controlled trial or comparative observational study; 3) the intervention group received PAC and the comparator group did not; and 4) the outcomes included one or more of the following: mortality (primary outcome), hospital and intensive care unit (ICU) length of stay, and procedural complications (e.g., infection, pneumothorax, arryhthmia, vascular injury).

Search strategy

We searched MEDLINE, CENTRAL, and EMBASE from inception to November 2020, as well as relevant unpublished grey literature including clinical trial registries and major conference proceedings from the past three years (search strategy found in ESM eAppendix). We screened citations of potentially eligible articles without language or publication date restrictions. Two reviewers independently screened titles and abstracts to identify potentially eligible articles for full-text review. Subsequent full-text review was also performed in duplicate. Disagreements between reviewers were resolved by consensus, and if necessary, involvement of a third-party adjudicator.

Data extraction

Two reviewers independently extracted pertinent data from included studies using pre-piloted data collection forms. Disagreements were resolved by consensus. We contacted the corresponding authors if relevant outcome data were missing.

Risk of bias assessment

Two reviewers assessed risk of bias independently and in duplicate using tools developed by the Clinical Advances Through Research and Information Translation (CLARITY) group.[18] Studies were judged as having low, unclear, or high risk of bias on the following domains: selection bias, assessment of exposure, assessment of outcomes, adjustment for prognostic variables, assessment for presence/absence of prognostic factors, adequacy of follow-up, and assessment of co-interventions. The overall risk of bias for each included study was categorized as "low" if the risk of bias was low in all domains, "unclear" if the risk of bias was unclear in at least one domain and with no high risk of bias domain, or "high" if the risk of bias was high in at least one domain. Disagreements were resolved by consensus.

Quality of evidence

We used the Grading of Recommendations, Assessment, Development and Evaluations (GRADE) method[19] to assess the overall quality of evidence for each outcome.

Statistical analysis

We analyzed data using RevMan software (Review Manager, version 5.3; The Nordic Cochrane Centre, The Cochrane Collaboration, Copenhagen, Denmark). We pooled both adjusted and unadjusted results using a random-effects model. Study weights were estimated using the generic inverse variance method[20] for adjusted outcomes and the method of DerSimonian and Laird[21] for unadjusted outcomes. We report pooled relative risks (RRs) for dichotomous outcomes and mean differences (MDs) for continuous outcomes with corresponding 95% confidence intervals (CIs). We assessed statistical heterogeneity using Chi square and I^2 statistics,[22] with substantial heterogeneity predefined as $P < 0.10$ or $I^2 > 50\%$.

We inspected funnel plots and performed Egger's tests to assess for publication bias.[23] We explored heterogeneity between studies by performing meta-analyses of predetermined clinically relevant subgroups and comparing effect estimates in RevMan. Subgroups included risk of bias (low, unclear, or high), HF hospitalization status (never hospitalized *vs* $\geq$ 1 prior HF hospitalization), cause of cardiogenic shock (acute coronary syndrome, post cardiac surgery, arrhythmia, cardiomyopathy, or not specified), cardiogenic shock post-arrest (or not), and inpatient critical care location (ICU *vs* cardiac care unit) (ESM eTable 3).

Results

Screening process

Of 833 unique citations, 121 underwent full-text review and 19 studies including $\geq$ 2,716,287 patients met the

eligibility criteria (flow diagram in ESM eFig. 1). Two studies[24,25] included in this review did not adequately report breakdown of PAC and no PAC groups and could not be included in the meta-analysis.

Study characteristics

Table 1 describes characteristics of the included studies. Eleven were published peer-reviewed journal articles[26–36] and eight were conference proceedings.[24,25,37–42] We unsuccessfully sought additional data and/or full publications from the authors of two included studies.[24,25]

Fifteen articles were retrospective cohort studies[24,26–30,34–42] and four were prospective cohort studies;[25,31–33] no randomized controlled trials met eligibility criteria. One study focused on post cardiac surgery patients[39] and seven reported specifically on patients with acute coronary syndromes.[26,28,30,34,35,37,40] Length of follow-up varied from index hospitalization up to five years.

Patient characteristics

Nine studies[27–29,31,32,35,36,38,41] described patient characteristics on admission (Table 2). Patients who received PAC tended to be younger and more often male compared with those that did not. With the exception of one included study,[36] comorbidities were similar between groups. Four studies[31,32,36,38] reported on etiologies of cardiogenic shock; acute coronary syndrome was the most common.

Eight studies[27,29,31,32,35,36,40,41] described interventions during hospitalization as shown in Table 3. Most patients with cardiogenic shock who underwent revascularization received percutaneous coronary intervention. With the exception of one study,[36] patients who received PAC were more likely to receive MCS, vasopressors/inotropes, and mechanical ventilation and require renal replacement therapy.

Risk of bias

We judged risk of bias to be low in two studies,[31,33] moderate/unclear in three studies,[28,38,39] and high in 14 studies[24–27,29,30,32,34–37,40–42] (see Fig. 1). Domains accounting for the most judgements of high or unclear risk of bias were adjustment for prognostic variables (11 studies) and assessment of co-interventions (17 studies).

Mortality

We included 17 studies (n = 2,716,206 patients) reporting mortality in our meta-analysis. We evaluated mortality at longest available follow-up, 30-day mortality, and in-hospital mortality. One article reported 28-day mortality;[39] we included it in analyses for 30-day mortality.

