



Stroke volume estimation using electrical impedance tomography: a systematic review of preclinical and clinical studies



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Transthoracic echocardiography and cardiac magnetic resonance can accurately determine stroke volume (SV), however, both require specialized expertise and infrastructure for analysis and interpretation. Electrical impedance tomography (EIT) has emerged as a non-invasive imaging modality that still requires appropriate training and equipment, but that can be implemented with comparatively simpler training and acquisition protocols. While EIT has been applied clinically for pulmonary assessment, it remains unestablished for cardiac applications. We performed a systematic review according to PRISMA 2020 guidelines and the Cochrane Handbook for Systematic Reviews to determine the performance of cardiac EIT in estimating SV (PROSPERO #CRD420251155255). PubMed and Google Scholar were queried from inception to October 31, 2025. Nine human studies included feasibility, simulation, physiological observational, and reference-comparison designs. Among studies incorporating independent reference measurements, cardiac EIT exhibited moderate correlation with established methods (R^2 up to 0.74). Similarly, eight porcine studies exhibited a wide range of correlations between EIT-derived and invasive SV estimates ($R^2 = 0.34$ – 0.96). Risk of bias assessed using the QUADAS-2 tool was low across studies; applicability and generalizability can be improved with wider patient selection. Cardiac EIT can track relative SV changes with reasonable reproducibility compared with reference assessments.

Electrical impedance tomography (EIT) is a low-cost and portable imaging modality that reconstructs cross-sectional images of thoracic conductivity by injecting small electrical currents through surface electrodes and measuring the resulting voltages¹. EIT is increasingly used in clinical practice, particularly in the assessment of lung ventilation and perfusion in the critically ill². However, the role of EIT in the clinical evaluation of cardiac function (cardiac EIT) has not yet been established. Since 2000, this imaging modality has been explored for heart function assessment in animal models and small-scale prototype human studies. Cardiac EIT has potential as a rapid prescreening tool to assess global cardiac function prior to referral for transthoracic echocardiography (TTE) or cardiac magnetic resonance (CMR) imaging. TTE and CMR provide episodic measurements and require extensive expertise, the latter of which is also limited by various contraindications (e.g., ferromagnetic implants, severe claustrophobia) that may prevent patient acceptance. By contrast, EIT can continuously monitor cardiac function with beat-to-beat estimates over an extended period, and

data can be acquired by operators with minimal training, which may support more standardized acquisition and reduce inter-operator and inter-observer variation^{3,4}. Such an approach could reduce costs for clinical practice and public health and improve access to cardiac assessment, particularly in settings where expertise, resources and equipment are lacking.

The spectrum of etiologies resulting in cardiac dysfunction is broad⁵. The functional division of the cardiac cycle into mechanical systole and diastole, and the prognostic importance of cardiac output, have implications for our understanding of cardiovascular pathophysiology, risk stratification, and disease management. Cardiac output (CO) is the product of heart rate and stroke volume (defined as the difference between end-diastolic volume [EDV] and end-systolic volume [ESV]). Similarly, left ventricular ejection fraction (LVEF) is defined as SV divided by EDV, and is an important parameter in clinical practice. Current heart failure (HF) management guidelines have classified the disease into HF with reduced ejection fraction (HFrEF, LVEF < 40%), HF with mildly reduced EF (HFmrEF, LVEF

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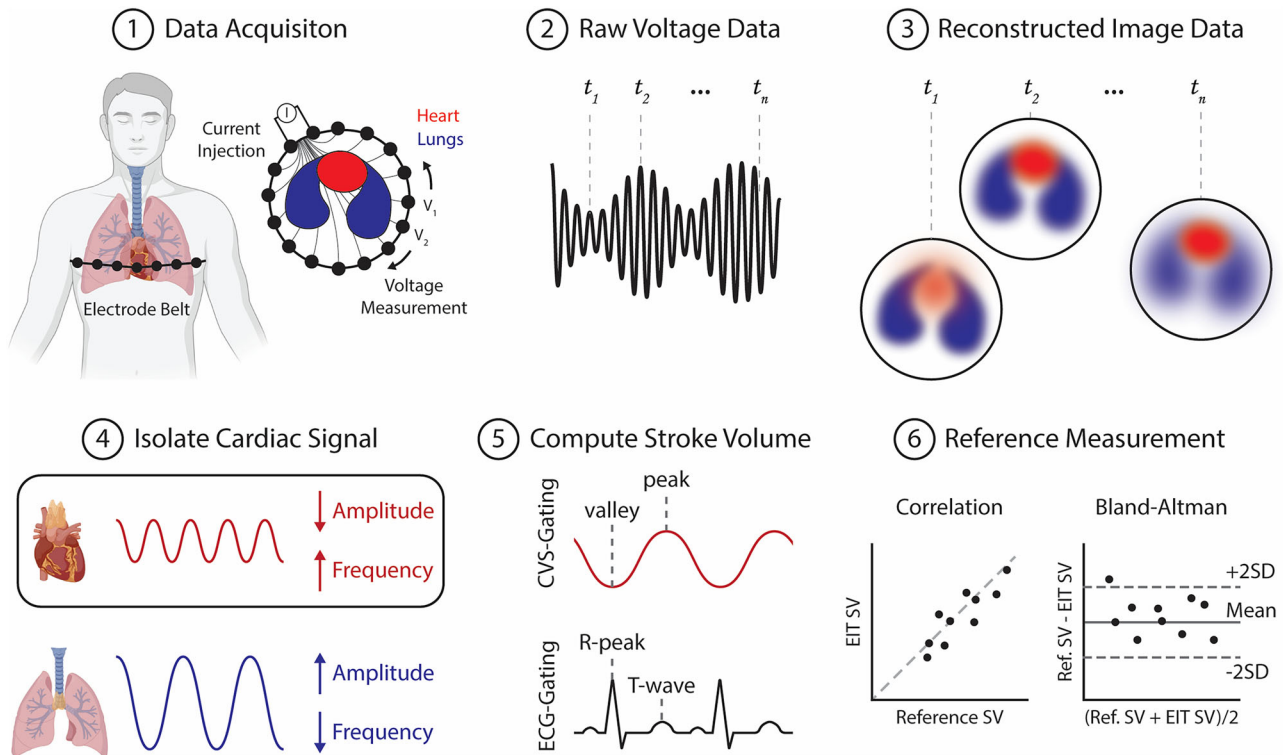


Fig. 1 | Conceptual workflow of using electrical impedance tomography (EIT) to estimate and validate cardiac stroke volume (SV). (1) EIT measures voltage changes from small alternating currents applied through an electrode belt around the thorax. (2) Sequential voltage measurements are acquired and (3) reconstructed into time-resolved cross-sectional images representing conductivity changes of the heart and lungs. (4) Cardiac and respiratory components are separated using

techniques that leverage their relative amplitude and frequency. (5) SV is often derived from the cardiac impedance waveform using ECG-gating, cardiac volume signal (CVS) gating, or no gating. (6) The resulting SV estimates are validated against reference measurements such as thermodilution, pulse contour analysis, or flow probes using correlation and Bland-Altman analyses. Some images in (1) and (4) were created in BioRender. Edelman, B. (2026) <https://BioRender.com/2muvio1>.

40–49%), or HF with preserved EF (HFpEF, LVEF $\geq 50\%$)⁶. This classification may appear oversimplified but is evidenced-based and informs clinical practice^{6,7}.

