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Advanced Haemodynamic Monitoring and Clinical Outcome Using Uncalibrated Technologies in TAVI (AHEMO-TAVI)

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List of Abbreviations

ANOVA	analysis of variance
APCO	arterial pressure-based cardiac output
ARDS	acute respiratory distress syndrome
AS	aortic (valve) stenosis
ASA	American Society of Anesthesiologists
AUC	area under the curve
AV	aortic valve
AVA	aortic valve area
AVI	aortic valve implantation
BA-RM	repeated-measures Bland–Altman approach
BAV	bicuspid aortic valve
BMI	body mass index
BNP	B-type natriuretic peptide
CABG	coronary artery bypass graft
CAVS	calcific aortic valve stenosis
CCC	concordance correlation coefficient (Lin's)
CI	cardiac index
CMR	cardiac magnetic resonance
CO	cardiac output
COPD	chronic obstructive pulmonary disease
CS	conscious sedation
CVP	central venous pressure
DIA	diastolic arterial blood pressure
DSE	dobutamine stress echocardiography
dZ/dt	rate of change in thoracic electrical bioimpedance
EC	Electrical Cardiometry
ECG	electrocardiogram
eCRF	electronic case report form
EPCO	European Perioperative Clinical Outcome
EuroSCORE	European System for Cardiac Operative Risk Evaluation
FCM	finger-cuff monitoring
GA	general anaesthesia
GLS	global longitudinal strain
HFpEF	heart failure with preserved ejection fraction
HFrEF	heart failure with reduced ejection fraction
ICD	implantable cardioverter-defibrillator
ICG	impedance cardiography
ICU	intensive care unit

IQR	interquartile range
LOA	limits of agreement
LV	left ventricle
LVEF	left ventricular ejection fraction
LVOT	left ventricular outflow tract
MAC	minimum alveolar concentration
MACCE	major adverse cardiac and cerebrovascular events
MAP	mean arterial pressure
NIBP	non-invasive blood pressure
NSTEMI	non-ST-elevation myocardial infarction
NT-proBNP	N-terminal pro-B-type natriuretic peptide
PACU	post-anaesthesia care unit
PCA	pulse contour analysis
PE	percentage error (Critchley)
PGmean	mean transvalvular pressure gradient
PPV	pulse pressure variation
PR	pulse rate
SAVR	surgical aortic valve replacement
SD	standard deviation
SD_RM	standard deviation of the variance-components limits of agreement
SpO_2	peripheral oxygen saturation (pulse oximetry)
SPI	systolic pressure-time integral
SQI	signal quality index
STEMI	ST-elevation myocardial infarction
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
STS-PROM	Society of Thoracic Surgeons Predicted Risk of Mortality
SV	stroke volume
SVi	stroke volume index
SVR	systemic vascular resistance
SVV	stroke volume variation
SYS	systolic arterial blood pressure
TAVI	transcatheter aortic valve implantation
TEB	thoracic electrical bioimpedance
TWA	time-weighted average
VARC-3	Valve Academic Research Consortium 3
VHD II	EURObservational Research Programme Valvular Heart Disease II Survey
Vmax	peak transvalvular velocity

1. Abstract

Background and Aims: Advanced haemodynamic monitoring during transcatheter aortic valve implantation (TAVI) is challenging due to rhythm disturbances and abrupt changes in afterload and cardiac output, especially at the time of valve deployment. Whilst invasive uncalibrated arterial pulse contour analysis (PCA) is widely applied as the standard of care, non-invasive alternatives such as Electrical Cardiometry (EC) and finger-cuff monitoring (FCM) offer a less invasive approach. No study has to date simultaneously compared all three modalities under real-world TAVI conditions. The primary aim of this study was to assess the accuracy and precision of EC and FCM in comparison with invasive PCA as the reference method. The secondary aim was to evaluate the trending ability of each modality during critical procedural phases.

Methods: This was a prospective, single-centre observational study conducted at the Heart Centre of the University Hospital of Cologne. 59 patients scheduled for elective TAVI were enrolled; 49 were included in the final analysis. All three monitoring modalities were applied simultaneously throughout the procedure. Static agreement was assessed by Bland-Altman analysis; the Critchley percentage error (PE) was used as the primary measure of interchangeability for cardiac index (CI). Blood pressure agreement was benchmarked against ANSI/AAMI/ISO 81060-2 criteria. Trending ability was assessed using four-quadrant concordance analysis.

Results: PE for CI was 56.4% for FCM and 80.2% for EC, both substantially exceeding the 30% interchangeability threshold. Both modalities systematically underestimated CI relative to PCA, with mean biases of −0.51 and −0.75 $L \cdot min^{-1} \cdot m^{-2}$, respectively. Variance-components analysis indicated that disagreement was driven predominantly by within-patient beat-to-beat variability. FCM failed ANSI/AAMI/ISO 81060-2 criteria for all three blood pressure variables across all clinically relevant pressure strata; a directional inversion of mean arterial pressure bias was observed, with overestimation in the hypotensive and underestimation in the hypertensive stratum. Four-quadrant concordance for CI was 43.9% (FCM) and 43.0% (EC), both below the level expected from chance alone; MAP concordance was 53.6%. Whilst cohort-level hypotension exposure was broadly comparable between FCM and PCA, individual-level agreement was insufficient, with concordance correlation coefficients of 0.38 to 0.58.

Conclusion: Neither Electrical Cardiometry nor finger-cuff monitoring achieved clinically acceptable agreement with invasive pulse contour analysis under real-world TAVI conditions. In their current form, neither device is suitable as a standalone replacement for invasive haemodynamic monitoring during TAVI. Continued refinement of both procedural techniques and device algorithms may improve the role of non-invasive monitoring in future minimalist TAVI concepts.

1.1. Zusammenfassung

Hintergrund und Zielsetzung: Erweitertes hämodynamisches Monitoring während Transkatheter-Aortenklappenimplantationen (TAVI) ist aufgrund von Rhythmusstörungen sowie abrupten Änderungen von Nachlast und Herzzeitvolumen besonders während der Klappenfreisetzung anspruchsvoll. Während die invasive unkalibrierte arterielle Pulskonturanalyse (PCA) als Standardverfahren weit verbreitet ist, bieten nicht-invasive Alternativen wie die Elektrische Kardiometrie (EC) und das Fingermanschetten-Monitoring (FCM) einen weniger invasiven Ansatz. Bislang fehlt ein simultaner Vergleich aller drei Modalitäten unter realen TAVI-Bedingungen. Primäres Ziel dieser Studie war die Beurteilung von Genauigkeit und Präzision der EC und des FCM im Vergleich zur invasiven PCA als Referenzverfahren. Sekundäres Ziel war die Bewertung der Trenderfassung der einzelnen Verfahren während kritischer Phasen der Prozedur.

Methoden: Es handelt sich um eine prospektive, monozentrische Beobachtungsstudie, die am Herzzentrum der Universitätsklinik Köln durchgeführt wurde. Es wurden 59 elektive TAVI PatientInnen eingeschlossen; 49 gingen in die abschließende Analyse ein. Alle drei Monitoring-Verfahren wurden während des gesamten Eingriffs simultan angewendet. Die statische Übereinstimmung wurde mittels Bland-Altman-Analyse beurteilt; der Critchley-Prozentfehler (PE) diente als primäres Maß der Austauschbarkeit für den Herzindex (CI). Die Blutdruckübereinstimmung wurde anhand der Kriterien der ANSI/AAMI/ISO 81060-2 bewertet. Die Trenderfassung wurde mittels Vier-Quadranten-Konkordanzanalyse beurteilt.

Ergebnisse: Der PE für den CI betrug 56,4 % für das FCM und 80,2 % für die EC und überschritt damit den 30%-Grenzwert nach Critchley deutlich. Beide Modalitäten unterschätzten den CI systematisch gegenüber der PCA, mit mittleren Abweichungen von −0,51 bzw. −0,75 $L \cdot min^{-1} \cdot m^{-2}$. Die Varianzkomponentenanalyse zeigte, dass die Abweichungen überwiegend durch intraindividuelle Schlag-zu-Schlag-Variabilität bedingt waren. Das FCM konnte die ANSI/AAMI/ISO 81060-2-Kriterien für alle drei Blutdruckvariablen in keinem der klinisch relevanten Druckbereiche erfüllen; für den mittleren arteriellen Druck wurde eine Inversion des Fehlers beobachtet, mit Überschätzung im hypotensiven und Unterschätzung im hypertensiven Bereich. Die Vier-Quadranten-Konkordanz für den CI lag mit 43,9 % (FCM) und 43,0 % (EC) unterhalb des Zufallsniveaus; die MAP-Konkordanz betrug 53,6 %. Obwohl das Ausmaß periinterventioneller Hypotonieepisoden auf Gruppenebene zwischen FCM und PCA vergleichbar war, blieb sie auf Patientenebene unzureichend, mit Konkordanz-Korrelationskoeffizienten zwischen 0,38 und 0,58.

Schlussfolgerung: Weder die EC noch das FCM erreichten unter realen TAVI-Bedingungen eine klinisch akzeptable Übereinstimmung mit der invasiven PCA. In ihrer derzeitigen Form ist keines der beiden Verfahren als eigenständiger Ersatz für das invasive hämodynamische Monitoring während der TAVI geeignet. Eine weitere Optimierung sowohl der interventionellen Techniken als auch der Gerätealgorithmen könnte die Rolle des nicht-invasiven Monitorings in künftigen Konzepten der minimalistischen TAVI verbessern.

2. Introduction

2.1. Aortic Valve Stenosis

Aortic valve stenosis (AS) is the most common valvular heart disease in industrialised countries with its prevalence increasing with age. Its clinical and socioeconomic relevance is further increased by an ongoing demographic ageing [1, 2]. The introduction and development of minimally invasive procedures such as the transcatheter aortic valve implantation (TAVI) have substantially transformed the management of AS over the past two decades.

2.1.1. Epidemiology and Aetiology

More than 2% of the population are affected by valvular heart disease [4]. Within this group the AS is the most common primary valve lesion admitted for intervention [2]. During the EURObservational Research Programme Valvular Heart Disease II Survey (VHD II), a prospective, multicentre observational study conducted in 2017 involving 5,219 patients with severe native valvular heart disease across 28 European countries, AS accounted for 41.2% of all cases, ahead of mitral regurgitation (21.3%), aortic regurgitation (5.3%) and mitral stenosis (4.5%) [3]. Compared with the first Euro Heart Survey in 2001, in which AS accounted for 33.9% of cases, this share has since risen significantly, which is probably due to the ageing of the population [3]. The median age of patients with AS in the VHD-II Survey was 76 years (IQR 67-83), 37.6% were aged 80 or over, up from 8.3% in 2001 [3].

This trend is also reflected in the reality of cardiological and cardiac surgical care in Germany. In 2024, 21,299 TAVI procedures were performed, which accounts for 71.2% of all isolated aortic valve procedures [5].