Pulmonary artery catheterization use was associated with significantly reduced mortality at longest available follow-up for both adjusted (Fig. 2, panel A) and unadjusted results. Nevertheless, there was marked heterogeneity in these results, with all I^2 values $\geq$ 98%. In the adjusted pooled estimate (RR, 0.72; 95% CI, 0.60 to 0.87; I^2 = 99%; P = 0.005), eight out of 13 studies[27,29,33,35,36,38,40,42] reported in-hospital mortality as the longest follow-up. PAC use was not associated with improved 30-day survival (RR, 0.62; 95% CI, 0.28 to 1.33; I^2 = 98%; P = 0.22) but was associated with reduced in-hospital mortality (RR, 0.77; 95% CI, 0.64 to 0.91; I^2 = 98%; P = 0.003) (Fig. 2, panel B). The ESM (eFigs 2 to 5) presents charts depicting unadjusted outcomes and 30-day mortality data.

Length of stay

Six studies (n = 1,378,959) reported on hospital length of stay.[28,29,32,35,40,41] Pooled unadjusted meta-analysis showed a mean increase of 3.5 days (95% CI, 1.49 to 5.54; I^2 = 100%; P = 0.0007) in patients who received PAC (Fig. 3). No studies reported ICU length of stay.

Subgroups and investigation for sources of heterogeneity

Insufficient detail was presented in the included studies to explore the effect of HF hospitalization(s), post-arrest status, or inpatient critical care location on heterogeneity. Analysis by risk of bias did not show a significant difference in effect size across subgroups (ESM, eFig. 6A/B). Although Fig. 2A suggests a subgroup difference pertaining to the etiology of CS, we note that this interpretation is limited by 1) having only one study of patients post cardiac surgery, and 2) the high likelihood that many patients in the "etiology of cardiogenic shock not specified" subgroup having CS due to acute coronary syndromes although not explicitly stated. Accordingly, we felt that our prespecified subgroup analyses did not identify any clear sources of heterogeneity.

Procedural access complications

Data were insufficient to perform a pooled quantitative analysis for procedural access complications. Three studies described complications. In Sionis (2019), seven of 71 patients (9.9%) experienced complications:[32] four patients had ventricular tachycardia (VT) during PAC insertion, two of which required cardioversion and three patients

Table 1 Summary and characteristics of included studies (19 studies)

Study ID	n (%)	Design	Country	Population	Inclusion and exclusion criteria	Definition of cardiogenic shock	Length of follow-up	Mortality n (%)
Ashraf (2020)[40]	20,758 PAC 1,892 (9.1) No PAC 18,866 (91.9)	Retrospective cohort Adjusted (method not described)	USA	National Inpatient Sample (2005–2014)	Inclusion: Patients with AMI-CS who underwent revascularization (percutaneous coronary intervention or thrombolysis) and received mechanical circulatory support Exclusion: Not provided	Not provided	Duration of hospitalization	PAC 590 (31.2) No PAC 5,399 (28.6)
Cohen (2005)[26]	11,817 PAC *vs* no PAC cohort not provided within subgroup of cardiogenic shock	Retrospective cohort Adjusted (causal inference model)	USA	Patients with ACS from GUSTO IIb and GUSTO III trials Cardiogenic shock cohort provided as a subgroup	Inclusion: Patients with ACS that were part of GUSTO IIb and GUSTO III trials Exclusion: Any patient referred for CABG on day of or before PAC	Not provided	30 days	Not provided
Doshi (2018)[27]	842,369 PAC 71,452 (8.5) No PAC 770,917 (91.5)	Retrospective cohort Adjusted (multivariable logistic regression)	USA	National Inpatient Sample (2005–2014)	Inclusion: Hospitalized patients aged ≥18, diagnosis of cardiogenic shock Exclusion: Age < 18 yr	Not provided	Duration of hospitalization	PAC 24,222 (33.9) No PAC 299,115 (38.8)
Fernandez (2013)[39]	895 PAC 564 (63) No PAC 331 (37)	Retrospective cohort Adjusted (multivariable logistic and Cox regression)	Spain	Patients in ICU post cardiac surgery	Inclusion: Patients with low cardiac output syndrome who underwent cardiac surgery in ICU between 2003 and 2011 Exclusion: Not provided	CI < 2.2 as measured by PAC or FloTrac®/Vigileo® device	28 days	PAC 102 (18.1) No PAC 66 (19.9)
Garan (2020)[36]	1414 PAC 598 (42.4) No PAC 260 (18.4) 556 patients had incomplete PAC data and were excluded from analyses	Retrospective cohort Adjusted (multivariable logistic regression)	USA	Cardiogenic Shock Working Group registry (2016–2019)	Inclusion: Diagnosis of cardiogenic shock Exclusion: Age < 18 yr and those with unknown morality status at hospital discharge	SBP < 90 mm Hg for ≥ 30 min OR Use of vasoactive agents and/or a cardiac index value of < 2.2 $L \cdot min^{-1} \cdot m^{-2}$ determined to be secondary to cardiac dysfunction, in the absence of hypovolemia OR Use of mechanical circulatory support for suspected CS	Duration of hospitalization	Not provided