In healthy individuals, homeostatic perfusion of vital organs is achieved through dynamic chronotropic regulation of heart rate, in conjunction with SV, which is also modulated by the neurohormonal system to alter inotropy and lusitropy. SV can be regulated by altering the preload (venous return and ventricular filling), afterload (arterial resistance and mean arterial blood pressure), and myocardial contractility⁸. In chronic heart disease (e.g. cardiomyopathy) or during acute injury (e.g. acute myocardial infarction), changes in cardiac output that are detectable and quantified as LVEF may provide prognostic information leading to actionable disease management^{9,10}.

In clinical practice, SV and CO are conventionally measured using non-invasive imaging or, infrequently, by invasive hemodynamic catheterization. TTE requires professionally trained personnel for image acquisition, analysis, and data interpretation. CMR provides detailed structural and functional evaluation of the heart, and is a gold standard for volumetric quantification and geometric assessment of both left and right ventricles^{11–13}. However, the long acquisition times, high costs, and operational requirements of CMR, as well as its associated equipment, facility, and personnel have limited widespread use. Importantly, both of these methods require averaging across multiple cardiac cycles to obtain proper SV estimates, making continuous monitoring impractical. Invasive hemodynamic catheterization^{14,15} allows accurate measurement of SV and CO¹⁶ but may entail procedural and iatrogenic risks. In experimental animal models, catheters equipped with pressure sensors, ultrasound probes, optical fiber and laser Doppler transducers have been used to measure and quantify absolute SV with

high precision^{3,16}. In fact, invasive hemodynamic catheterization is important for benchmarking and validating different methods and technologies in experimental models.

EIT has already been applied successfully in large-scale pulmonary screening (>3000 individuals), where it has proven effectiveness in the early detection of lung impairment¹⁷. This modality is also capable of extracting dynamic cardiac impedance waveforms from each cardiac cycle, which can be used to estimate relative changes in SV and CO, and to provide a continuous assessment of cardiac function over long periods of time. The ability to capture physiological changes across cardiac and respiratory cycles suggests that it has the potential for similar large-scale applications in heart health screening in diverse settings, and may complement TTE and CMR^{18,19}. Two landmark studies from the early 2000s demonstrated the capability of thoracic EIT in detecting impedance fluctuations from cardiac contractions and blood volume shifts^{20,21}. When cardiac and pulmonary signals can be separated, the former provides a signal from which SV can be estimated and validated (Fig. 1). Indeed, experimental studies have demonstrated this capacity for continuously monitoring beat-to-beat heart function^{3,22}. However, progress and development of cardiac EIT during the last decade has been hampered by challenges in signal processing and methodological reliability issues. As no previous review has systematically evaluated the methodologies used to estimate SV from EIT in either human or animal models, we conducted a systematic review specifically focused on EIT-based SV estimation. This review aims to summarize existing experimental designs, cardiac signal extraction methods (from EIT images), and validation strategies used in order to inform future developments and clinical translations of EIT-based cardiac monitoring.

Results

Study characteristics

Seventeen studies published between 2000 and 2024 met the inclusion criteria. Of these, nine involved human individuals (three in silico) and eight involved porcine models. Although foundational studies using healthy human participants date back to 2000, interest in EIT-based SV estimation has grown in recent years; 11 of 17 studies were published within the last decade alone (Fig. 2). An overview of study designs, reference standards, and main results is summarized in Tables 1 and 2, and technical details are available in Supplementary Tables 1 and 2.

Foundational studies in human

The earliest human studies shared the common goal of demonstrating that cardiac-related impedance changes measured by EIT could be meaningfully linked to SV using independent reference standards. Clinical studies prior to 2010, therefore, focused primarily on feasibility, whereas work after 2020 has emphasized reliability and robustness. In the first clinical study of its kind, by Vonk-Noordegraaf et al. evaluated 25 patients undergoing right heart catheterization and demonstrated a strong correlation between an ECG-gated SV index derived from EIT signals and invasive thermodilution

measurements ($R^2 = 0.74$)²⁰. A subset of 11 patients from that study also underwent magnetic resonance (MR) velocimetry, with Bland-Altman analysis showing that EIT-based SV estimation exhibited a mean bias of around -1.0 mL with limits of agreement (LoA) of roughly ± 11.0 mL, relative to MR-derived SV values. Importantly, the authors highlighted the sensitivity of cardiac SV estimation to electrode configuration, showing that an oblique (slanted) configuration improved isolation of ventricular impedance changes relative to conventional transverse (horizontal) layouts. Building on this feasibility work, Zlochiver et al. combined numerical simulations with in vivo validation to assess an optimized EIT reconstruction approach for SV estimation. Simulations demonstrated near one-to-one correspondence between reconstructed impedance changes and true SV ($R^2 = 0.94$)²¹. When subsequently compared to impedance cardiography measurements, a non-imaging technique that estimates SV from thoracic impedance changes between surface electrodes, EIT-derived measurements from 28 healthy volunteers correlated well ($R^2 = 0.74$), with minimal bias (mean bias -1.3 mL, LoA approximately ± 18.5 mL). Taken together, the above studies established a foundation for EIT-based SV monitoring in humans.

Benchmarking EIT against established methods

Subsequent studies focused on evaluating EIT against independent SV measurement techniques. Proença et al. conducted a case-study simulation using a dynamic four-dimensional MRI-derived chest model to investigate the influence of cardiac motion on EIT-based SV estimates²³. The authors found that although myocardial motion and displacement effects together contributed 56% of the thoracic impedance signal, motion-induced impedance changes were also highly correlated with the volumetric changes of the LV ($R^2 = 0.96$). Nevertheless, EIT simulations isolating the effects of blood volume changes and heart movement accurately estimated a wide range of SV values (mean error of 0.6 mL and 2.19 mL, respectively), suggesting that ventricular impedance magnitude can serve as a reliable proxy for SV when confounding factors such as heart movement are accounted for. Similarly, Braun et al. evaluated three-dimensional EIT in 10 healthy volunteers during postural changes and mild exercise scenarios, using oxygen uptake (VO_2)-derived measurements as a non-invasive reference for SV²⁴. While cardiac and pulmonary impedance signals could be delineated, absolute SV estimates exhibited wide scatter (± 15 mL error) when using an EIT-derived proxy of systolic heart volume even after subject-specific calibration. By contrast, trending analysis using an exclusion zone of $\pm 30\%$ demonstrated that relative SV estimates exhibited a concordance rate of

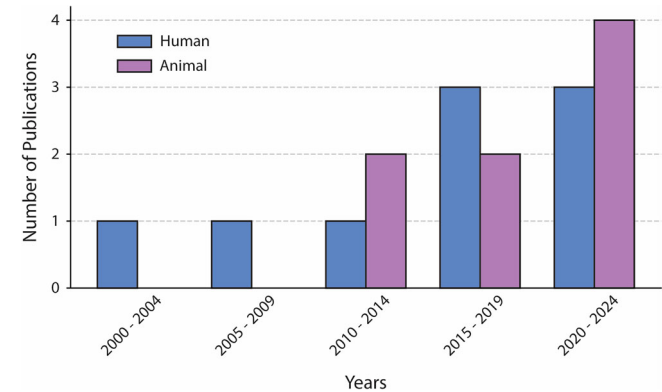


Fig. 2 | Number of published studies on EIT-based SV estimation as of October 31, 2025. Studies are categorized by human (blue) and animal (purple) studies for every five-year period. A steady increase in publications over the past decade highlights growing interest in EIT for cardiac monitoring in both humans and animal models.