With regard to aetiology, 67.8% of all native valvular heart diseases in the VHD-II Survey showed a degenerative aetiology. In Western Europe, this proportion was as high as 81.3% [3]. The 2025 ESC/EACTS guidelines on the management of valvular heart disease confirm that degenerative calcific aortic valve stenosis (CAVS) is the leading cause in high-income countries and that its prevalence is increasing rapidly due to the ageing population [2]. Congenital valve defects rank second, particularly the bicuspid aortic valve (BAV), which, with a prevalence of around 1% in the population, is the most common congenital heart defect [6, 7, 9]. It is the dominant morphology among younger patients requiring aortic valve surgery and is frequently associated with dilatation of the aortic root or the ascending aorta [2, 7]. In low- and middle-income countries, the rheumatic aetiology remains significant, with AS typically occurring in combination with rheumatic mitral valve disease [2]. In Europe, however, the rheumatic aetiology has become rare: in the VHD II Survey, its prevalence in Western Europe was only 4.0%, whereas it reached 72% in North Africa [3].

2.1.2. Pathophysiology

Our understanding of the pathophysiology of the CAVS has evolved considerably in recent years. Whilst long assumed to be a passive, age-related degenerative process, histopathologic and clinical data now point to an active and multifaceted process [6]. Recent data have shown that oxidation and lipid infiltration of the aortic valve generate several bioactive lipid-species inducing inflammation of the aortic valve. The subsequent activation of multiple partially convergent signalling pathways - including the NOTCH1/Wnt/β-Catenin axis, BMP2/RUNX2 and the RANKL/RANK/OPG system - disrupts normal valve biology and promotes progressive fibro-calcific remodelling of the leaflets [6].

The pathophysiology of BAV differs fundamentally from CAVS. It develops embryologically from fusion of two aortic valve cushions. This abnormal leaflet geometry leads to turbulent, asymmetric flow with accelerated mechanical stress, promoting dystrophic calcification and fibro-calcific remodelling typically one to two decades earlier than in tricuspid valves [6, 8]. BAV has a significant genetic component, with NOTCH1 and MUC4 mutations identified as risk factors among others, and screening of first-degree relatives is recommended per AHA/ACC 2022 guidelines [6, 8]. Notably, the BAV is not solely a condition of the valve but also of the ascending aorta. Due to intrinsic degenerative changes of the tunica media a significant proportion of BAV patients develop progressive dilatation of the ascending aorta and show a relevant risk for aneurysm formation and aortic dissection [8].

Lastly, rheumatic AS results from valvular damage following acute rheumatic fever, itself a consequence of an untreated group A *Streptococcus* infection. Unlike CAVS, the rheumatic AS is characterised by a commissural fusion of the leaflets, typically producing a combined valvular lesion. Isolated aortic valve involvement is uncommon; the mitral valve is also affected in the majority of cases. Whilst increasingly rare in developed countries, rheumatic heart disease remains a relevant cause of AS in resource poor settings [6].

Regardless of the underlying aetiology, progressive obstruction of the left ventricular outflow tract (LVOT) with a resulting increased transvalvular pressure gradient leads to chronic pressure overload of the left ventricle (LV), triggering cardiomyocyte hypertrophy and the addition of sarcomeres, thus increasing myocyte width. The resulting increase in wall thickness helps normalise wall stress and maintain ejection performance. This process is accompanied by an alteration of the connective tissue as well as the capillary and nerve networks through diffuse interstitial fibrosis by increased collagen synthesis. This interstitial fibrosis may be reversible following aortic valve replacement [6].

AS severity, age, sex, and comorbidities such as hypertension, metabolic syndrome and coronary artery disease influence the adaptation of the LV. Women predominantly develop concentric remodelling, whilst men tend to show eccentric hypertrophy. Concentric remodelling or hypertrophy are associated with worse myocardial function and increased risk of adverse cardiac events [6, 9].

Increased LV muscle mass leads to impaired coronary flow reserve, due to reduced coronary arteriolar density and increased transmural pressure [10, 11]. The resulting lack of

adaptability to increased oxygen demand leads to recurring exhaustion of this reserve and ultimately to myocardial ischaemia, myocyte apoptosis and replacement fibrosis. This mechanism explains why patients with severe AS may present with angina pectoris despite angiographically normal coronary arteries [6,12].

Because of increased wall thickness, wall stress remains relatively normal even in patients with severe AS, resulting in a generally well-preserved LV ejection fraction (LVEF). Impairment of LVEF or cardiac output (CO) therefore tends to become apparent only in end-stage disease [6].

2.1.3. Diagnosis

First signs of suspected AS may include a systolic murmur on auscultation or more advanced symptoms such as dizziness, dyspnoea, angina pectoris or syncope. The standard diagnostic tool for confirmation of diagnosis, quantification of severity and assessment of haemodynamic parameters is echocardiography. Whilst most patients are diagnosed prior to the onset of clinical symptoms, approximately 5-10% first present at an advanced stage of the disease with signs of heart failure [6].

In accordance with the 2025 ESC/EACTS Guidelines for the management of valvular heart disease, three echocardiographic parameters allow an assessment of AS severity: the mean transvalvular pressure gradient (PGmean), the peak transvalvular velocity (Vmax) and the effective aortic valve area (AVA) [2]. When these parameters are concordant, severe AS is defined by a PGmean ≥40 mmHg, Vmax ≥4.0 m/s and AVA ≤1.0 cm² (or ≤0.6 cm²/m² indexed to body surface area), irrespective of LV function and flow conditions [2].

When echocardiographic parameters are discordant, the guidelines distinguish four subtypes based on stroke volume index (SVi) and LVEF as shown in Table 1. Dobutamine stress echocardiography can help discriminate between true and pseudo-severe AS in low-flow, low-gradient AS with reduced LVEF if flow reserve is detected. Normal-flow, low-gradient AS with preserved LVEF usually shows moderate stenosis and discordant high-gradient AS is considered severe if not caused by a reversible high-flow status [2].

Subtype	PGmean	AVA	SVi	LVEF
LF/LG AS with reduced LVEF	<40 mmHg	≤1.0 cm²	≤35 ml/m²	<50%
LF/LG AS with preserved LVEF	<40 mmHg	≤1.0 cm²	≤35 ml/m²	≥50%
NF/LG AS with preserved LVEF	<40 mmHg	≤1.0 cm²	>35 ml/m²	≥50%
Discordant high-gradient AS	≥40 mmHg	>1.0 cm²	any	any

Table 1: Discordant subtypes of AS according to the 2025 ESC/EACTS Guidelines for the management of valvular heart disease [2]. *LF/LG. low-flow/low-gradient; NF/LG, normal-flow/low-gradient; AS, aortic stenosis; PGmean, mean pressure gradient; AVA, aortic valve area; SVi, stroke volume index; LVEF, left ventricular ejection fraction.*

Cardiac CT calcium scoring provides complementary information and correlates with haemodynamic severity and clinical outcomes [13, 14].

2.1.4. Therapy (SAVR vs. TAVI)

Surgical or interventional replacement of the stenotic valve remains the only effective therapy for severe AS. No pharmacological therapy capable of halting or delaying disease progression is currently available [2]. The decision for intervention is guided by the severity of AS, the presence or absence of symptoms, left ventricular function, and individual clinical and anatomical characteristics, and should be made within a multidisciplinary Heart Team [2].
In symptomatic severe AS, the indication for early intervention is well established for all eligible patients with an estimated life expectancy exceeding one year [2, 15]. The low-flow, low-gradient variant, however, poses a distinct diagnostic and therapeutic challenge.
Regardless of LVEF, intervention is recommended following confirmation of severe AS by CT calcium scoring or dobutamine stress echocardiography. In case of pseudo-severe stenosis, pharmacological heart failure treatment is recommended [2, 22]. Patients with normal-flow, low-gradient AS with preserved LVEF have a similar prognosis to patients with moderate AS and intervention is only indicated after clear diagnosis of severe AS [2, 23, 24].
In asymptomatic patients, exercise testing may unmask symptoms, in which case management follows the recommendations for symptomatic AS. Furthermore, intervention is recommended in patients with asymptomatic severe high-gradient AS and a reduced LVEF below 50% in the absence of another identifiable cause [2].
Notably, growing evidence from recent trials suggests that early intervention is also superior to close clinical surveillance in the remaining population of asymptomatic severe AS [16 - 21].
Concerning moderate AS, intervention is only recommended if patients undergo surgery for other indications, such as CABG, ascending aorta surgery, or other valve disease [2, 25].
Once the decision for intervention has been made, both conventional surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI) are available as treatment options. The field of transcatheter valve therapy has been evolving rapidly, and the indications for TAVI have been progressively expanded over recent years. This shift is reflected in the current ESC/EACTS guidelines: whereas the 2021 iteration recommended TAVI as the preferred approach in patients aged 75 years or older, this threshold has been lowered to 70 years in the 2025 update [2, 26].
Beyond age, individual procedural risk must be incorporated into the decision-making process. Conventional surgical risk scores, including EuroSCORE II and STS-PROM, were developed in surgical populations and have been shown to overestimate procedural risk when applied to TAVI candidates [28 - 30]. Furthermore, clinically relevant comorbidities such as frailty, chronic kidney disease, and concomitant cardiac disease are insufficiently captured by these tools, despite their known impact on outcomes [2, 34]. TAVI-specific risk models have been developed to address these shortcomings, but have yet to achieve widespread