Table 1 continued

Study ID	n (%)	Design	Country	Population	Inclusion and exclusion criteria	Definition of cardiogenic shock	Length of follow-up	Mortalityn (%)
Gore (1987)[28]	441 PAC 231 (52.4) No PAC 210 (47.6)	Retrospective cohort	USA	Inpatients in 16 general hospitals in Worcester Standard Metropolitan Statistical Area (1975, 1978, 1981, 1984) Cardiogenic shock cohort provided as a subgroup	Inclusion: Discharge diagnosis acute myocardial infarction, living inside Worcester Metropolitan Area Exclusion: Not provided	SBP < 80 mm Hg, cyanosis, cold extremities, CHF and persistent oliguria	5 years	PAC 16/31 (52) No PAC 11/23 (48) Raw mortality data only provided for a subset of study patients
Ha (2018)[37]	89,718 PAC 5,503 (6.1) No PAC 84,215 (93.9)	Retrospective cohort Adjusted (method not described)	USA	National Inpatient Sample (dates not provided)	Inclusion: Patients with ACS who underwent coronary angiography Exclusion: Non-cardiogenic shock, patients who underwent cardiac surgery, patients with missing data	Not provided	Duration of hospitalization	PAC 1,920 (34.9) No PAC 24,759 (29.4)
Hernandez (2019)[29]	915,416 PAC 79,682 (8.7) No PAC 835,734 (91.3)	Retrospective cohort Adjusted (propensity-score matching)	USA	National Inpatient Sample (2004–2014) Cardiogenic shock cohort provided as a subgroup	Inclusion: Adults admitted with primary diagnosis of heart failure. Patients with cardiogenic shock provided as a separate cohort Exclusion: Right heart catheterization in catheterization lab, age <18, missing mortality data	Not provided	Duration of hospitalization	PAC 27,968 (35.1) No PAC 327,608 (39.2)
Isseh (2020)[25]	81 PAC *vs* no PAC cohort not provided	Prospective cohort	USA	Not provided	Inclusion: Patients with worsening CS requiring temporary mechanical circulatory support escalation. Worsening CS was defined as persistent hypotension, increasing doses of vasopressors/inotropes, worsening end-organ perfusion parameters, and/or worsening invasive hemodynamics. Exclusion: Not provided	Not provided	Duration of hospitalization	Not provided

Table 1 continued

Study ID	n (%)	Design	Country	Population	Inclusion and exclusion criteria	Definition of cardiogenic shock	Length of follow-up	Mortalityn (%)
Masoomi (2016)[24]	Not provided	Retrospective cohort	USA	National Inpatient Sample (2008–2012) Cardiogenic shock cohort provided as a subgroup	Inclusion: Adults >18 yr, primary diagnosis of CHF Exclusion: Not provided	Not provided	Duration of hospitalization	Not provided
Oliveros (2020)[41]	78,511 PAC 6,107 (7.8) No PAC 72,404 (92.2)	Retrospective cohort	USA	National Inpatient Sample (2009–2014)	Inclusion: Adults with diagnosis of cardiogenic shock Exclusion: Not provided	Not provided	Duration of hospitalization	PAC 1,515 (24.8) No PAC 24,762 (34.2)
O' Neill (2018)[30]	13,984 PAC 5,217 (37.3) No PAC 8,767 (62.7)	Retrospective cohort	USA	Inpatients at multiple US hospitals admitted with AMI-CS who received Impella (2009–2016)	Inclusion: Inpatients admitted with AMI-CS who received Impella – enrolled in IQ registry Exclusion: Not provided	SBP $\leq$ 90 mm Hg OR need for vasopressors to maintain SBP $\geq$ 90 mm Hg	Survival to explantation	PAC 1,930 (37.0) No PAC 4,471 (51.0)
Ranka (2020)[42]	269,475 PAC 25,840 (9.6) No PAC 243,635 (90.4)	Retrospective cohort Adjusted (method not described)	USA	Nationwide Readmissions Database (NRD) (2016–2017)	Inclusion: Hospitalization with CS Exclusion: Not provided	Not provided	Duration of hospitilization	PAC 6,667 (25.8) No PAC 96,236 (39.5)
Rossello (2017)[31]	129 PAC 83 (64) No PAC 46 (36)	Prospective cohort Adjusted (multivariable and Cox regression)	Spain	Single centre CCU inpatients	Inclusion: All patients admitted to CCU from December 2005 to May 2009 with diagnosis of CS Exclusion: Not provided	SBP < 90 mm Hg (in absence of hypovolemia and after adequate fluid challenge) for 30 min OR need for vasopressors to maintain adequate perfusion pressure AND $\geq$ 2 symptoms and/or signs of hypoperfusion (altered mental status/confusion, cold periphery, oliguria)	63 months	PAC 45 (54) No PAC 35 (76)
Sidhu (2017)[38]	106,258 PAC 7,440 (7) No PAC 98,818 (93)	Retrospective cohort Adjusted (propensity-score matching)	USA	National Inpatient Sample (2010–2014)	Inclusion: Diagnosis of cardiogenic shock Exclusion: Not provided	Not provided	Duration of hospitalization	PAC 2,254 (30.3) No PAC 36,957 (37.4)

Table 1 continued

Study ID	n (%)	Design	Country	Population	Inclusion and exclusion criteria	Definition of cardiogenic shock	Length of follow-up	Mortalityn (%)
Sionis (2019)[32]	219 PAC 82 (37) No PAC 137 (63)	Prospective cohort Adjusted (propensity-score matching)	Spain	Inpatients in 9 hospitals in 8 European countries (Czech Republic, Denmark, Finland, Greece, Italy, Poland, Portugal, Spain) from Oct 2010 to Dec 2012	Inclusion: Age $\geq$ 18 within 6 hr of diagnosis of cardiogenic shock, hypotension or low output syndrome Exclusion: Shock post cardiac or noncardiac surgery, hemodynamically significant arrhythmia as cause of hypotension	SBP < 90 mm Hg (in absence of hypovolemia and after adequate fluid challenge) for 30 min OR need for vasopressors to maintain SBP > 90 mm Hg AND Symptoms and/or signs of systemic and/or pulmonary congestion AND Symptoms and/or signs of hypoperfusion (altered mental status/confusion, cold periphery, oliguria < 0.5 $mL \cdot kg^{-1} \cdot hr^{-1}$ for 6 hr, lactate > 2 $mmol \cdot L^{-1}$)	30 days	PAC 37 (45) No PAC 57 (42)
Sotomi (2014)[33]	220 PAC 108 (49.1) No PAC 112 (50.9)	Prospective cohort Adjusted (propensity-score matching)	Japan	Inpatients enrolled in the multicentre ATTEND registry from April 2007 to December 2011 Patients requiring inotropic support provided as a subgroup	Inclusion: Acute heart failure syndromes who met modified Framingham criteria on admission Exclusion: Those considered unsuitable by attending physician, age < 20 yr, patients admitted with ACS, patients requiring mechanical circulatory support	Not provided	30 days	PAC 5 (4.6) No PAC 23 (20.5)
Vallabhajosyula (2020)[35]	364,001 PAC 29,609 (8.1) No PAC 334,392 (91.9)	Retrospective cohort Adjusted (propensity-score matching)	USA	National Inpatient Sample (2000–2014)	Inclusion: Patients with diagnosis of AMI-CS Exclusion: Missing in-hospital mortality data, admissions that ultimately required cardiac surgery	Not provided	Duration of hospitalization	PAC 13,709 (46.3) No PAC 140,445 (42.0)