Table 1 | Summary of human studies evaluating EIT for stroke volume estimation

First author	Year of Pub.	PMID	Sample size	Reference Meas.	Main finding(s)
Vonk-Noordegraaf et al.	2000	10847195	25 patients 11 healthy volunteers	TPT, MRI	R^2_{SV} (EIT vs TPT) = 0.74, ^a mean bias -1.0 mL, LoA ± 11.0 mL
Zlochiver et al.	2006	16636406	28 healthy volunteers	IC	R^2_{SV} (EIT vs IC) = 0.74, ^a mean bias -1.3 mL, LoA ± 18.5 mL
Mhajna et al.	2013	23606371	Simulation	N/A	Validated parametric EIT for ground truth left ventricle estimation
Proença et al.	2015	26006113	Simulation	N/A	Myocardial movement contributes 56% of the thoracic impedance signal
Braun et al.	2018	29373611	10 healthy volunteers	VO_2	CR_{SV} (EIT vs VO_2) = 84.2% subject-independent calibration, CR_{SV} = 93% subject-specific calibration (relative SV, EIT lung volume)
Braun et al.	2018	29099725	Simulation	N/A	Vertical electrode shifts induce large errors in EIT-based SV estimation
Braun et al.	2020	31624996	20 patients	TPT	R^2_{SV} (EIT vs TPT) = 0.02, CR = 52.6%, mean bias 11.9 mL, LoA ± 32.2 mL (EIT heart amplitude) R^2_{SV} (EIT vs TPT) = 0.52, CR = 94.4%, mean bias 2.7 mL, LoA ± 14.9 mL (EIT lung amplitude)
Arshad et al.	2020	33021240	20 patients	TTE	R^2_{SV} (CEI vs TTE) = 0.64 R^2_{SV} (RCGA vs TTE) = 0.50
Kwon et al.	2021	33683548	9 patients	None	Respirophasic variation in SV: decreased by 8.8% during apneas and by 4.2% during hypopneas

CEI cardiac eigen imaging, CR concordance rate, EIT electrical impedance tomography, IC impedance cardiography, LoA limits of agreement, MRI magnetic resonance imaging, N/A not applicable, RCGA regular cardiac gated averaging, SV stroke volume, SVV stroke volume variation, TPT transpulmonary thermodilution, TTE transthoracic echocardiography, VO_2 oxygen uptake.

^aBland-Altman reporting not explicitly stated in original source paper, and instead approximated from the provided plot.

Table 2 | Summary of animal studies evaluating EIT for stroke volume estimation

First author	Year of pub.	PMID	Sample size	Reference meas.	Main finding(s)
Maisch et al.	2011	21666450	8 Healthy	UFP, APCA	R^2_{SVV} (EIT vs UFP) = 0.69, mean bias 1.87%, LoA $\pm 7.02\%$ R^2_{SVV} (EIT vs APCA) = 0.73, mean bias 0.49%, LoA $\pm 5.85\%$
Pikkemaat et al.	2014	24810260	14 Healthy	TPT	R^2_{SV} (EIT vs TPT) = 0.34 uncalibrated, R^2_{SV} = 0.72 calibrated
Treppe et al.	2017	28039243	28 Healthy	TPT, APCA	R^2_{SVV} (EIT vs APCA) = 0.59 healthy R^2_{SVV} = 0.71 lavage injury R^2_{SVV} = 0.23 oleic acid injury
da Silva Ramos et al.	2018	28742775	12 Healthy	TPT, APCA	R^2_{SV} (EIT vs TPT) = 0.35 R^2_{SV} (EIT vs TPT + anthropometric measurement) = 0.85, CR = 91.2%, mean bias 0.0 mL, LoA ± 7.97 mL, $^{95}\%$ CI ± 2.0 mL for upper and lower LoA)
Jang et al.	2020	32647206	6 Healthy	TPT	R^2_{SV} (EIT vs TPT) = 0.86, mean bias -0.01 mL, LoA ± 4.7 mL
Ko et al.	2021	33534400	5 Healthy	APCA	R^2_{SV} (EIT vs APCA) = 0.96, mean bias 0.0 mL, LoA ± 3.6 mL
Jang et al.	2023	35960417	5 Healthy	APCA	Trending agreement (EIT vs APCA)
Chung et al.	2024	38316842	32 Healthy	UFP, PAC-CCO, APCO	CR_{SV} (EIT vs UFP) = 94.2%, mean bias -1.98 mL, LoA ± 10.8 mL, PE = 24.8% CR_{SV} (EIT vs PAC-CCO _{adjust}) = 72.4%, PE = 34.6% CR_{SV} (EIT vs APCO) = 69.3%, PE = 38.8%

APCA arterial pulse contour analysis, EIT electrical impedance tomography, PAC pulmonary artery catheterization, PAC-CCO pulmonary artery catheterization—continuous cardiac output, SV stroke volume, SVV stroke volume variation, TPT transpulmonary thermodilution, UFP ultrasonic flow probe, CR concordance rate, LoA limits of agreement.

*Bland-Altman reporting not explicitly stated in reference, and instead approximated from the provided plot.

93.0% when using EIT-derived lung volume features and subject-specific calibration (compared to 84.2% with subject-independent calibration). It should be noted that in this work a substantial proportion of measurements (~38%) were excluded due to signal instability or noise, which highlights practical limitations in cardiac EIT data quality. Overall, this work concluded that purely amplitude-based EIT features are highly sensitive to posture, electrode contact, and lung conductivity variations. This conclusion was supported in a later study by Braun et al. that quantified how various factors impacted EIT-based SV estimations, and indicates that additional constraints or advanced feature extraction are required to achieve clinically robust SV estimation²⁵. Specifically, simulations of electrode belt shifts and lung volume changes showed notable errors in absolute SV estimation, especially when the belt was misplaced in the vertical (-29.2 to $+28.2\%$) or horizontal (-3.4 to $+13.4\%$) dimension, or when electrodes close to the heart were detached (4–5%). While these errors may be ameliorated by recalibrating the system, the ability to detect relative SV changes (i.e., trends) from EIT was far more robust even without recalibration. This finding strongly supports the concept of focusing on trend monitoring rather than single-beat absolute volumes when using EIT in practice.

Cardiorespiratory interactions in human

Recent clinical studies have also applied EIT to investigate cardiorespiratory interactions and physiological changes in SV. A study by Kwon et al. using EIT in conjunction with overnight polysomnography measured beat-by-beat SV and breath-by-breath tidal volume in nine patients with obstructive sleep apnea (OSA)²². SV was found to be significantly decreased during apneas (-8.8%) and certain hypopneas (-4.2%) based on over 2000 respiratory events recorded. In another study by Braun et al., 20 ventilated, critically ill patients in the intensive care unit were monitored with both EIT and thermodilution while receiving 500 ml intravenous fluid bolus challenge²⁶. This study showed that impedance changes marked by a lung region of interest (ROI) concurred with SV increase (concordance rate = 94.4%, R^2 = 0.52, mean bias 2.7 mL, LoA ± 14.9 mL) to a much greater extent than a heart ROI (concordance rate = 52.6%, R^2 = 0.02, mean bias 11.9 mL, LoA ± 32.2 mL), suggesting that the former may provide an alternative and sensitive indicator of preload (and/or possibly pulmonary edema in patients with elevated LV end-diastolic pressure). Further invasive hemodynamic research is needed to determine any potential utility of a lung EIT ROI in patients with left HF, and to clarify the dynamic relationship between a heart EIT ROI and changes in inotropy in response to intravenous fluid challenge. Towards improvement in cardiac EIT signal extraction,

Arshad et al. recently developed a novel decomposition approach (“eigen-imaging”)²⁷ and demonstrated in 20 patients with HF higher correlation between eigen-imaging and TTE-derived SV (R^2 = 0.64), compared with conventional ECG-gated averaging (R^2 = 0.52).