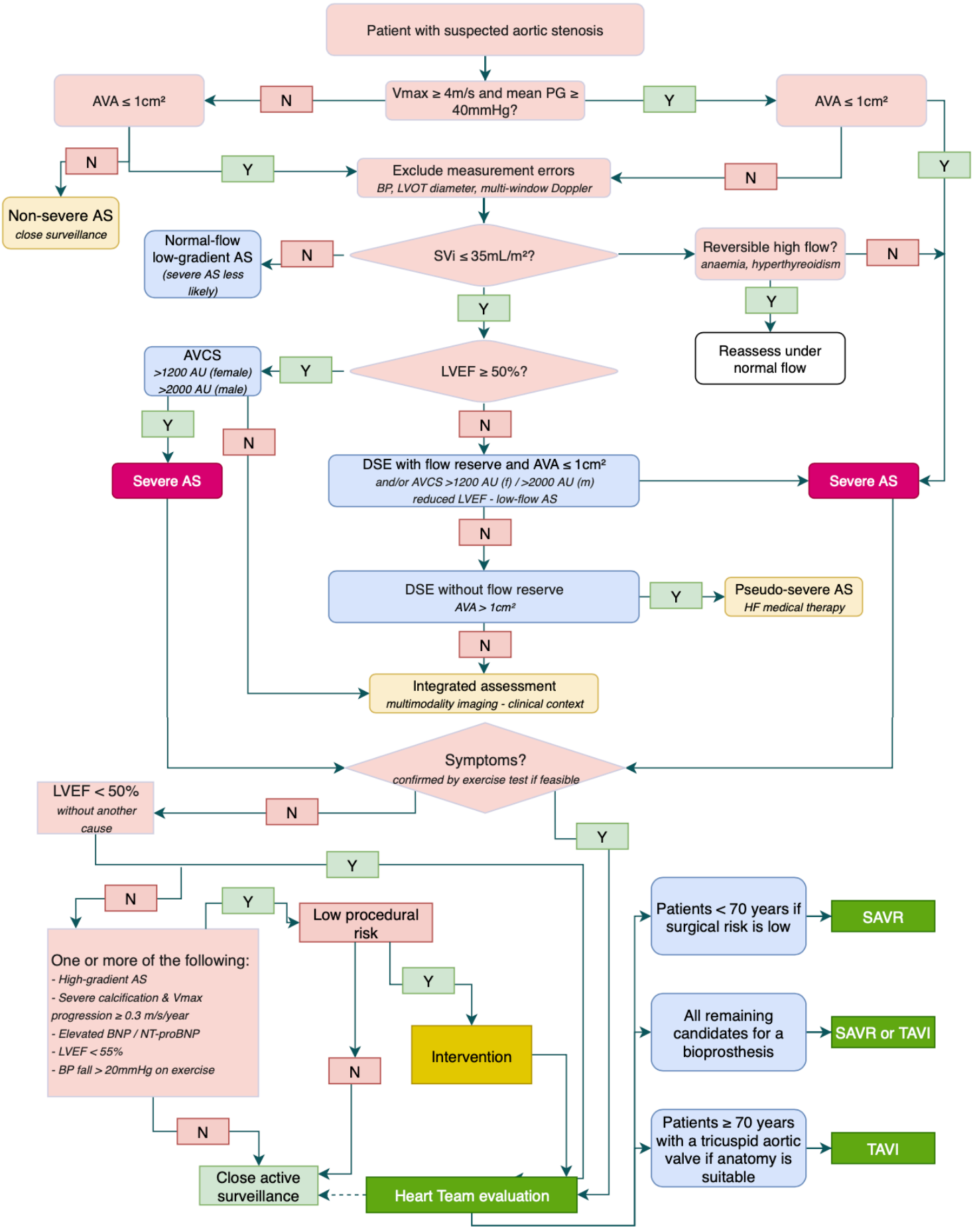


Figure 1: Overview of diagnostic and therapeutic decision-making for aortic stenosis. *Modified from: Praz F et al. 2025 ESC/EACTS Guidelines for the management of valvular heart disease [2]. AS = aortic stenosis; AU = Agatston units; AVA = aortic valve area; AVAi = AVA indexed to body surface area; AVCS = aortic valve calcium score; BP = blood pressure; BNP = B-type natriuretic peptide; DSE = dobutamine stress echocardiography; HF = heart failure; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-BNP; PG = pressure gradient; SAVR = surgical aortic valve replacement; SVi*

adoption in routine clinical practice and show limited predictive performance [31 - 33]. An overview of diagnostic and therapeutic decision-making is shown in Figure 1.

2.2. Haemodynamics

The haemodynamics of the cardiovascular system are determined by a finely balanced interaction of stroke volume, myocardial contractility, vascular resistance, and valve orifice areas. If one of these variables changes acutely or chronically, compensatory adaptations throughout the entire system are required. In aortic stenosis, this interdependence is particularly striking, as the progressive narrowing of the valve orifice area not only alters local flow dynamics but, positioned at the transition from the heart to the systemic circulation, has profound effects on both the upstream and downstream system - with far-reaching consequences for pressure gradients, wall stress, filling dynamics, and the arterial pulse contour.

2.2.1. Haemodynamics with Aortic Stenosis

The stenotic aortic valve represents a fixed flow obstruction within the left ventricular outflow tract. According to the continuity principle, blood must pass through the reduced orifice area at increased velocity. In a healthy aortic valve, systolic flow velocity is typically below 1.5 m/s, whereas in severe AS it may exceed 4 m/s [2, 6]. This flow acceleration is directly associated with a transvalvular pressure gradient, which can be quantified using the simplified Bernoulli equation:

$$\Delta P = 4 \times V^2$$

A peak jet velocity of 4 m/s thus corresponds to a maximum gradient of 64 mmHg. This equation enables non-invasive, Doppler-based gradient assessment, but assumes laminar flow and optimal beam alignment - conditions rarely met in severe AS due to the poststenotic turbulence that develops [36]. Calcified valves with eccentric opening, as seen particularly in BAV, further amplify these asymmetric, helical flow patterns in the ascending aorta [6]. Doppler assessment may therefore underestimate the true pressure gradient, and simultaneous invasive measurement of both LV and central aortic pressures remains the most accurate tool for assessment of the mean transvalvular gradient [36].

To maintain adequate systemic pressure, the LV must generate substantially higher contractile force, resulting in chronic pressure overload with markedly elevated systolic peak pressures, which triggers hypertrophy as an adaptive mechanism (see 2.1.2). However, the hypertrophied, stiffened myocardium exhibits impaired diastolic relaxation, such that LV filling pressures rise progressively and advanced diastolic dysfunction is common even in the asymptomatic stage [6].

The altered LV haemodynamics also shape the arterial pulse pressure profile: pulsus parvus (reduced pulse pressure) and pulsus tardus (delayed pressure upstroke) are signs of fixed

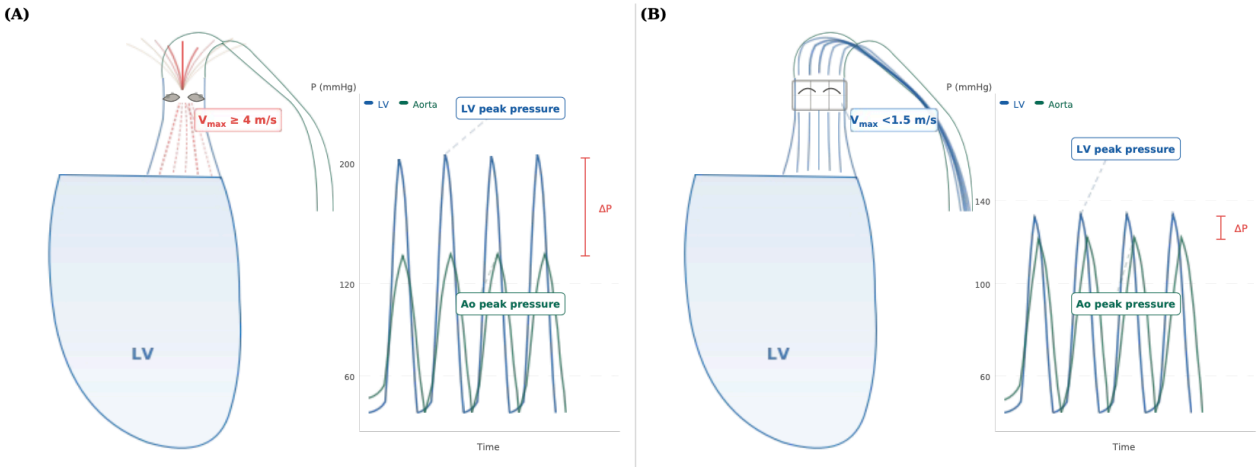


Figure 2: Schematic illustration of haemodynamic changes across the aortic valve before and after TAVI. *Original schematic; pressure waveforms adapted from Graziani et al. [35]. (A) Severe aortic stenosis: turbulent, high-velocity jet (V_{max} ≥ 4 m/s) through the calcified, stenotic valve with markedly elevated left ventricular (LV) systolic pressure and a large pressure gradient (ΔP) between LV and aorta (Ao). (B) After transcatheter aortic valve implantation (TAVI): laminar flow through the prosthetic valve (V_{max} < 1.5 m/s), normalisation of LV peak pressure, and near-equalisation of the transvalvular pressure gradient. Blue, LV pressure; green, aortic pressure.*

outflow obstruction. In the aortic pressure waveform, this manifests as a flattened, slowly rising systolic upstroke with a delayed pressure peak, in contrast to the spike-and-dome morphology seen in dynamic obstruction [36].

2.2.2. Periprocedural Haemodynamic Changes during TAVI

The immediate removal of valvular obstruction via TAVI makes it possible to observe the acute haemodynamic consequences of aortic stenosis directly in the catheterisation laboratory. Graziani et al. investigated invasively measured LV and aortic pressure parameters immediately before and after valve implantation by left heart catheterisation in a prospectively designed registry study of 245 consecutive AS patients [35]. Pre-implantation, the haemodynamic profile reflected severe pressure overload with mean LV peak pressures at 198 ± 37 mmHg and a mean AV gradient of 60 ± 22 mmHg, and the time to aortic peak pressure averaged 285 ms, which is consistent with the typical pulsus tardus described above [35]. More than 70% of patients exhibited advanced diastolic dysfunction (grade II–III), and mean LV end-diastolic pressure was 26 mmHg [35].

Immediately after valve implantation, significant changes were observed across nearly all measured pressure parameters. As a direct expression of afterload relief, LV peak pressure fell to 164 ± 32 mmHg, mean AV gradient to below 7 mmHg, and aortic peak pressure rose from 139 to 158 mmHg [35]. The time to aortic peak pressure shortened to 221 ms, reflecting normalisation of the pulse contour following removal of the obstruction. Notably, LV end-diastolic pressure did not change significantly in the immediate post-procedural period, while LV proto-diastolic pressure increased. This was attributed to transient myocardial stunning from procedural rapid pacing, as well as the inertia of diastolic remodelling relative to the immediate systolic unloading [35]. Contractility and relaxation indices also decreased

significantly after TAVI. Graziani et al. interpret this as evidence that patients with AS and preserved LVEF efficiently recruit their contractile reserve to overcome the elevated afterload [35].

Several studies have demonstrated that acute afterload relief following TAVI leads to immediate improvement of cardiac function, followed by reverse remodelling with regression of LV volumes and mass as well as improvement of both systolic and diastolic function [35, 37-40]. Patients with worse pre-procedural systolic function appear to benefit most in terms of LVEF recovery, and a prompt reduction in pulmonary artery systolic pressure has been observed in the immediate post-procedural period [35].

2.3. Periprocedural Haemodynamic Monitoring Methods

As TAVI procedures become increasingly routine, minimising invasiveness has become a priority for both the procedural technique and anaesthetic management. Consequently, there is growing interest in haemodynamic monitoring modalities that reduce or avoid vascular access. Alternative minimally invasive and non-invasive technologies aim to meet these requirements whilst maintaining sufficient diagnostic accuracy. At the time of writing, the standard TAVI monitoring at our centre comprises basic anaesthetic monitoring with non-invasive blood pressure measurement (NIBP), pulse oximetry (SpO_2), 5-lead ECG, and invasive arterial pressure measurement via a radial artery catheter. In the context of the present study, three advanced haemodynamic monitoring modalities were employed alongside this standard: electrical cardiometry (ICON, Osypka Medical GmbH, Berlin, Germany), uncalibrated arterial pulse contour analysis (FloTrac/Acumen IQ, Edwards Lifesciences, Irvine, CA, USA), and continuous non-invasive finger-cuff monitoring (ClearSight, Edwards Lifesciences, Irvine, CA, USA).