Table 1 continued

Study ID	n (%)	Design	Country	Population	Inclusion and exclusion criteria	Definition of cardiogenic shock	Length of follow-up	Mortalityn (%)
Zion (1990)[34]	581 PAC 154 (26.5) No PAC 427 (73.5)	Retrospective cohort	Israel	Inpatients admitted to 14 CCUs in Israel as part of SPRINT registry Cardiogenic shock cohort provided as a subgroup	Inclusion: Patients admitted with ACS included in the SPRINT registry Exclusion: Not provided	Not provided	Duration of hospitalization	PAC 138 (89.6) No PAC 388 (90.9)

ACS = acute coronary syndrome; AMI-CS = acute myocardial infarction and cardiogenic shock; ATTEND registry = Acute Decompensated Heart Failure Syndromes registry; CABG = coronary artery bypass graft; CCU = cardiac care unit; CHF = congestive heart failure; CI = cardiac index; CS = cardiogenic shock; GUSTO = global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries; ICU = intensive care unit; IQ registry = Quality Improvement registry initiated by Abiomed (Danvers, MA, USA; manufacturer of Impella®)

PAC = pulmonary artery catheterization; SBP = systolic blood pressure; SPRINT registry = Secondary Prevention study with Nifedipine registry.

experienced minor bleeding. In Rossello (2017), four of 83 patients (4.8%) had major complications attributable to PAC use including heart block, VT, pneumothorax, and catheter-associated bloodstream infection.[31] In Sidhu (2017), a higher incidence of pneumothorax was observed in the PAC group; absolute numbers could not be obtained.[38]

Publication bias

We visually inspected funnel plots for both adjusted and unadjusted outcomes for mortality at longest available follow-up, which included 13 and 15 studies, respectively (see ESM, eFig. 7A/B). We proceeded with Egger's test as the funnel plots appeared to be asymmetric.[23] For adjusted and unadjusted outcomes, we obtained *P* values of 0.702 and 0.698 respectively, indicating that no publication bias was present.

Quality of evidence

We reviewed the quality of evidence using the GRADE framework (see ESM, eTable 4). All outcomes began as low-quality evidence given the observational nature of data; they were eventually downgraded to very low-quality evidence. All outcomes (hospital length of stay, mortality at longest follow-up, mortality at 30 days, in-hospital mortality) were downgraded for risk of bias due to the greater use of co-interventions during hospitalization among PAC patients. Mortality at 30 days was further downgraded for imprecision because a CI included both benefit and harm of PAC. Although significant heterogeneity ($I^2 \geq 90\%$) was present in our meta-analyses for length of stay and mortality, we elected to not downgrade for serious inconsistency as we felt this was explained by differences in magnitude of effect (large *vs* small) with overall consistent direction of effect; only one study[40] in our review found increased mortality with PAC.

Discussion

In this systematic review and meta-analysis of observational studies, the use of PAC in adults with CS was associated with reduced mortality in hospital and at the longest available follow-up. Thirty-day mortality was not statistically significant, though the direction of effect at this timepoint was consistent. The confidence in our results is limited by the observational nature of included studies, risk of bias, and marked heterogeneity. Furthermore, the quality of evidence for all outcomes was very low.

Despite the PAC population generally being more acutely ill (see Table 3), our adjusted and unadjusted

Table 2 Characteristics of patients on admission (nine studies)

	Age (yr) mean (SD)		Male sex n (%)		Comorbidities n (%)		Hemodynamic presentation		Biochemistry		Etiology of CS n (%)	
Study ID	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC
Doshi (2018)[27]	65 (14.8)	69 (14.8)	45,961 (64.3)	458,808 (59.5)	DM 18,209 (25.5) HTN 35,187 (49.3) COPD 16,235 (22.7) CKD 20,366 (28.5) PVD 8,307 (11.6)	DM 194,589 (25.2) HTN 408,630 (53.0) COPD 188,344 (24.4) CKD 209,143 (27.1) PVD 96,461 (12.5)	NR	NR	NR	NR	NR	NR
Garan (2020)[36]	58.5 (15.6)	62.4 (15.4)	416 (69.6)	196 (73.4)	DM 198 (33.1) HTN 274 (45.8) CKD 170 (28.4) PVD 34 (5.7) COPD 56 (9.4) CVA/TIA 85 (14.2)	DM 79 (29.6) HTN 145 (54.3) CKD 43 (16.1) PVD 13 (4.9) COPD 13 (4.9) CVA/TIA 20 (7.5)	Mean SBP 98 (18.8) Mean HR 90.5 (20)	Mean SBP 101 (26.3) Mean HR 90.0 (28)	Lactate (mmol·L^{-1}) 4.2 (4.1) Creatinine (mg·dL^{-1}) 1.7 (1.1)	Lactate (mmol·L^{-1}) 6.1 (4.8) Creatinine (mg·dL^{-1}) 1.7 (1.3)	ACS 159 (26.6) CM 369 (61.7) Other 69 (11.5)	ACS 123 (47.3) CM 57 (21.9) Other 59 (22.7)
Gore (1987)[28]	68 (SD not provided)	72 (SD not provided)	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR

Table 2 continued

	Age (yr)mean (SD)		Male sexn (%)		Comorbiditiesn (%)		Hemodynamic presentation		Biochemistry		Etiology of CSn (%)	
Study ID	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC
Hernandez (2019)[29]	64.0 (14.7)	68.1 (14.4)	50,837 (63.8)	495,590 (59.3)	DM 25,259 (31.7) HTN 38,247 (48.0) COPD 18,168 (22.8) PVD 8,924 (11.2) CAD 3,5618 (44.7) CVA/TIA 3,586 (4.5)	DM 263,256 (31.5) HTN 433,746 (51.9) COPD 203,083 (24.3) PVD 101,124 (12.1) CAD 393,631 (47.1) CVA/TIA 46,801 (5.6)	NR	NR	NR	NR	NR	NR
Oliveros (2020)[41]	Not provided		PAC used more often in males (#'s not provided)		NR	NR	NR	NR	NR	NR	NR	NR
Rossello (2017)[31]	66 (15.5)	71 (12.5)	54 (65)	30 (65)	DM 34 (41) HTN 59 (71) Smoking 22 (27) CKD 13 (16) PVD 9 (10.8) CAD 37 (44) CVA/TIA 8 (9.6)	DM 15 (33) HTN 25 (56) Smoking 13 (29) CKD 9 (20) PVD 2 (4.4) CAD 24 (52) CVA/TIA 4 (8.9)	Mean SBP 81 (17.1)	Mean SBP 79 (20.6)	NR	NR	ACS 40 (48) CM 18 (22) Arrhythmia 3 (4)	ACS 24 (52) CM 10 (22) Arrhythmia 2 (4)
Sidhu (2017)[38]	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	ACS ? (6) CM ? (15.5)	NR

Table 2 continued

	Age (yr)mean (SD)		Male sexn (%)		Comorbiditiesn (%)		Hemodynamic presentation		Biochemistry		Etiology of CSn (%)	
Study ID	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC
Sionis (2019)[32]	65 (12)	68 (11)	64 (78)	98 (72)	DM 22 (27) HTN NR COPD 7 (9) CKD 8 (10) CAD 26 (32) PVD 5 (6) CVA/TIA 5 (6)	DM 39 (28) HTN NR COPD 11 (8) CKD 17 (12) CAD 50 (36) PVD 16 (12) CVA/TIA 15 (11)	Mean SBP 80 (16) Mean HR 95 (27)	Mean SBP 76 (12) Mean HR 88 (29)	Lactate (mmol·L^{-1}) 4.2 (3.4) Creatinine (mmol·L^{-1}) 123 (61)	Lactate (mmol·L^{-1}) 4.4 (4.0) Creatinine (mmol·L^{-1}) 133 (101)	ACS 61 (74) CM 10 (12) Mechanical complication 8 (10)	ACS 116 (85) CM 13 (9) Mechanical complication 11 (8)
Vallabhajosyula (2020)[35]	68.3 (12.5)	70.1 (13.4)	18,446 (62.3)	197,291 (59)	NR	NR	NR	NR	NR	NR	All patients were admitted with AMI-CS	

ACS = acute coronary syndrome; AMI-CS = acute myocardial infarction and cardiogenic shock; CAD = coronary artery disease; CKD = chronic kidney disease; CM = cardiomyopathy; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; DM: = diabetes mellitus; HR = heart rate; HTN = hypertension; NR = not reported; PAC = pulmonary artery catheterization; PVD = peripheral vascular disease; SBP = systolic blood pressure; TIA = transient ischemic attack.

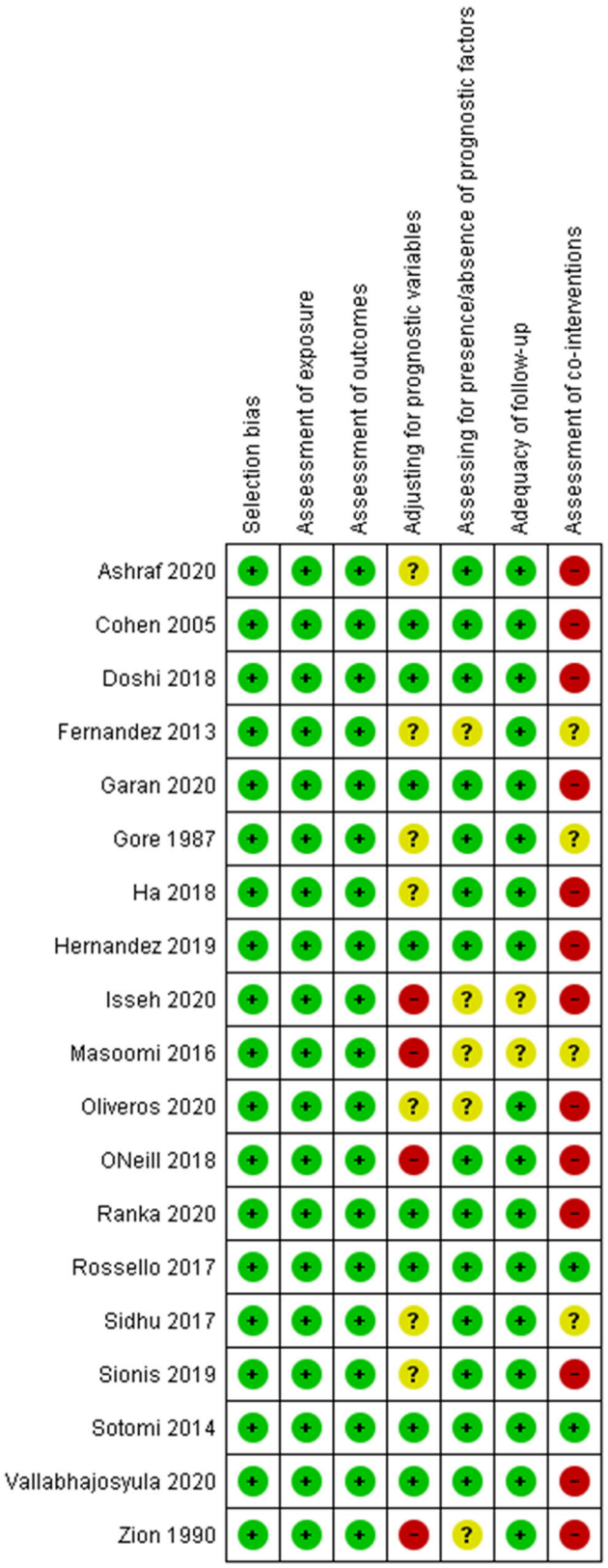
Table 3 Interventions during hospitalization (eight studies)