Animal studies: hemorrhage, volume shifts, and resuscitation

Pig models have also played a central role in validating EIT for cardiac function estimation under controlled hemorrhage and resuscitation scenarios. Maisch et al. evaluated ventilation-induced SV variation (SVV) using EIT in eight pigs fitted with an aortic ultrasonic flow probe and thermodilution-based pulse contour analysis²⁸. In contrast to SV, SVV reflects preload responsiveness (primarily, in mechanically ventilated subjects) and is therefore an indirect measure of cardiac function. Preload was directly modulated through combinations of positive end-expiratory pressure (PEEP) (0–15 cm H₂O), tidal volume (8 vs. 16 mL/kg), hemorrhage, and intravenous fluid administration. Across these conditions, EIT-based SVV significantly correlated with both the flow-probe SVV (R^2 = 0.69, mean bias 1.87%, LoA $\pm 7.02\%$) and pulse contour SVV (R^2 = 0.73, mean bias 0.49%, LoA $\pm 5.85\%$). A similar study by Pikkemaat et al. tracked absolute SV values in 14 anesthetized pigs using EIT and transpulmonary thermodilution during stepwise PEEP elevations (0–20 cm H₂O)²⁹. While the initial correlation was moderate (R^2 = 0.34), subject-specific calibration markedly improved agreement (R^2 = 0.72). Importantly, modulating SV by means of PEEP can shift the heart relative to the imaging plane, which can alter the signal independent of true SV changes and potentially lead to SV underestimation. Nevertheless, this work highlights the sensitivity of EIT to factors such as lung volume and the position of the heart relative to the imaging plane.

In another study, da Silva Ramos et al. studied 12 pigs subjected to alternating hemorrhage and fluid challenges under varying ventilatory conditions (PEEP 5–10 cm H₂O; tidal volume 6 vs. 10 mL/kg) that mimicked ICU scenarios and represent a more advanced physiological condition compared to PEEP-induced SV changes³⁰. Hemorrhage was induced by draining 50% of the total blood volume, followed by resuscitation with 500 ml of lactated Ringer’s solution. EIT-derived systolic impedance variation reliably tracked direction changes in thermodilution-based SV values, achieving a concordance of 91% and an AUC 0.83 for predicting SV increases during fluid boluses. Despite these promising results, absolute SV correlation was modest (R^2 = 0.35). Interestingly, when anthropometric values were considered, the SV correlation greatly improved (R^2 = 0.85, mean bias 0.0 mL, LoA ± 7.97 mL). Finally, Ko et al. demonstrated that

during repeated “mini-fluid” challenges (up to 30% of blood volume removal followed by ten 100 ml crystalloid fluid infusions), EIT-derived SV values closely matched pulse contour analysis ($R^2 = 0.96$, mean bias 0.0 ml, LoA ± 3.6 mL)³. Furthermore, this work reported accurate beat-by-beat EIT-derived SV values, whereas those via pulse contour analysis were reported every 20 s. Importantly, a common thread across all of these studies is that they incorporate a subject-specific scaling procedure to account for individual variation in anatomy and absolute SV values, further emphasizing the need for individual calibration.

Animal studies: pulmonary endothelium injury models

Experimental lung injury models have also been used to assess how pulmonary pathologies influence the reliability of EIT-based cardiac function, albeit primarily using the indirect measure of SVV. Trepte et al. compared EIT-based SVV with arterial measurements in 28 pigs subjected to either repeated bronchoalveolar lavage or intravenous administration of oleic acid³¹, a fatty acid that can cause pulmonary edema, congestion, hemorrhage, and acute respiratory distress syndrome. EIT-based SVV tracked the arterial measurements well ($R^2 = 0.59$, $n = 48$ measurement pairs), and during lavage-based injury to deplete pulmonary surfactant, the correlation surprisingly improved ($R^2 = 0.71$, $n = 35$ measurement pairs). Oleic acid-induced lung injury resulted in more variability in EIT measurements and led to a reduced correlation with arterial counterparts ($R^2 = 0.23$, $n = 40$ measurement pairs). Overall, SVV estimates were unaffected by PEEP values (healthy: 10 cm H₂O, $n = 27$, $R^2 = 0.56$, mean bias -0.81% , LoA $\pm 4.86\%$, 20 cm H₂O, $n = 21$, $R^2 = 0.64$, mean bias 0.90%, LoA $\pm 5.12\%$; lavage injury: 10 cm H₂O, $n = 18$, $R^2 = 0.79$, mean bias 0.76%, LoA $\pm 7.49\%$, 20 cm H₂O, $n = 17$, $R^2 = 0.58$, mean bias 0.20%, LoA $\pm 6.50\%$; oleic acid injury: 10 cm H₂O, $n = 23$, $R^2 = 0.13$, mean bias -1.42% , LoA $\pm 7.63\%$, 20 cm H₂O, $n = 17$, $R^2 = 0.40$, mean bias 1.34%, LoA $\pm 7.36\%$), but differed significantly between injury type. Specifically, degraded performance was observed in diffuse lung injury (e.g., when caused by oleic acid) where widespread edema reduces the impedance contrast between lung tissue and heart structures. It is also notable that the large LoA (± 5 – 7%) reported across all conditions approach the clinical SVV threshold (around 10%) that is used to define fluid responsiveness, indicating that measurement variability may impact decision-making.

Real-time SV estimation in animals

More recently, a series of sophisticated porcine experiments by a Korean group has advanced EIT towards real-time cardiopulmonary monitoring. Jang et al. introduced a decomposition method to isolate cardiac impedance signals from ventilation counterparts, and validated it in six pigs by comparing the extracted cardiac signal against thermodilution-based SV values³². Overall, EIT-derived SV values exhibited a strong linear relationship with true SV values ($R^2 = 0.86$, mean bias -0.01 mL, LoA ± 4.7 mL). A similar spatial filtering framework based on leadforming³³ was deployed in a subsequent study by Chung et al. that involved comprehensive validation in 23 pigs combining many interventions (vasodilators, inotropes, hemorrhage, PEEP changes) to stress-test EIT against various invasive SV measurements such as pulmonary artery catheters and ultrasonic flow probes¹⁶. Notably, by including multiple vasoactive drug protocols, this work facilitated the evaluation of cardiac function across changes in vascular tone and hemodynamics, which has traditionally been an important limitation of many non-invasive monitoring approaches. Across thousands of heartbeats ($>12,000$), EIT-based SV calculations exhibited a small bias (-1.98 mL, LoA -10.8 mL) and a percent error of 24.8% compared to flow probes, which was similar to or better than that observed between various combinations of invasive reference measurements (flow probe vs. thermodilution: 34.8%, vs. thermodilution (time adjusted): 24.2%, vs. pulse contour analysis: 34.5%). Moreover, the 10-min concordance reached 94.2% (percent error 24.8%), 72.4% (percent error 34.6%), and 69.3% (percent error 38.8%) between EIT measurements and the flow probe, pulmonary artery catheterization—continuous CO, and arterial pulse contour analysis, respectively. Overall, these results indicate that with appropriate signal processing and calibration,

modern EIT systems can continuously measure SV with accuracy and reproducibility that approaches established invasive techniques.

Common cardiac signal extraction approaches

Throughout the 17 included studies, three broad methodological strategies were used to extract cardiac signal from thoracic EIT data: (1) parametric model fitting-based ROI, (2) pixel clustering-based ROI, and (3) blind source separation techniques. In practice, these approaches are often complemented by gating techniques to estimate SV values. An overview of these approaches is provided below and is summarized in Fig. 3, Tables 3 and 4.