2.3.1. Electrical Cardiometry

Electrical Cardiometry is a non-invasive method for the continuous measurement of various haemodynamic parameters, such as cardiac output (CO), cardiac index (CI), and stroke volume index (SVI), based on thoracic electrical bioimpedance (TEB). The underlying principle of TEB measurement relies on the fact that blood is the most electrically conductive tissue in the thorax, such that an alternating current conducted through the thorax passes predominantly through the aorta. Four surface electrodes are applied to the left side of the neck and the lower thorax. An alternating current of constant amplitude is applied via the two outer electrodes, whilst the two inner electrodes record the resulting voltage and a surface ECG. The ratio of applied current to measured voltage corresponds to the thoracic bioimpedance, which is recorded continuously over time [54].

The model underlying Electrical Cardiometry was introduced in 2001 by Bernstein and Osypka as Electrical Velocimetry and takes a different approach to signal interpretation than

classical impedance cardiography (ICG) [54, 55]. Whereas ICG attributes the characteristic decrease in impedance following aortic valve opening to volumetric expansion of the aorta, the Electrical Velocimetry model interprets this decrease as a consequence of the flow-induced parallel alignment of erythrocytes within the aorta, which varies in accordance with peak aortic blood acceleration [53, 54]. From this signal, the device derives stroke volume as the product of a body weight-based tissue volume constant, a mean blood velocity index, and left ventricular ejection time; cardiac output is subsequently calculated as the product of stroke volume and heart rate [53, 54]. As a flow-based method, Electrical Cardiometry does not yield blood pressure measurements.

Compared with classical ICG, Electrical Cardiometry offers improved accuracy through its revised signal model, particularly in haemodynamically unstable conditions [48]. In a meta-analysis of minimally invasive cardiac output monitoring in paediatric patients, Electrical Cardiometry demonstrated the smallest bias (-0.03 l/min) and lowest percentage error (23.6%) of all devices examined [50]. Validation studies in adult cardiac surgical patients confirm clinically acceptable agreement with thermodilution as the reference method, with percentage error in most studies within or close to the accepted threshold of 30% [48, 49].

The device used in the present study is the ICON monitor (Osypka Medical GmbH, Berlin, Germany).

2.3.2. Uncalibrated Pulse Contour Analysis

Arterial pulse contour analysis provides a minimally invasive approach to continuous cardiac output monitoring. Rather than requiring placement of a pulmonary artery catheter, these systems derive haemodynamic variables from the arterial pressure waveform obtained via a standard arterial line. Depending on whether an independent reference method (e.g. transpulmonary thermodilution or Doppler) is used for initial calibration, pulse contour systems are classified as either calibrated or uncalibrated [47].

The physiological considerations used for uncalibrated pulse contour analysis are based on the Windkessel model first described by Otto Frank in 1899, which conceptualises the arterial system as an elastic reservoir dampening the pulsatile output of the left ventricle into a more continuous peripheral flow [58, 47]. To estimate cardiac output (CO), these systems use a flow-time integral to calculate stroke volume (SV), which is then multiplied with the heart rate. To derive flow from a pressure curve, arterial impedance and arterial compliance are incorporated into the algorithm. Since neither impedance nor compliance can be measured non-invasively, all uncalibrated pulse contour systems must estimate these parameters from surrogate data. In this regard, most commercially available systems use nomograms based on demographic parameters such as age, sex, height, and weight [47]. Beyond CO and the derived cardiac index (CI), these systems continuously provide SV and its index SVI, as well as the dynamic preload parameters pulse pressure variation (PPV) and stroke volume variation (SVV). Continuous measurement of systemic vascular resistance (SVR) is also

available, given a central venous line in situ, since its calculation requires central venous pressure (CVP).

A good arterial waveform signal is crucial for reliable measurements. Both overdamping and underdamping degrade the input signal and may lead to inaccurate CO estimation. The responsibility for ongoing assessment of an adequate waveform lies with the user [47]. Also, states of low SVR such as sepsis or chronic hepatic disease seem to lead to poor concordance with thermodilution reference methods [47], while elevated SVR, as encountered in low-output states or high-dose vasopressor therapy, leads to overestimation of CO [56, 57].

Haemodynamic monitoring by uncalibrated pulse contour analysis in the present study was performed using the Acumen IQ sensor connected to the Hemosphere Advanced Monitor (both Edwards Lifesciences, Irvine, CA, USA, distributed by BD, Franklin Lakes, NJ, USA).

2.3.3. Continuous Non-Invasive Pulse Contour Analysis

Continuous non-invasive arterial pressure monitoring via a finger cuff represents a fully non-invasive approach to haemodynamic monitoring, requiring neither arterial cannulation nor venous access. The technique is based on the volume clamp method, first described by Peñáz in 1973, to derive a continuous beat-to-beat arterial pressure waveform from the finger using photoplethysmography combined with an inflatable finger cuff [46, 59, 63]. An infrared light source and detector continuously measure the diameter of the finger artery. The cuff pressure is adjusted at high frequency to maintain a constant arterial volume, such that the cuff pressure equals the arterial pressure and the arterial wall remains in its unloaded state [46, 63]. To maintain this unloaded state during changes in vascular tone, all systems employ a periodic physiological recalibration of the unloaded volume [46, 60, 61]. The resulting cuff pressure signal thus reflects the arterial pressure waveform in real time [46, 63]. Two commercially available systems based on this principle are the ClearSight system (Edwards Lifesciences, Irvine, CA, USA) and the CNAP system (CNSystems Medizintechnik, Graz, Austria), which differ primarily in their approach to vascular tone compensation and brachial pressure reconstruction [63]. Once the finger arterial pressure waveform has been reconstructed to brachial level, stroke volume and cardiac output are derived using a pulse contour algorithm analogous in principle to the minimally invasive methods described in section 2.3.2 [62]. Beyond arterial pressure, these systems provide CI, SVI, SVV, and SVR, making them comprehensive non-invasive haemodynamic monitoring platforms [42].

Validation of the volume clamp method against invasive arterial pressure has shown acceptable agreement under haemodynamically stable conditions, with Bogert et al. reporting a percentage error within the 30% interchangeability threshold for CO measurement against pulmonary artery thermodilution [60], and Vos et al. demonstrating acceptable agreement of finger cuff MAP measurement with invasive radial artery pressure, with a percentage error of 22% [46]. In the TAVI context specifically, though, Stolte et al.

found that CO, CI, and SV were consistently lower than invasive measurements with a mean percentage error of 52%, exceeding the accepted threshold for interchangeability, whilst continuous trending of haemodynamic changes remained feasible [42]. In a preliminary report, Rittershofer et al. demonstrated acceptable MAP concordance before and after valve deployment, but identified a mean delay of 6.13 seconds in signal restoration following rapid pacing compared with 1.0 second for the invasive method [41].

Haemodynamic monitoring by continuous non-invasive finger-cuff measurement in the present study was performed using the Acumen IQ cuff connected to the Hemosphere Advanced Monitor (both Edwards Lifesciences, Irvine, CA, USA, distributed by BD, Franklin Lakes, NJ, USA).

2.3.4. Limitations of Advanced Haemodynamic Monitoring in the TAVI Setting

As discussed in section 2.3.2, pulse contour analysis requires a stable and well-defined arterial waveform for accurate parameter estimation. Conditions that alter waveform morphology or cardiac rhythm therefore compromise the validity of area-under-the-curve-based calculations. Although limited data suggest that finger-cuff blood pressure monitoring may remain feasible during atrial fibrillation [64], and the FloTrac algorithm, owing to its use of pulse rate rather than ECG-triggered beat detection, explicitly states that arrhythmias do not constitute a limitation for cardiac output calculation [70], the general consensus remains that pulse contour-based methods in general are inherently susceptible to rhythm irregularities, as beat-to-beat variability in stroke volume and pulse pressure may introduce systematic error into derived flow parameters such as cardiac index.

The TAVI procedure, however, presents haemodynamic challenges that extend beyond rhythm irregularity alone. Rapid ventricular pacing and valve deployment produce abrupt, profound changes in stroke volume, arterial pressure and afterload that are qualitatively different from the beat-to-beat variability of atrial fibrillation. Ventricular extrasystoles during catheter manipulation are common, and the accuracy of haemodynamic monitoring during rapid pacing and valve deployment remains a central question for all modalities under investigation. As noted in section 2.3.3, Rittershofer et al. documented a measurable signal recovery delay in non-invasive pulse contour analysis following rapid pacing [41], indicating that the immediate post-pacing recovery phase may not be accurately captured. Furthermore, in TAVI patients managed without central venous access, SVR cannot be directly calculated, as outlined in section 2.3.2. SVR values are therefore estimates based on assumed rather than measured filling pressures, representing a technical limitation common to all pulse contour-based modalities in this study.

Electrical Cardiometry, while based on a fundamentally different physical principle, is similarly affected by rhythm disturbances. The algorithm relies on ECG-triggered, beat-to-beat identification of defined reference points in the impedance waveform, and irregular rhythms

with variable ventricular filling and ejection directly compromise the accuracy of this identification and the derived stroke volume. Additionally, the presence of severe aortic stenosis itself has been identified as a potential source of measurement error for electrical velocimetry-based cardiac output estimation [65], which is of particular relevance given the patient population under investigation.

These procedural conditions pose challenges not only for the modalities under investigation, but also for thermodilution-based reference methods, which are known to be inaccurate in the presence of irregular rhythms and low cardiac output states [36]; indeed, thermodilution carries an inherent measurement variability of approximately 20% even under stable conditions [65, 66], and its limitations as a reference standard have been explicitly discussed in the context of bioimpedance validation studies [67].

2.4. Clinical Outcome

The assessment of clinical outcome in the present study follows the European Perioperative Clinical Outcome (EPCO) framework published by Jammer et al. on behalf of the ESA-ESICM joint taskforce, which provides standardised definitions for adverse events and composite outcome measures applicable to periprocedural medicine research [68]. The EPCO framework encompasses individual adverse event definitions including acute kidney injury, stroke, myocardial infarction, and respiratory complications, as well as composite endpoints and measures of healthcare resource utilisation such as ICU length of stay and total hospital stay. This framework was chosen in preference to the VARC-3 endpoint definitions - the established standard for dedicated TAVI outcome trials [69] - as the primary aim of this study is haemodynamic monitoring comparison rather than structured valve outcome assessment. Comprehensive 30-day echocardiographic follow-up, as required for full VARC-3 adjudication, was not available in the present cohort.

Outcome data were collected for the duration of the in-hospital stay and adjudicated according to the EPCO definitions.