	Mechanical circulatory support n (%)		Vasopressors and/or inotropes n (%)		Advanced ventilatory support n (%)		Renal replacement therapy n (%)		Revascularization n (%)	
Study ID	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC
Ashraf (2020)[40]	All patients required mechanical circulatory support (details unavailable)		NR	NR	NR	NR	98 (5.2)	691 (3.7)	All patients were revascularized (details unavailable)	
Doshi (2018)[27]	IABP 26,080 (36.5) LVAD/Impella 1,715 (2.4)	IABP 180,395 (23.4) LVAD/Impella 8,480 (1.1)	7,207 (10.1)	62,398 (8.1)	NR	NR	NR	NR	NR	NR
Garan (2020)[36]	IABP 302 (50.5) LVAD/Impella 161 (26.9) VA-ECMO 77 (12.9) Any MCS 419 (70.1)	IABP 130 (50.0) LVAD/Impella 80 (30.8) VA-ECMO 95 (36.5) Any MCS 230 (88.5)	NR	NR	NR	NR	NR	NR	NR	NR
Hernandez (2019)[29]	IABP NR LVAD/Impella 3,665 (4.6) Any MCS 30,598 (38.4)	IABP NR LVAD/Impella 8,357 (1.0) Any MCS 208,934 (25.0)	NR	NR	NIV NR Intubated 44,861 (56.3)	NIV NR Intubated 413,688 (49.5)	7,649 (9.6)	48,473 (5.8)	PCI 14,662 (18.4) Thrombolytic NR CABG 11,474 (14.4)	PCI 192,219 (23.0) Thrombolytic NR CABG 101,960 (12.2)
Oliveros (2020)[41]	IABP 2266 (37.1) LVAD/Impella 232 (3.8) VA-ECMO 140 (2.3)	IABP 17,015 (23.5) LVAD/Impella 1,303 (1.8) VA-ECMO 869 (1.2)	617 (10.1)	6,082 (8.4)	NR	NR	550 (9.0)	5,720 (7.9)	PCI NR Thrombolytic NR CABG 1,765 (28.9)	PCI NR Thrombolytic NR CABG 12,743 (17.6)
Rossello (2017)[31]	IABP 31 (37) LVAD/Impella 1 (1)	IABP 10 (22) LVAD/Impella 1 (2)	81 (98)	39 (85)	NIV 11 (13) Intubated 59 (71)	NIV 4 (9) Intubated 25 (54)	21 (25)	6 (13)	PCI 20 (51) Thrombolytic 3 (8) CABG 2 (5)	PCI 15 (63) Thrombolytic 2 (8) CABG 0 (0)

Table 3 continued

	Mechanical circulatory supportn (%)		Vasopressors and/or inotropesn (%)		Advanced ventilatory supportn (%)		Renal replacement therapyn (%)		Revascularizationn (%)	
Study ID	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC	PAC	No PAC
Sionis (2019)[32]	IABP	IABP	76 (93)	105 (77)	NIV	NIV	18 (22)	12 (9)	PCI	PCI
	64 (78)	58 (42)			16 (20)	28 (20)			39 (72)	77 (82)
	LVAD/Impella	LVAD/Impella			Intubated	Intubated			Thrombolytic	Thrombolytic
	4 (4.9)	5 (3.7)			73 (89)	64 (47)			8 (15)	10 (11)
	VA-ECMO	VA-ECMO							CABG	CABG
	2 (2.5)	2 (1.5)							NR	NR
	Any MCS	Any MCS								
	56 (68)	60 (44)								
Vallabhajosyula (2020)[35]	IABP	IABP	NR	NR	NIV	NIV	1,717 (5.8)	9,697 (2.9)	PCI	PCI
	15,634 (52.8)	125,731 (37.6)			NR	NR			14,331 (48.4)	178,565 (53.4)
	LVAD/Impella	LVAD/Impella			Intubated	Intubated			Thrombolytic	Thrombolytic
	711 (2.4)	4,347 (1.3)			18,180 (61.4)	140,779 (42.1)			NR	NR
	VA-ECMO	VA-ECMO							CABG	CABG
	178 (0.6)	1,003 (0.3)							Excluded from study	Excluded from study

CABG = coronary artery bypass graft; IABP = intra-aortic balloon pump; LVAD = left ventricular assist device; MCS = mechanical circulatory support; NIV = non-invasive ventilation; NR = not reported; PCI = percutaneous coronary intervention; VA-ECMO = veno-arterial extracorporeal membrane oxygenation.