Parametric model fitting

ROI-based methods represent a straightforward approach for quantifying cardiac-related impedance changes in EIT. In these cases, the goal is to identify anatomical areas corresponding to the heart or major vessels, and to then track the behavior of this ROI over time to quantify heart function by means of SV or other metrics. Where simple thresholding approaches have been effective²⁰, parametric approaches aim to explicitly model the geometry of the heart within EIT images. This is typically performed by representing the left ventricle (LV) as an ellipse (or ellipsoid in 3D), the parameters of which are optimized to match the observed impedance data. Zlochiver et al. introduced an early example of this strategy by fitting an elliptical LV model using Newton-Raphson optimization across cardiac phases²¹. Work by Mahajna and Abboud extended this concept using Levenberg-Marquardt optimization in simulation studies, and demonstrated high agreement with ground truth values (error rate of $<1\%$)³⁴.

Pixel-based clustering

Contrary to parametric fitting, pixel-based clustering approaches exploit temporal features of the EIT signal to delineate heart-related structures. Early work by Maisch et al. and Trepte et al. applied the principle of pulse transit time analysis, and clustered pixels based on the arrival time of impedance pulses associated with cardiac ejection of blood to identify vascular structures such as the descending aorta^{28,31}. Later approaches shifted to cardiac frequency-based clustering where heart-related pixels were identified using variance, phase, or amplitude characteristics at the cardiac frequency. For example, Proenca et al.²³ defined heart ROIs using pixel-wise cardiac power maps segmented via the watershed algorithm, while Braun et al.²⁵ did so based on phase relationships and end-systolic to end-diastolic difference images. Subsequent studies by the same research group adopted this framework, and repeatedly applied it to identify heart ROIs across various datasets and conditions^{24,26}.

Blind source separation techniques

Blind source separation techniques seek to isolate cardiac-related impedance changes without defining an explicit spatial ROI. These approaches assume that signal sources from ventilation, cardiac activity, and/or perfusion have distinct spatio-temporal profiles that can manifest as unique statistical components^{35,36}. Principal component analysis (PCA) has been used to separate dominant ventilation-induced impedance changes from comparatively smaller, faster cardiac-related oscillations²⁹. More advanced extensions, such as cardiac eigen imaging (CEI) developed by Arshad et al. applies singular value decomposition (SVD) to reconstructed EIT images and selects components based on their comparison with a canonical cardiac template²⁷. More recently, Jang et al. combined PCA and independent component analysis (ICA) directly to raw EIT voltage data to separate respiratory and cardiac signals³², and in other work to recover both tidal volume and SV signals^{3,37}.

Gating techniques

Gating strategies are commonly used to support cardiac signal extraction and SV computation. ECG gating has been employed both to align EIT frames across the cardiac cycle²⁰ and to facilitate automated heart ROI segmentation³⁰. However, other studies derive a continuous cardiac volume

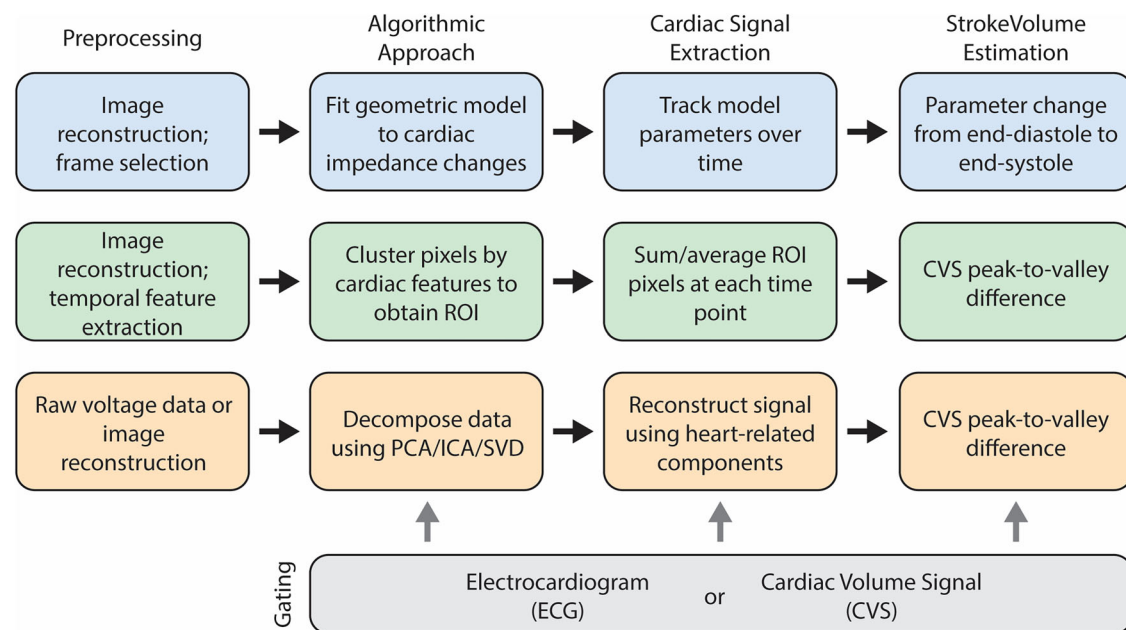


Fig. 3 | Overview of EIT-based cardiac signal extraction approaches. Cardiac signal extraction can mostly be categorized as parametric model fitting (blue), pixel-based clustering (green), and blind-source separation (orange) techniques. Each of these approaches uses as input either raw EIT voltage data or reconstructed EIT

images, and estimates cardiac SV from a temporally resolved cardiac signal. Gating techniques (gray) using electrocardiogram or the cardiac volume signal can be applied at various steps in the processing streams depending on its specific utility.

signal (CVS) either via ECG gating or using only the extracted cardiac impedance waveform. In the latter cases, SV is computed directly from the peak-to-valley (end-systole to end-diastole) amplitudes of the CVS, enabling purely EIT-based SV estimation^{3,22–26,29,32,37}.

Study quality

Among the nine human studies (Fig. 4), most showed low risk of bias in the “flow and timing” and “index test” domains, suggesting reliable measurement procedures and minimal data exclusion. Risk of bias was more variable in the “patient selection” and “reference standard” domain, with three simulation-based studies showing high or unclear risk due to the lack of clinical subjects. Similarly, there was little risk of bias for applicability, except for concerns regarding simulation studies as these were far removed from practical clinical use of EIT for SV estimation.

The eight animal studies (Fig. 4) demonstrated consistent methodological rigor, with low risk of bias across most domains and well-documented acquisition protocols. However, all animal studies were judged as having a high applicability concern for “patient selection” due to the limited generalizability of porcine models to human populations. This pattern emphasizes the trade-off between methodological control and translational relevance across animal and human study types.

Discussion

The studies evaluated in this review suggest that EIT can track SV-related changes under controlled (resting) conditions, but current evidence does not yet fully support clinical readiness. Unlike TTE and CMR, EIT can continuously follow cardiac output trends through time in the hospital and ambulatory settings³⁸. In conjunction with pulmonary artery pressure monitoring, continuous SV monitoring may add to our current understanding of changes in hemodynamics before and after acute HF decompensation^{39,40}.