2.5. Aims and Objective

Periprocedural haemodynamics during TAVI are characterised by abrupt and clinically significant changes in afterload, stroke volume, and cardiac output, particularly during rapid pacing and valve deployment. Whilst invasive arterial pressure monitoring combined with uncalibrated pulse contour analysis is widely applied as the standard of care [41, 42], non-invasive modalities such as Electrical Cardiometry and finger-cuff monitoring offer a less invasive approach. Both systems are easily deployable, carry fewer vascular risks, and enable continuous haemodynamic monitoring. The suitability of these alternatives under highly dynamic periprocedural conditions like during TAVI procedures has not been comprehensively evaluated, and existing data show heterogeneous results. In particular, no

study to date has simultaneously compared all three modalities under real-world TAVI conditions, leaving a clear gap regarding their relative accuracy, precision, and clinical utility.

The primary aim of this study was therefore to assess the accuracy and precision of Electrical Cardiometry and finger-cuff monitoring in comparison with invasive uncalibrated arterial pulse contour analysis as the reference method during TAVI. Bias and limits of agreement were quantified for cardiac index and mean arterial pressure using Bland-Altman analysis; blood pressure variables were evaluated for finger-cuff monitoring only, as Electrical Cardiometry does not provide a pressure signal.

The secondary aim was to evaluate the ability of each non-invasive modality to track haemodynamic trends during critical procedural phases, with particular attention to rapid pacing and valve deployment. Based on these findings, the clinical utility of non-invasive monitoring was assessed with regard to its potential role in minimalist TAVI concepts.

3. Material and Methods

3.1. Material

3.1.1. Invasive Pulse Contour Analysis (Acumen IQ Sensor/Hemosphere, BD)

Invasive arterial pressure monitoring was performed using the Acumen IQ sensor connected to the Hemosphere Advanced Monitor (both Edwards Lifesciences, Irvine, CA, USA, distributed by BD, Franklin Lakes, NJ, USA). The Acumen IQ sensor is the next-generation development of the FloTrac sensor and employs the same underlying pulse contour algorithm to derive stroke volume and cardiac output continuously from the peripheral arterial pressure waveform [70].

The algorithm calculates stroke volume according to the equation SV (mL/beat) = σAP × *x*, where σAP is the standard deviation of arterial pressure and *x* (Khi) is a multivariate conversion factor that converts the pressure signal into mL/beat. Arterial pressure-based cardiac output (APCO) is subsequently derived as APCO = PR × (σAP × *x*), where pulse rate (PR) is used rather than ECG-triggered heart rate, rendering the algorithm inherently robust to cardiac rhythm irregularities [70]. σAP is sampled continuously at 100 Hz over a rolling 20-second window, serving as a surrogate for pulse pressure proportional to stroke volume. Khi is a polynomial equation incorporating pulse rate, mean arterial pressure, large-vessel compliance estimated from patient demographics, and the skewness and kurtosis of the arterial waveform, updated on a rolling 60-second average. The algorithm requires no external calibration, as Khi continuously adjusts for changes in vascular tone [70].

3.1.2. Finger-Cuff Monitoring (Acumen IQ Cuff / Hemosphere, BD)

Continuous non-invasive finger-cuff monitoring was performed using the Acumen IQ cuff connected to the Hemosphere Advanced Monitor (both Edwards Lifesciences, Irvine, CA, USA, distributed by BD, Franklin Lakes, NJ, USA). The Acumen IQ cuff is the next-generation development of the ClearSight system and employs the same volume clamp measurement principle, as described in section 2.3.3 [71]. Stroke volume is derived according to the equation SV = SPI / Afterload, where SPI represents the systolic pressure-time integral, defined as the area under the systolic portion of the reconstructed brachial pressure waveform, and afterload is individualised by age, gender, height, and weight [71]. Cardiac output is calculated as the product of stroke volume and heart rate, updated on a beat-to-beat basis [71].

3.1.3. Electrical Cardiometry *(ICON, Osypka Medical)*

Non-invasive haemodynamic monitoring by Electrical Cardiometry was performed using the ICON monitor (Osypka Medical GmbH, Berlin, Germany). As described in section 2.3.1, the device measures changes in thoracic electrical bioimpedance via four surface electrodes to

derive stroke volume on a beat-to-beat basis. In the Electrical Cardiometry algorithm, stroke volume is calculated from the maximum rate of change in thoracic impedance during systole (dZ/dt max) and the ECG-derived ventricular ejection time [52 - 55]. Cardiac index is calculated as the product of stroke volume, heart rate, and body surface area. The device computes a Signal Quality Index (SQI) from the recorded impedance signal; values above 70 are considered adequate by the manufacturer [54]. The algorithm requires no external calibration.

3.2. Study Design and Patient Population

This was a prospective, single-centre observational study conducted at the Heart Centre of the University Hospital of Cologne, Germany. Ethical approval was obtained from the local ethics committee (No. 23-1310, 14 November 2023; Chairperson: Univ.-Prof. Dr. med. R. Voltz), and the study was registered in the German Clinical Trials Register (DRKS00038984). Written informed consent was obtained from all participants prior to inclusion. The study was conducted and reported in accordance with the STROBE statement for observational studies.

A total of 59 consecutive patients scheduled for elective TAVI were screened for eligibility and enrolled following written informed consent.

3.2.1. Inclusion and Exclusion Criteria

Patients were eligible for inclusion if they were aged 18 years or older, scheduled for elective TAVI for severe aortic stenosis, in preoperative predominant sinus rhythm, and able to provide written informed consent. Patients were excluded if the indication for TAVI was aortic regurgitation rather than stenosis, if acquisition of an adequate arterial pressure waveform throughout the procedure was not feasible, if they had a cardiac rhythm other than predominant sinus rhythm preoperatively, if they declined to provide informed consent, or if the planned procedure was cancelled or postponed.

3.3. Anaesthetic and Haemodynamic Monitoring Protocol

All procedures were performed in the hybrid operating theatre of the Heart Centre at the University Hospital of Cologne. Conscious sedation (CS) was the standard anaesthetic approach, using continuous infusions of Remifentanil (starting dose 0.03 - 0.05 µg/kg/min) and Propofol (starting dose 0.5 - 1 mg/kg/h), titrated to patient response, with supplemental oxygen administered via nasal cannula. General anaesthesia (GA) was reserved for cases in which periprocedural transoesophageal echocardiography was requested. Induction of GA was performed with a Remifentanil infusion (0.4 - 0.5 µg/kg/min) and a Propofol bolus (1-2 mg/kg), with maintenance using Remifentanil (0.1 - 0.2 µg/kg/min) and Sevoflurane (MAC 0.7

- 0.9). The airway was secured via endotracheal intubation. All anaesthetic management was provided by a senior anaesthesiology resident and a consultant specialised in cardiothoracic anaesthesia.

Haemodynamic support was provided primarily with Norepinephrine (starting dose 0.03 - 0.05 µg/kg/min), targeting a minimum mean arterial pressure of 65 mmHg. Push-dose Norepinephrine (10 - 100 µg) was administered in cases of severe haemodynamic instability where physiologically appropriate. All patients received a baseline infusion of balanced crystalloid solution, with the total volume administered according to clinical judgement and haemodynamic response.

Standard monitoring comprised continuous five-lead ECG, pulse oximetry, non-invasive blood pressure measurement, and capnometry. Prior to CS or GA, arterial access was established via the left radial artery, or, if not feasible, the brachial artery.

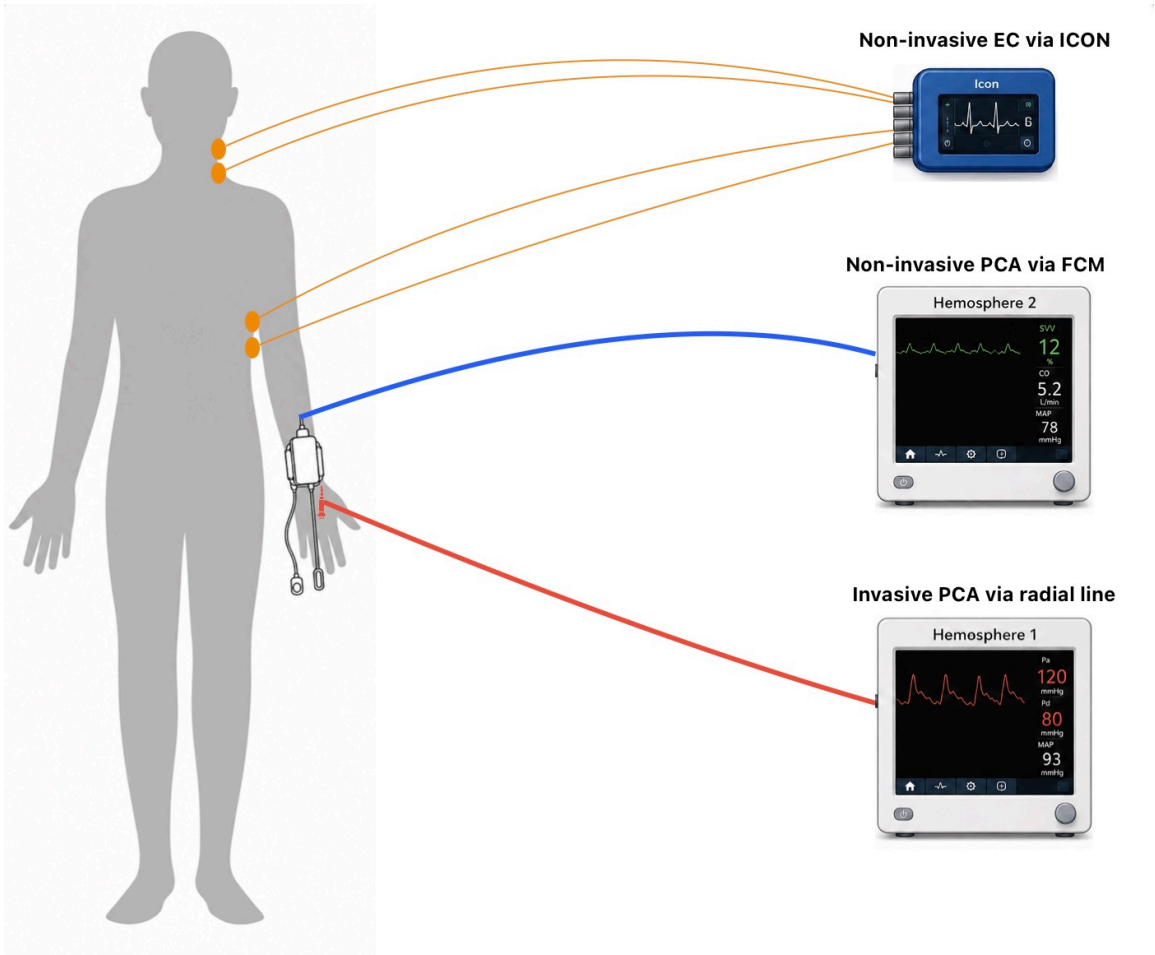


***Figure 3: Schematic illustration of the simultaneous periprocedural monitoring setup**. Invasive pulse contour analysis was performed via an arterial cannula placed in the left radial artery, connected to the Acumen HemoSphere monitor (Hemosphere 1; Edwards Lifesciences). Finger-cuff monitoring was conducted using the Acumen IQ Cuff applied to the second or third finger of the left hand, connected to a second Acumen HemoSphere monitor (Hemosphere 2; Edwards Lifesciences). Electrical Cardiometry was measured via four electrodes positioned at the left side of the neck and left thorax according to the manufacturer's specifications, connected to the ICON monitor (Osypka Medical). All three modalities acquired data simultaneously and continuously throughout the procedure. Device depictions are representative only. EC, Electrical Cardiometry; FCM, finger-cuff monitoring; PCA, pulse contour analysis.*

Puncture was performed either by palpation or under ultrasound guidance. Catheters used were a 20G × 80 mm Vygon Leadercath or a 20G × 45 mm BD Flowswitch. Two peripheral venous catheters were inserted in all patients.