◀**Fig. 1** Risk of bias summary; review authors' judgements about each risk of bias item for included studies. Green circles with "+" sign indicate a judgement of low risk of bias. Red circles with "-" sign indicate a judgement of high risk of bias. Yellow circles with "?" sign indicate a judgement of unclear risk of bias.

results show an association between PAC use and decreased mortality. Garan *et al.* (2020) suggest that early complete hemodynamic profiling reduces the need for MCS in the PAC group.[36] Our results contrast with existing randomized data of PAC in other populations;[4–8] it is plausible that precise knowledge of cardiac indices, vascular resistances, and biventricular filling pressures may allow clinicians to make critical clinical decisions within a narrow therapeutic window to positively impact mortality in patients with CS. Literature indicates the majority of deaths in CS occur early,[11,16,17] suggesting PAC may allow for tailored therapy to optimize hemodynamic status, potentially leading to improved outcomes. Because the longest available follow-up in most of our included studies was only during patients' hospitalization, any benefit shown appears to be driven by reduced in-hospital mortality.

Pulmonary artery catheterization use was associated with longer hospital stay by a mean of 3.5 days. The increased length of hospitalization with PAC could result from more profound CS, more aggressive hemodynamic-guided management, or potentially procedural complications. It may also stem from the higher mortality in patients who did not undergo PAC, leading to shorter hospital stays. Lack of granularity of study-level data did not allow us to explore the timing of death in each group.

Pulmonary artery catheterization is not without harm. In the three studies that reported procedural complications,[31,32,38] complications occurred in up to 10% of procedures and ranged from minor bleeding to serious life-threatening adverse events including ventricular dysrhythmias, pneumothorax, and sepsis. These results are consistent with reported complication rates in randomized data from other populations.[4–7]Our work has several limitations, the main one being the very low quality of evidence based solely on observational data and the marked heterogeneity of our outcomes. This limits the ability to infer an effect of PAC use on mortality. As PAC is a diagnostic tool that provides data for clinicians to base clinical decisions, it is conceivable that inherent clinical sources of heterogeneity (e.g., provider expertise/training, practitioner/institutional variability, timing of PAC, different definitions of CS) could contribute to the marked heterogeneity seen in our results. Although this may call into question our decision to pool studies, we proceeded with meta-analysis because