There are technical and methodological challenges that hamper the clinical adoption of EIT-based SV monitoring. Cardiac-related impedance changes remain technically difficult to differentiate from simultaneous lung-induced changes. Lung impedance fluctuations are roughly an order of magnitude larger than cardiac-related impedance changes^{41,42}, making it

difficult to extract a clear cardiac waveform even when using advanced filtering or gating techniques^{18,24}. While ECG-gated averaging of many heartbeats can improve the signal-to-noise ratio, real-time tracking with this approach is not feasible. Similarly, decomposition techniques can isolate the cardiac signal, but are difficult to implement online due to the relatively large amount of data required to run these algorithms³⁵. Compounding this issue is whether cardiac signals are extracted from reconstructed EIT images or directly from raw voltage data (Tables 3 and 4). Image-domain approaches depend on the associated reconstruction algorithm, which influences spatial sensitivity and signal amplitude, whereas voltage-domain approaches separate signals prior to reconstruction and may avoid these confounders. This distinction is particularly relevant for key confounders identified by Braun et al^{24–26}, such as belt sensor displacement, lung conductivity, posture of the individual, and calibration stability, which together provide a practical framework for assessing the robustness of EIT-based SV estimation. Notably, the high-performing voltage-domain approaches discussed here^{3,32} have not yet been systematically evaluated against these confounders, and should therefore be interpreted as best-case results under controlled conditions.

As EIT produces a single cross-sectional image of the thorax with the heart being treated as one entity rather than distinct chambers, the spatial resolution is limited. This global heart representation makes it difficult to attribute observed SV changes to a specific ventricle, and is in contrast to many SV reference measurements that reflect only LV output. In healthy individuals, the output of the left and right ventricles is approximately equal⁴³, whereas unbalanced SV between ventricles, which may arise from conditions such as valvular disease, pulmonary hypertension, or ventricular dysfunction, can be indicative of cardiopulmonary pathologies and poor prognosis⁴⁴. Therefore, improving the spatial resolution of EIT imaging through artificial intelligence approaches^{45,46} and/or developing algorithms that discriminate the right from the left ventricle will be important technical advancements for deployment in the future monitoring of biventricular EF.

Another unresolved issue in cardiac EIT is the variable background impedance contrast, whereby the amplitude of pulsatile cardiac signals is influenced not only by ventricular blood volume changes but also by the impedance contrast between the heart, blood, and surrounding lung tissue.

Table 3 | Summary of EIT-based heart extraction methods in the included human studies

First author	PMID	Cardiac signal extraction	Gating	Image or voltage domain	Reconstruction algorithm	Key details
Vonk-Noordegraaf et al.	10847195	Other	ECG	Image	Not reported	Average EIT image thresholded above pulmonary signal to define cardiac ROI; SV computed relative to end-diastole.
Zlochiver et al.	16636406	Model Fitting	None	Image	Newton-Raphson Optimization	Fitting a parametric ellipsoidal LV model to electrode recordings using Levenberg–Marquardt optimization.
Mhajna et al.	23606371	Model Fitting	None	Image	Tikhonov Regularization	Derived from a differential ellipsoidal LV model optimized via Newton–Raphson iteration between systole and diastole
Proenca et al.	26006113	Pixel Clustering	CVS	Image	Graz consensus Reconstruction algorithm for EIT (GREIT)	Cardiac activity isolated via SVD; ventricular ROI auto-segmented from maximal cardiac power pixels; SV computed as peak-to-valley ROI impedance change
Braun et al.	29373611			Image	3D GREIT	
Braun et al.	29099725			Image	GREIT	
Braun et al.	31624996			Image	GREIT	
Arshad et al.	33021240	Decomposition	None	Image	Gauss–Newton linear solver, dual-mesh method, Tikhonov regularization	SVD-based eigen imaging isolates cardiac activity by matching singular vectors to a canonical template
Kwon et al.	33683548	Decomposition	CVS	Voltage	N/A	PCA and ICA applied to separate lung and cardiac components; cardiac waveform identified by dominant heart-rate frequency. SV computed as peak-to-valley amplitude of CVS.

CVS cardiac volume signal, ECG electrocardiogram, ICA independent component analysis, LV left ventricle, N/A not applicable, PCA principal component analysis, PMID PubMed identification number, ROI region of interest, SVD singular value decomposition.

Table 4 | Summary of EIT-based heart extraction methods in the included animal studies

First author	PMID	Cardiac signal extraction	Gating	Image or voltage domain	Reconstruction algorithm	Key details
Maisch et al.	21666450	Pixel Clustering	None	Image	Not reported (Enlight)	Pulse transit time-based clustering identified aortic pixels; SV estimated from cardiopulmonary frequency interaction energy
Pikkemaat et al.	24810260	Decomposition	CVS	Image	Proprietary (Dräger)	PCA-based signal separation, per-cycle SV surrogates computed as peak-to-valley of the cardiac impedance waveform
Treppe et al.	28039243	Pixel Clustering	None	Image	Not reported (Enlight)	Pulse transit time-based clustering identified aortic pixels; SV estimated from cardiopulmonary frequency interaction energy
da Silva Ramos et al.	28742775	Pixel Clustering	ECG	Image	Not reported (Enlight)	ECG gating segmented heart and lung ROIs pixel-wise based on impedance change direction after the R-wave.
Jang et al.	32647206	Decomposition	CVS	Voltage	N/A	PCA and ICA applied to separate lung and cardiac components; cardiac waveform identified by dominant heart-rate frequency. SV computed as peak-to-valley amplitude of CVS.
Ko et al.	33534400			Voltage	N/A	
Jang et al.	35960417			Voltage	N/A	
Chung et al.	38316842	Other	ECG	Voltage	N/A	Deterministic weight vectors applied to multi-channel impedance data, using ECG timing to isolate heart-related impedance changes

CVS cardiac volume signal, ECG electrocardiogram, ICA independent component analysis, N/A not applicable, PCA principal component analysis, PMID PubMed identification number, ROI region of interest.

This contrast is patient-specific and varies with lung aeration, PEEP, atelectasis, and fluid distribution, meaning that changes in ventilation or pathology may alter both true SV and the gain of the measured signal. In parallel, EIT-based SV measurements are also sensitive to subject-specific factors such as body position, electrode placement, chest geometry, and electrode-skin contact, which can shift thoracic structures and further modify baseline impedance distributions^{24,25}. This highlights a common theme in that subject-specific calibration and adjustment for anthropometric differences are needed to compare results across individuals^{30,47}, and can also tighten correlations between EIT-derived SV values and invasive counterparts^{29,30}. With this said, calibration may not remain valid under changing conditions (e.g., postural shifts), and most current approaches rely on retrospective rather than prospective, time-stable adjustments. Further adding to individual variability, different research groups employ varying hardware configurations (Supplementary Tables S1 and S2) and cardiac

signal extraction methods. Future developments must reduce calibration to a short, one-time procedure prior to acquisition or eliminate the need for calibration altogether. In addition, larger clinical trials with participants of diverse ages, ethnicities, body types, and standardized data acquisition and analysis methods may help account for differences across individuals and enable EIT to be used broadly in clinical and community settings.