After theatre start, the Acumen IQ sensor was connected to the arterial line and PCA monitoring was initiated via the Hemosphere Advanced Monitor. The Acumen IQ cuff was applied to the second or third finger of the left hand and connected to a second Hemosphere Advanced Monitor. For the ICON monitor, two electrodes were applied to the left side of the neck and two electrodes to the lower left thorax according to the manufacturer's specifications and secured with adhesive tape. All three monitoring systems were applied and initiated by two trained members of the study group. Continuous recordings were time-stamped by each device independently. Temporal alignment across modalities was performed during data processing, as described in section 3.4. The study setup is shown in Figure 3.

3.4. Data Collection and Management

Pre- and intraoperative clinical data, including patient demographics, comorbidities, preoperative diagnostic findings, and procedural information were extracted from routine hospital records and manually entered into Microsoft Excel (version 16.100; Microsoft, Redmond, WA, USA).

Postoperative complications and major adverse cardiac and cerebrovascular events (MACCE) were likewise identified from routine hospital records and classified according to the EPCO definitions, as described in section 2.4 [68].

Haemodynamic variables obtained from PCA, FCM, and EC were recorded continuously throughout the entire procedure and exported for subsequent offline analysis, yielding high-resolution longitudinal beat-to-beat data. Continuous recordings were time-stamped independently by each device; temporal alignment across modalities was performed during offline data processing using device-specific export timestamps.

Signal quality was ensured through routine system calibration and continuous verification by the attending anaesthesiologist. Time periods affected by technical artefacts or impaired signal integrity - including dampened or manipulated arterial line waveforms, finger-cuff adjustment intervals, or insufficient EC signal quality as indicated by an SQI below 70 - were identified, flagged, and excluded prior to analysis.

3.5. Statistical Analysis

Continuous variables are reported as median [Q1; Q3] and categorical variables as n (%). The statistical analysis plan was developed prior to data access. Baseline characteristics and perioperative outcomes are described in aggregate for the whole cohort.

Agreement between PCA as the reference method and the two non-invasive modalities, FCM and EC, was evaluated at three complementary levels of analysis. First, static agreement on time-aligned beat-to-beat measurements was assessed by classical Bland-Altman analysis, with mean bias calculated as the mean of paired differences (alternative method - PCA) and 95% limits of agreement (LOA) defined as bias ± 1.96 × SD. To account for the repeated-measurements structure of the data, Bland-Altman limits of agreement were additionally computed using a variance-components (ANOVA) approach that partitions within-patient and between-patient variance [72]. For CI, the Critchley percentage error (PE) was calculated as 1.96 × SD divided by the mean of the two methods; a PE < 30% was considered indicative of clinical interchangeability [74]. For blood pressure variables (mean arterial pressure (MAP), systolic arterial blood pressure (SYS), diastolic arterial blood pressure (DIA)), agreement was additionally benchmarked against the ANSI/AAMI/ISO 81060-2 criterion (mean bias ≤ 5 mmHg and SD ≤ 8 mmHg), with stratified analyses performed across clinically relevant pressure ranges (MAP < 65, 65-95, ≥ 95 mmHg; SYS < 90, 90-130, ≥ 130 mmHg; DIA < 60, 60-80, ≥ 80 mmHg).

Second, trending ability was assessed using the Critchley four-quadrant concordance method, computed from consecutive within-patient delta-pairs after excluding pairs where both changes fell within a pre-specified exclusion zone (±0.3 $L \cdot min^{-1} \cdot m^{-2}$ for CI; ±5 mmHg for blood pressure). A concordance of ≥ 92% was considered clinically acceptable [75].

Third, the clinical significance of disagreement for intraoperative hypotension was evaluated at the patient level using three summary measures derived from each monitor: time-weighted average of MAP below 65 mmHg (TWA-MAP<65), percentage of procedural time spent below 65 mmHg, and cumulative area under the threshold (AUC deficit). Agreement between patient-level summary measures was quantified using Lin's concordance correlation coefficient (CCC), Bland–Altman limits of agreement, and Wilcoxon signed-rank tests.

Longitudinal CI trajectories were visualised as the median (line) and interquartile range (ribbon) across all cases, aligned to the aortic valve implantation (AVI) pacing marker (t = 0 s) within a ±1000 s window in 20 s bins; a ±30 s peri-implantation window was highlighted to visualise beat-to-beat changes during valve deployment. As a sensitivity analysis, agreement metrics were recomputed after excluding the peri-implantation period (±120 s around the pacing marker).

All analyses were performed using R (version 4.4; R Foundation for Statistical Computing, Vienna, Austria) with the packages tidyverse, gtsummary and flextable. A two-sided $p < 0.05$ was considered statistically significant; given the exploratory, method-comparison design, no adjustment for multiple testing was applied and secondary findings are regarded as hypothesis-generating.

4. Results

4.1. Study Population and Procedural Characteristics

Of 59 patients enrolled between January and March 2026, 10 were excluded prior to analysis. Two patients were found to have severe aortic regurgitation without relevant stenosis and were therefore outside the predefined inclusion criteria. One patient underwent vascular access via the transaxillary route, precluding reliable radial arterial pressure measurement and thus rendering the reference signal unusable for analysis. Five procedures were cancelled on the day of surgery due to theatre capacity constraints, and one was withdrawn by the responsible cardiologist prior to the procedure owing to an unacceptably high estimated procedural risk. One patient presented with absolute arrhythmia prior to induction, and was therefore excluded as the study protocol required predominant sinus rhythm. The final analysis population comprised 49 patients.

Baseline demographic and clinical characteristics are summarised in Table 2. The cohort had a median age of 84 years [IQR 78-87]. Twenty-six patients (53%) were female and 23 (47%) were male. Median height was 170 cm [162-175], median weight 74 kg [66 - 86], and median BMI 26.3 kg/m² [23.7 - 30.4]. ASA classification was III in 29 patients (59%) and IV in 20 patients (41%).

Echocardiographic assessment confirmed severe aortic stenosis in all 49 patients, with a median aortic valve area of 0.83 cm² [0.70 - 0.90] and a median peak aortic jet velocity of 4.10 m/s [3.65 - 4.43]. Two patients had a valvular stenosis graded II°, whilst the remaining 47 had III° stenosis. Clinically relevant concomitant aortic regurgitation was present in 26 patients (53%), mitral regurgitation in 19 (39%), and tricuspid regurgitation in 15 (31%). Left ventricular ejection fraction (LVEF) was documented as a numeric value in 42 patients, with a median of 58% [42 - 63]; the remaining seven patients had LVEF recorded as a non-numeric estimate ("preserved" or "mildly reduced”).

The comorbidity burden was high and consistent with the expected profile of an elderly TAVI population. Arterial hypertension was documented in 47 patients (96%), coronary artery disease in 31 (63%), and a history of arrhythmias in 21 patients (43%). Heart failure with reduced ejection fraction (HFrEF) was documented in 16 patients (33%) and heart failure with preserved ejection fraction (HFpEF) in five (10%). Chronic kidney disease or other nephropathy was recorded in 10 patients (20%), and diabetes mellitus in 14 patients (29%; 12 non-insulin-dependent, 2 insulin-dependent). Chronic pulmonary disease was present in six patients (COPD: 4 [8%], asthma: 2 [4%]). Pulmonary arterial hypertension was documented in four patients (8%). A permanent pacemaker or ICD was in situ in three patients (6%). Neurological comorbidities were documented in seven patients (14%).

Antihypertensive medication was recorded in 44 patients (90%), most commonly beta-blockers (32 patients, 65%), AT1-receptor antagonists (20, 41%), ACE inhibitors (17, 35%), and calcium-channel antagonists (15, 31%).

Table 2. Baseline patient and procedural characteristics (n = 49)

Variable	n = 49
Demographics	
Age [years], median [IQR]	84 [78; 87]
Female sex, n (%)	26 (53%)
Height [cm], median [IQR]	170 [162; 175]
Weight [kg], median [IQR]	74 [66; 86]
BMI [kg/m²], median [IQR]	26.3 [23.7; 30.4]
ASA classification, n (%)	
III	29 (59%)
IV	20 (41%)
Echocardiographic findings	
Aortic valve area [cm²], median [IQR]	0.83 [0.70; 0.90]
Peak aortic jet velocity [m/s], median [IQR]	4.10 [3.65; 4.43]
Aortic stenosis grade, n (%)	
II°	2 (4%)
III°	47 (96%)
LVEF [%], median [IQR] [1]	58 [42; 63]
Clinically relevant aortic regurgitation, n (%)	26 (53%)
Clinically relevant mitral regurgitation, n (%)	19 (39%)
Clinically relevant tricuspid regurgitation, n (%)	15 (31%)
Comorbidities	
Arterial hypertension, n (%)	47 (96%)
Coronary artery disease, n (%)	31 (63%)
History of arrhythmias, n (%)	21 (43%)
HFrEF, n (%)	16 (33%)
HFpEF, n (%)	5 (10%)
Chronic kidney disease / nephropathy, n (%)	10 (20%)
Diabetes mellitus, n (%)	14 (29%)
Non-insulin-dependent	12 (24%)
Insulin-dependent	2 (4%)
COPD, n (%)	4 (8%)
Asthma, n (%)	2 (4%)
Pulmonary arterial hypertension, n (%)	4 (8%)
Permanent pacemaker or ICD, n (%)	3 (6%)
Neurological comorbidities, n (%)	7 (14%)
Antihypertensive medication	
Any antihypertensive agent, n (%)	44 (90%)
Beta-blockers	32 (65%)
AT1-receptor antagonists	20 (41%)
ACE inhibitors	17 (35%)
Calcium-channel antagonists	15 (31%)
Procedural characteristics	
Vascular access, n (%)	
Transfemoral	49 (100%)
Anaesthetic technique, n (%)	
Conscious sedation	47 (96%)
General anaesthesia	2 (4%)
Intraoperative crystalloids [ml], median [IQR]	500 [500; 1000]
Colloid administration, n (%)	0 (0%)

[1] LVEF documented as numeric value in 42 of 49 patients; remaining 7 patients recorded as “preserved" or "mildly reduced".