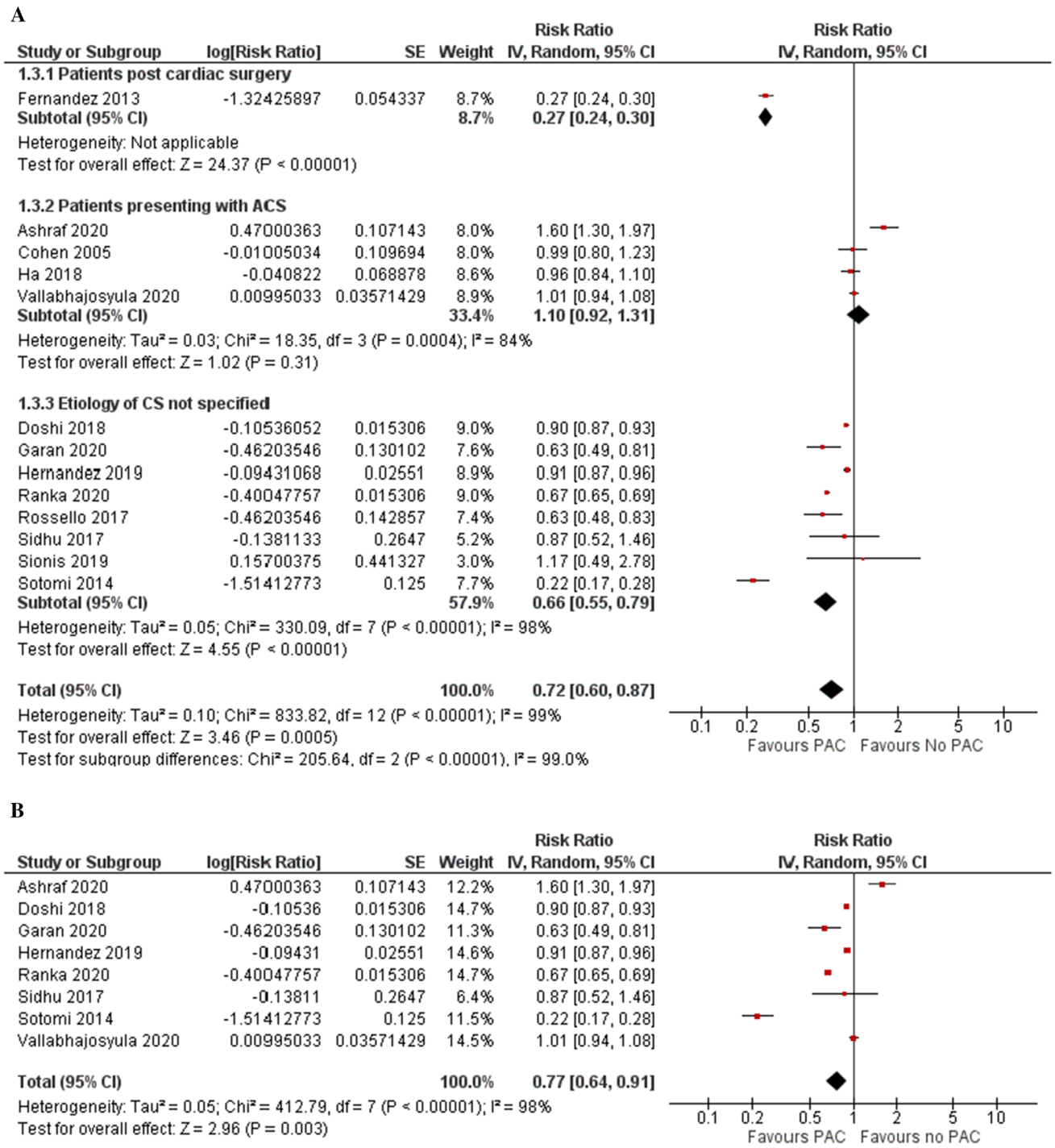


Fig. 2 Adjusted mortality at (A) longest available follow-up and (B) in hospital; pulmonary artery catheterization *vs* no pulmonary artery catheterization. Square markers represent the risk ratio point estimate for each primary study, with size of each square proportional to the weight of the given study in the meta-analysis. Horizontal lines indicate 95% CIs. The solid diamond represents the estimated 95% CI for effect size of all meta-analyzed data. ACS = acute coronary syndrome; CS = cardiogenic shock; PAC = pulmonary artery catheterization.

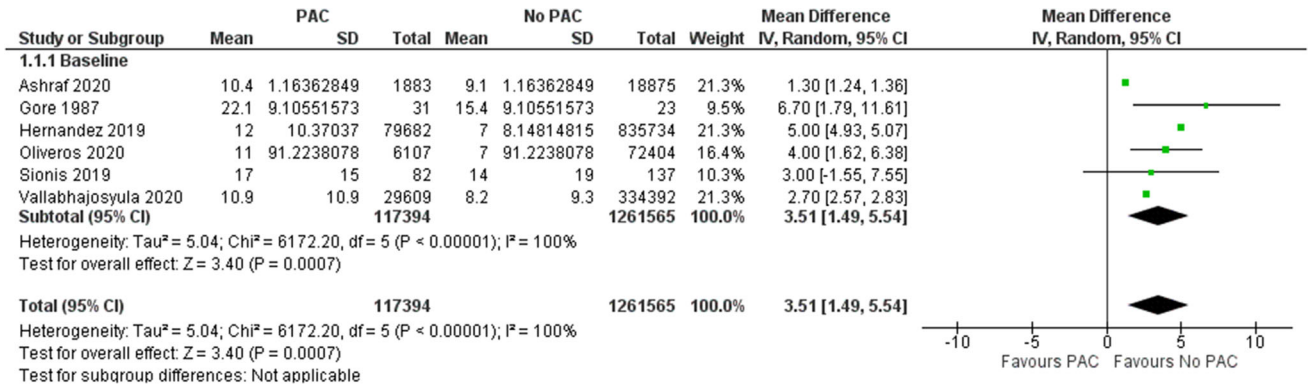


Fig. 3 Mean difference in hospital length of stay; pulmonary artery catheterization *vs* no pulmonary artery catheterization. Square markers represent the point estimate of mean difference in hospital length of stay for each primary study, with size of each square proportional to the weight of the given study in the meta-analysis. Horizontal lines indicate 95% CIs. The solid diamond represents the estimated 95% CI for effect size of all meta-analyzed data.

visual inspection of forest plots showed consistent directions of effect in the majority of included studies and the topic of study remains one where there is clinical equipoise.

Although mean duration of hospitalization was $\leq$ 30 days in studies reporting hospital length of stay, study-level granularity did not permit comparison of mortality curves across the temporal continuum (in hospital, at 30 days, and at longest available follow-up) between studies. Similarly, as the Society for Cardiovascular Angiography and Intervention only recently updated their classification (stages A to E) of CS,[14] it is difficult to determine where along this spectrum of shock our study cohorts fall, making it somewhat challenging to frame in contemporary context.

Lastly, eight of 19 studies[24,27,29,35,37,38,40,41] drew from overlapping cohorts of the National Inpatient Sample (NIS). The NIS is the largest database of inpatient hospital stays in the United States, incorporating data from all payers and approximately 20% of American community hospitals.[43] A limitation of the NIS is the sheer volume of patient data and with it, potential for marked residual and unmeasured confounding. We cannot exclude the possibility that some patients are being counted more than once; we elected to include all such studies as they were deemed sufficiently distinct in their design and inclusion/exclusion criteria. The observational nature of the pooled studies in this review further increases the risk of confounding by indication; as such, our results should be interpreted with caution.

Contemporary recommendations[11–14] for PAC use in CS are based on individual observational studies and expert consensus. Our meta-analysis provides the most comprehensive and rigorous analysis of PAC in CS published to date, following a prespecified protocol and using high methodological standards.[15] In patients who do not have a separate indication for invasive hemodynamic assessment (e.g., those being worked up for advanced therapies), prospective randomized clinical trials are needed to further characterize the role of PAC in patients presenting with CS. In addition, further work should assess the cost-effectiveness of routine PAC in CS, study the optimal timing of PAC, and/or seek to understand practitioner-to-practitioner variation in decision-making based on hemodynamic values.

Conclusions

In this systematic review and meta-analysis of observational studies, our work suggests that PAC use in patients with CS is associated with lower mortality. The observed increase in hospital length of stay may represent survivor bias or relate to more aggressive management with PAC. Overall, these results support consideration of PAC for hemodynamic assessment in CS; however, our confidence in this conclusion is diminished by the very low quality of available evidence and marked heterogeneity of included studies.

Author contributions *Justin Y. Chow*, *Maria E. Vadakken*, and *Emilie P. Belley-Côté* contributed to all aspects of this manuscript, including study conception and design and acquisition, analysis, and interpretation of data. Together, they generated the initial manuscript draft. *Alex Koziarz* contributed to statistical analysis of the data. *William F. McIntyre* and *Richard P. Whitlock* contributed to methodologic review/oversight of the manuscript. All authors contributed to data analysis, data interpretation, and iterative revision of the final manuscript.

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