Finally, one of the largest translational limitations of EIT-based cardiac assessment is that robust validation of SV estimation during extraphysiological hemodynamic perturbations remains largely confined to animal models (Table 5). Protocols such as PEEP titration, induced hemorrhage with/without resuscitation, and vasoactive and/or positive inotropic drug administration have demonstrated strong concordance in porcine studies^{3,16,28–30}, whereas comparable human data are extremely limited (Table 5). The only human perturbation study by Braun et al.²⁶ showed that

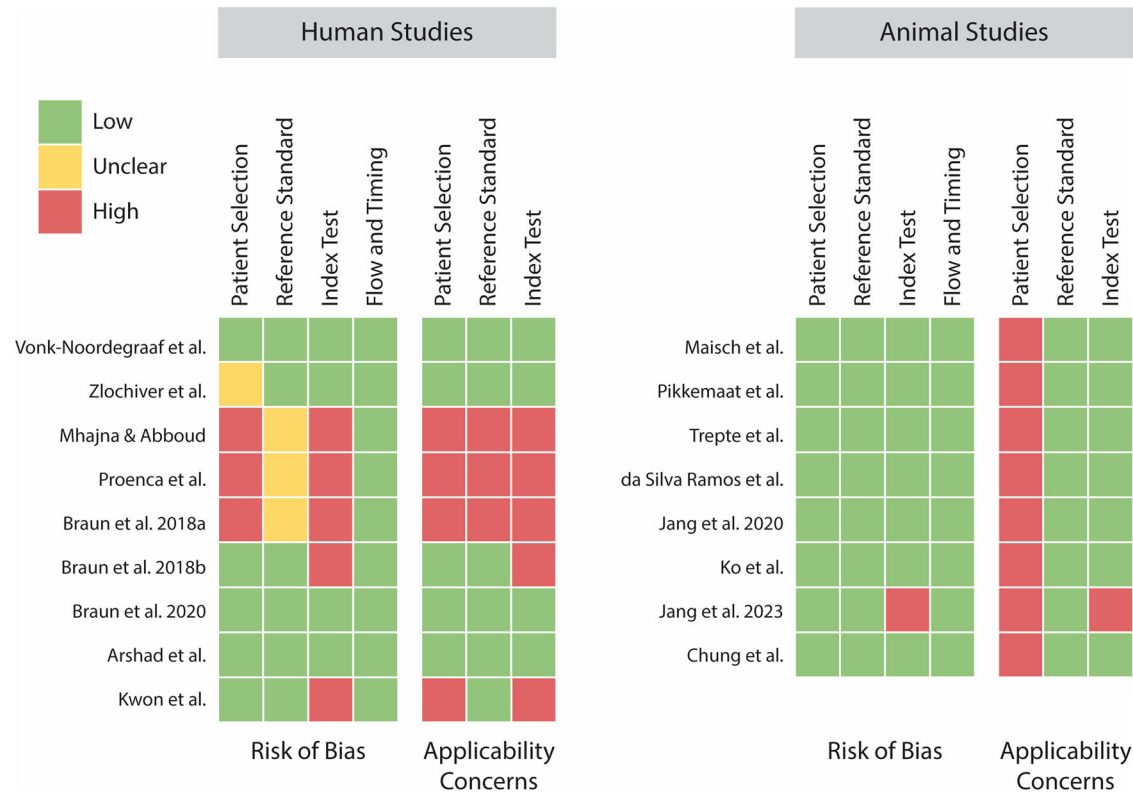


Fig. 4 | QUADAS-2 risk of bias and applicability assessment for the included studies. Risk of bias was evaluated for nine human studies (left) and eight animal studies (right) across four domains: patient selection, index test, reference standard, and flow/timing. Applicability concerns were assessed for the first three domains. Green, yellow, and red colors represent low, unclear, and high risk of bias, respectively.

Table 5 | Summary of available studies on cardiac EIT that utilized extraphysiological challenges and/or iatrogenic insults to induce changes in key parameters of stroke volume

First author	PMID	Species	Type of challenge	Right ventricle			Left ventricle		
				Preload	Contractility	Afterload	Preload	Contractility	Afterload
Maisch et al.	21666450	Pig	PEEP titration	↓	↔	↑	↓	↔	↓
Pikkemaat et al.	24810260	Pig	PEEP titration	↓	↔	↑	↓	↔	↓
da Silva Ramos et al.	28742775	Pig	Pulmonary hemorrhage/resuscitation	↓	↓	↑	↓	↓	Depends ^a
Ko et al.	33534400	Pig	Pulmonary hemorrhage/resuscitation	↓	↓	↑	↓	↓	Depends ^a
Chung et al.	38316842	Pig	Peripheral vasodilators	↓	↔	↓	↓	↔	↓
			Inotrope drugs	↓	↑	↓	↓	↑	Depends ^b
da Silva Ramos et al.	28742775	Pig	Fluid bolus challenge	↑	↑ ^c	Depends ^d	↑	↔	Depends ^e
Braun et al.	31624996	Human	Fluid bolus challenge	↑	↑ ^c	Depends ^d	↑	↔	Depends ^e

PEEP positive end-expiratory pressure, PMID PubMed identification number.
^aTransient sympathetic activation and systemic vasoconstriction in response to hypovolemia, inflammatory response and vasodilation.
^bNet change may depend on drug response and extent of compensatory mechanisms.
^cThrough Frank-Starling mechanism rather than a direct effect on myocardial contractility.
^dNo change or slight decrease due to recruitment of pulmonary capillaries in normal individuals.
^eNo change or slight increase depending on physical effects of fluid bolus, baroreceptor response and sympathetic tone.

lung-based signals outperformed heart-based ROIs during fluid challenge. Those results indicate that the heart-based ROI failed to reliably capture SV, and suggest that clinically useful EIT-derived signals may instead reflect integrated cardiopulmonary interactions rather than direct cardiac volume changes. Notably, clinically relevant protocols such as passive leg raise, PEEP titration, and structured fluid responsiveness testing have not yet been systematically evaluated in humans with simultaneous reference measurements. Moreover, the relevant induced hemodynamic perturbations are more common in animal studies largely because temporally aligned reference standards can be more easily and robustly implemented and executed. By contrast, their use in humans is more constrained. For example, invasive techniques such as thermodilution are limited to specific patient populations, and EIT cannot be acquired simultaneously with CMR due to device incompatibility. Together, these limitations represent a major