Table 2: Baseline demographic, clinical, and procedural characteristics of the analysis population (n = 49). *Continuous variables are presented as median [interquartile range]; categorical variables as n (%). ASA, American Society of Anesthesiologists; BMI, body mass index; COPD, chronic obstructive pulmonary disease; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; IQR, interquartile range; LVEF, left ventricular ejection fraction.*

All 49 procedures were performed via transfemoral access. Two patients required conversion to general anaesthesia: one due to a pre-procedural indication for transoesophageal echocardiography, and one following intraoperative respiratory failure necessitating controlled ventilation. Intraoperative fluid administration consisted exclusively of crystalloids, with a median of 500 ml [500 - 1000]; no colloids were administered in any patient.

4.2. Cardiac Index

4.2.1. Longitudinal Trajectory Around Valve Implantation

Longitudinal CI trajectories aligned to the aortic valve implantation (AVI) pacing marker are shown in Figure 4. With FCM, median CI tracked closely with invasive PCA across the periprocedural window; both modalities captured a brief transient rise at the moment of valve deployment, followed by a return to baseline within 200 - 300 s. In contrast, electrical cardiometry consistently underestimated CI relative to PCA throughout the observation window, with a median offset of approximately 0.8 - 1.0 L·min^{-1}·m^{-2} and a with a barely discernible haemodynamic response at the moment of valve deployment.

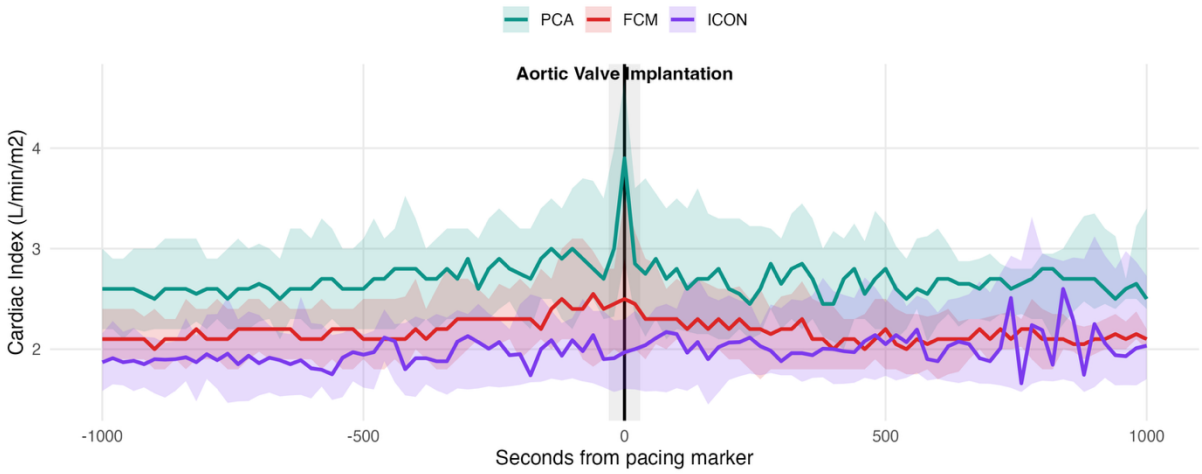
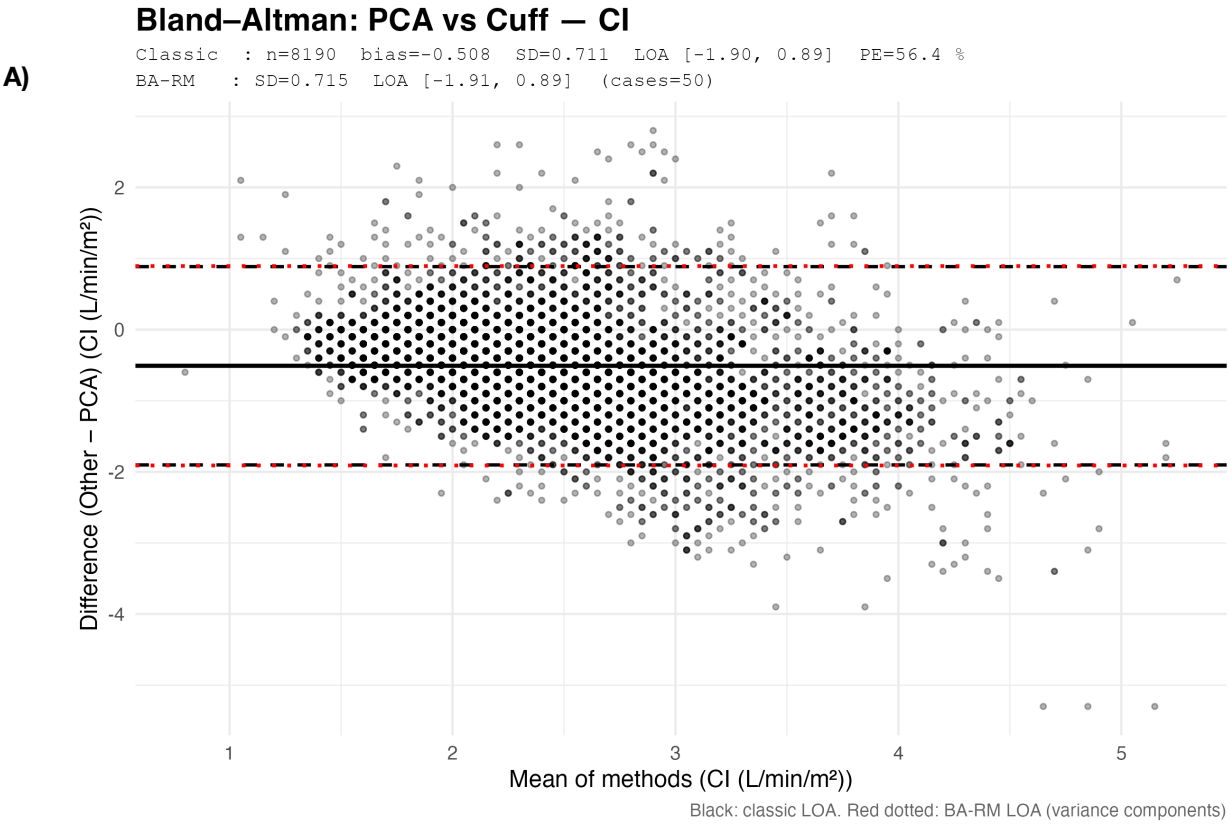


Figure 4: Longitudinal cardiac index (L·min^{-1}·m^{-2}) around the aortic valve implantation pacing marker. *Pulse contour analysis (PCA) versus finger-cuff monitoring (FCM) versus electrical cardiometry (ICON). Lines: cross-case median; ribbons: interquartile range. AVI window ±30 s; bin width 20 s; plot window ±1000 s.*

4.2.2. Static Agreement

Bland-Altman analyses for cardiac index are shown in Figure 5. For PCA versus FCM (n = 8190 paired measurements), the mean bias was –0.51 L·min^{-1}·m^{-2} (95% LOA –1.90 to +0.89 L·min^{-1}·m^{-2}), with a percentage error of 56.4%. For PCA versus EC (n = 3819 paired measurements), the mean bias was –0.75 L·min^{-1}·m^{-2} (95% LOA –2.70 to +1.20 L·min^{-1}·m^{-2}), with a percentage error of 80.2%. Both non-invasive modalities thus systematically

A)



B)

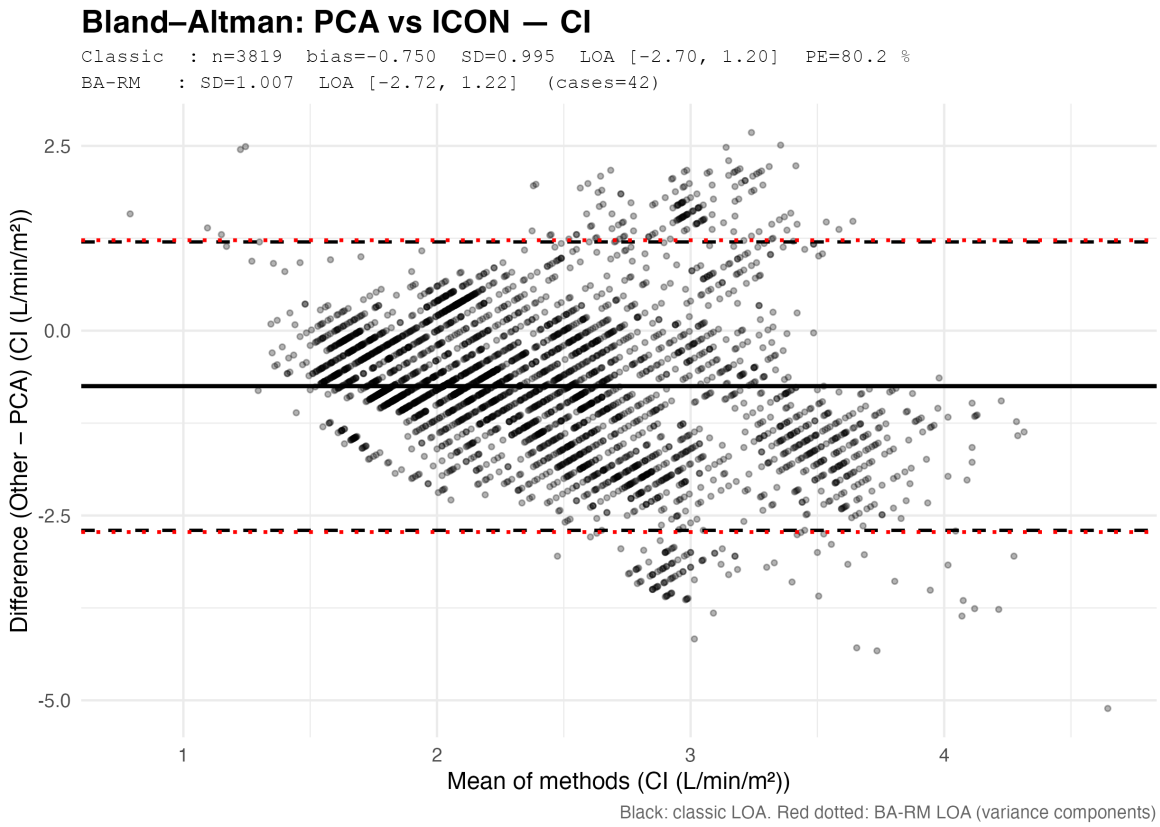


Figure 5: Bland–Altman plots for cardiac index. *(A) PCA versus finger-cuff monitoring (n = 8190). (B) PCA versus electrical cardiometry (n = 3819). Solid line: mean bias; dashed black lines: classical 95 % limits of agreement (LOA); dotted red lines: variance-components LOA accounting for repeated measurements. The striations visible in panel B reflect the 0.1 L·min⁻¹·m⁻² output resolution of the ICON device.*