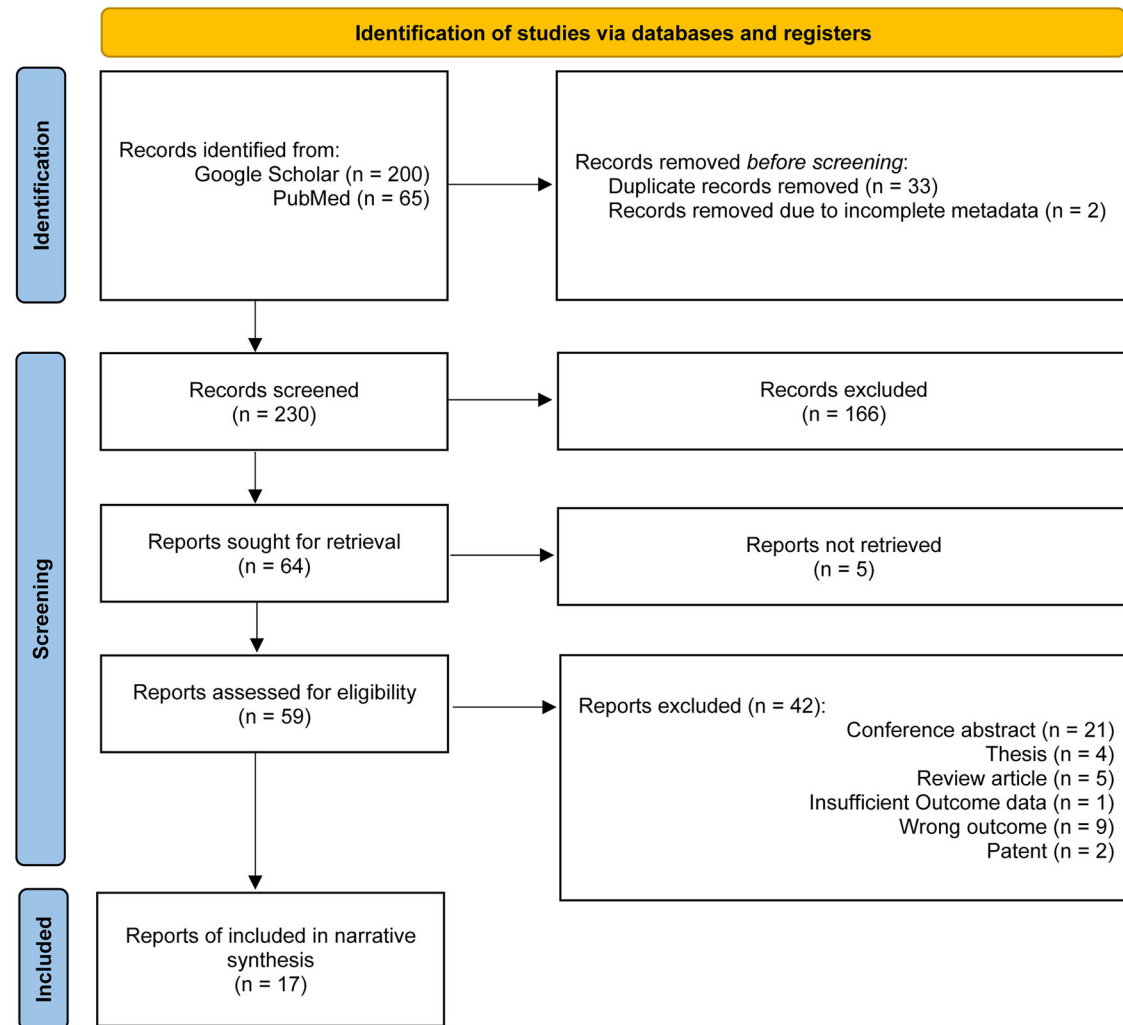


Fig. 5 | PRISMA 2020 flow diagram illustrating the process of study identification, screening, eligibility assessment, and inclusion in the current systematic review. A total of 265 articles were initially identified via PubMed and Google Scholar. After screening and exclusions, 17 studies met the inclusion criteria.

barrier to clinical translation and suggest that current performance cannot prove readiness for routine clinical use.

This systematic review has several limitations that should be considered. First, we have relied on PubMed and Google Scholar for our literature query and review. While the use of Google Scholar was pragmatic and provided extra reassurance to the query results from PubMed, it may have variable reproducibility over time due to the nature of the search engine or index, and this may represent a potential source of selection bias. Second, there is notably a limited number of studies that have explored the use of EIT for SV estimation. Within this small body of literature, there existed large variability across the included studies in domains such as subject population, SV reference standards, and technical EIT parameters (Supplementary Tables S1 and S2). The diversity in study methodologies precluded a quantitative meta-analysis and limits the ability to compare results across studies. Furthermore, the risk of bias varied across the included studies with human simulation work raising concerns over their real-world relevance as well as the usefulness of simulation-based protocols. Nevertheless, the use of *in silico* models can provide a wide range of testing parameters and conditions to determine the extents and limits of cardiac EIT. Animal studies using porcine models have also raised concerns regarding generalizability and translation to human. This risk assessment reiterates the importance of future research that both improves methodological rigor and clinical relevance. Collectively, these limitations highlight the need for future research

that includes larger, multi-center validation studies that utilize standardized protocols for EIT acquisition, SV estimation, and result reporting.

Methods

Search strategy

A systematic query using the search terms, “electrical impedance tomography” and “stroke volume”, was conducted in PubMed and Google Scholar with no restrictions on publication year up to October 31, 2025, to identify studies evaluating the use of EIT for estimating SV of the heart. For the latter, the first 200 records ranked by relevance were screened since relevance declined rapidly beyond this point; spot-checks past 200 did not yield additional eligible studies beyond those already captured with PubMed. Reference lists of included studies were also checked to identify additional reports.

Eligibility criteria

Studies were eligible if they met the inclusion and exclusion criteria. The inclusion criteria were original research involving the use of EIT to estimate SV; human participants, preclinical animal models, or numerical simulations; quantitative results involving SV estimation, including comparisons to a reference standard or validation metric; sufficient methodological details for assessment. The exclusion criteria were non-English publications; studies not utilizing EIT or estimating SV; studies using EIT for only non-

cardiac (e.g., pulmonary, neurologic) applications; conference abstracts, theses or dissertations, review articles, and patents.

Study selection

All records identified from PubMed and Google Scholar were reviewed. Duplicates and records with insufficient metadata were removed. Titles and abstracts were screened independently by two reviewers to exclude clearly irrelevant studies (e.g., those not involving EIT or not addressing SV). Full-text reports were then retrieved for potentially relevant studies. Reports were assessed for eligibility according to the predefined eligibility criteria. Reasons for exclusion at the full-text stage included being a conference abstract, thesis, review article, patent, or lacking sufficient or correct outcome data. Any disagreements between reviewers were resolved through discussion. The overall study selection process, including the number of records identified, screened, excluded, and included, is presented in the PRISMA 2020 flow diagram in Fig. 5.

Data extraction

Key study characteristics were extracted from each article, including study design (human [in vivo or in silico] or animal); subject population and size (e.g., healthy controls, patients, porcine models); SV reference standard used (e.g., TTE, CMR, thermodilution, oxygen uptake); main EIT methodology (e.g., cardiac signal extraction, SV calculation); outcome measures (e.g., correlation, Bland-Altman quantitative parameters, including mean bias and limits of agreement [with 95% confidence interval, when available], and concordance rates from trending analysis [four-quadrant plots]). Reference standards were considered valid if they represented an established clinical or experimental method for SV estimation. For correlation analyses, all “R” values were converted to “R²” to ensure consistency. For Bland-Altman plots, if parameters were not reported in the text, they were approximated from the plot itself. Data were extracted by one reviewer and independently cross-checked by a second to ensure accuracy and consistency.

Synthesis approach

Due to variability across the included studies in terms of subject population, reference standard, and outcome measures, a meta-analysis was not feasible. Instead, a structured narrative synthesis was conducted where studies were grouped by subject type (human or animal) and EIT-based SV estimation method. Where applicable, studies were also stratified by reference standard used (e.g., thermodilution, TTE, pulse contour analysis) to contextualize differences in validation outcomes.

Study quality assessment

The quality and risk of bias of the included studies were assessed using the QUADAS-2 tool designed to evaluate diagnostic accuracy of studies⁴⁸. QUADAS-2 assesses risk of bias across four key domains: (1) patient selection, (2) index test, (3) reference standard, and (4) flow and timing. Each domain was evaluated for risk of bias and applicability concerns. A single reviewer conducted the assessments using a standardized interpretation of QUADAS-2 tailored to studies evaluating EIT as an index test for SV estimation. Simulation studies were included in the risk assessment where appropriate, though some QUADAS-2 domains (e.g., patient selection) were not applicable and are noted accordingly.

Data availability

No datasets were generated or analyzed during the current study.

Received: 20 January 2026; Accepted: 18 August 2026;

Published online: 26 August 2026

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Acknowledgements

E.F. is grateful for support from HKUST's Discretionary Research Fund (Project R9570); Hong Kong Cyberport; the Grant for Good of Oxfam Hong Kong and the Social Innovation Entrepreneurship Development (SIE) Fund, Digital Policy Office of the Government of the Hong Kong Special Administrative Region of China. B.J.E., M.L., J.Q.J.G., and R.W.C. are grateful for the support from E-SENSE Innovation & Technology internal funds, Mitzi, Lee Leung Yin Ping Charitable Foundation, and Mitzi Lee Leung Yin Ping & Family Charitable Foundation, Inc.

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Competing interests

R.W.C. is a co-founder and shareholder of E-SENSE Innovation & Technology, Hong Kong, China. B.J.E. is a contractor and shareholder of E-SENSE Innovation & Technology, Hong Kong, China. The other authors do not have a competing interest.

Additional information

Supplementary information The online version contains supplementary material available at <https://doi.org/10.1038/s41746-026-03172-6>.

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