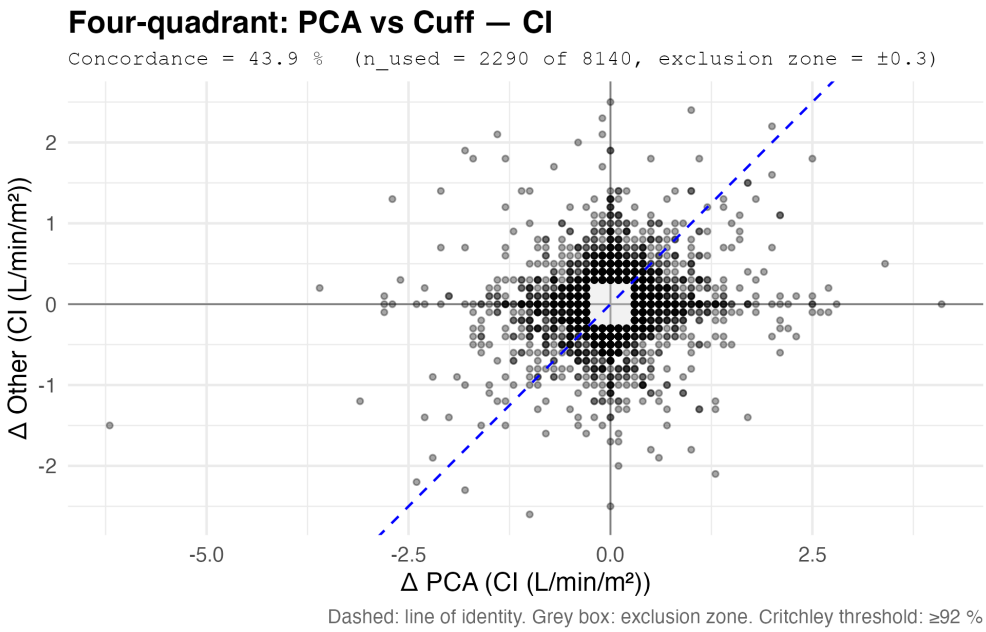
underestimated CI relative to PCA, and neither met the Critchley-Critchley threshold of PE < 30% required for clinical interchangeability.

Variance-component limits of agreement, computed using a repeated-measures Bland-Altman approach (BA-RM), were virtually identical to the classical LOA for both comparisons (FCM: SD_RM = 0.72 vs SD = 0.71; EC: SD_RM = 1.01 vs SD = 0.99), indicating that disagreement was driven predominantly by within-patient beat-to-beat variability rather than by systematic between-patient differences in device calibration. For both comparisons, bias widened at higher mean CI values, suggesting proportional rather than purely additive disagreement.

4.2.3. Trending Ability

Four-quadrant analyses of beat-to-beat CI changes are shown in Figure 6. Applying an exclusion zone of ±0.3 $L \cdot min^{-1} \cdot m^{-2}$, concordance was 43.9% for PCA versus FCM (n = 2290 delta-pairs) and 43.0% for PCA versus EC (n = 542 delta-pairs). Both values were below the 50% level expected from chance concordance alone, indicating that neither non-invasive modality reliably reflected the direction of PCA-derived CI changes at the beat-to-beat level.

A)



B)

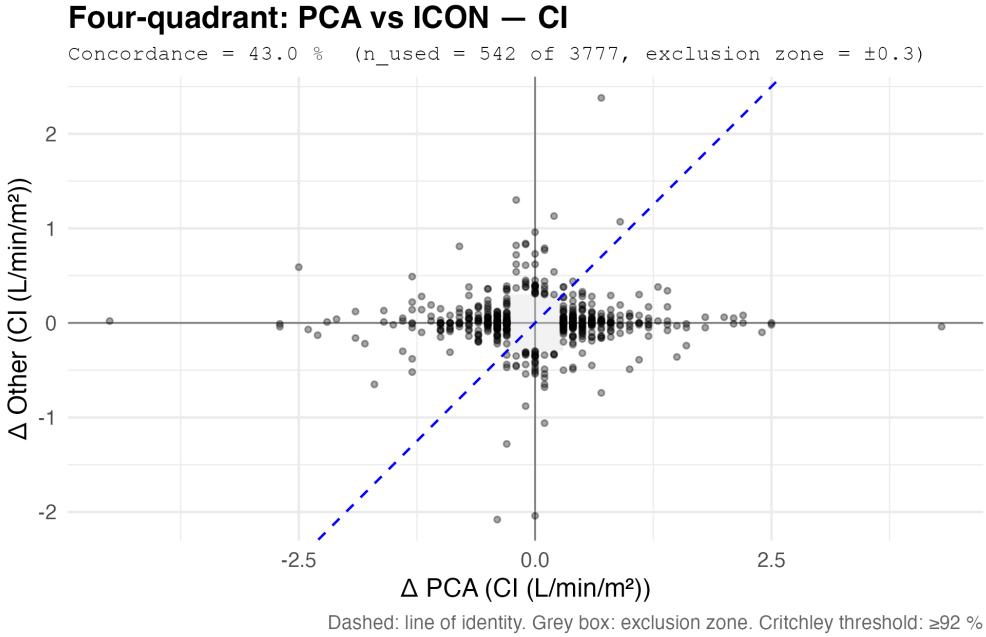


Figure 6: Four-quadrant plots for cardiac index trending. *(A) PCA versus finger-cuff monitoring. (B) PCA versus electrical cardiometry. Each point represents a pair of consecutive within-patient changes (Δ PCA, Δ other method). The grey central box indicates the exclusion zone (±0.3 L·min⁻¹·m⁻²); dashed blue line: line of identity. Critchley acceptability threshold for concordance: ≥ 92 %.*

4.3. Blood Pressure

4.3.1. Beat-to-beat Static Agreement

Agreement between finger-cuff monitoring and invasive pulse contour analysis was evaluated according to ANSI/AAMI/ISO 81060-2 criteria, stratified by clinically relevant pressure ranges (Figure 7). Across the entire cohort, FCM failed the AAMI criterion for all three pressure variables: MAP (bias −2.0 mmHg, SD 13.4 mmHg), SYS (bias −12.0 mmHg, SD 22.2 mmHg), and DIA (bias +3.7 mmHg, SD 12.6 mmHg). In no pressure stratum was the combined criterion (|bias| ≤ 5 mmHg and SD ≤ 8 mmHg) met.

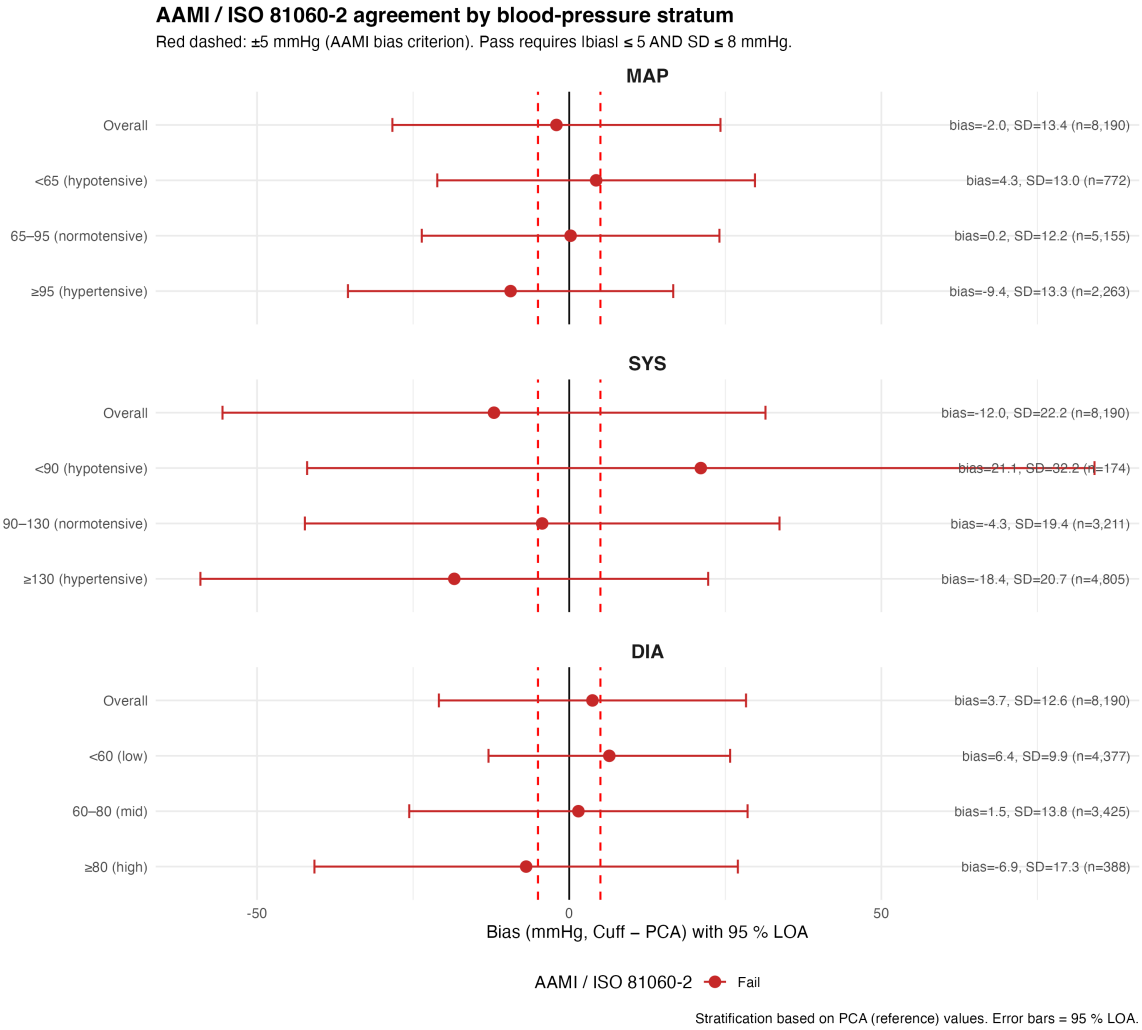


Figure 7: Agreement of finger-cuff blood-pressure measurements with invasive pulse contour analysis according to ANSI/AAMI/ISO 81060-2 criteria, stratified by clinically relevant pressure ranges. *Points: mean bias (Cuff − PCA); horizontal bars: 95 % limits of agreement. Red dashed vertical lines: AAMI bias criterion (±5 mmHg). Combined AAMI pass requires |bias| ≤ 5 mmHg and SD of differences ≤ 8 mmHg; no stratum met both criteria (all shown in red = Fail).*

Of particular clinical relevance, the MAP bias was directionally inverted across the physiological range: FCM overestimated MAP in the hypotensive stratum (bias +4.3 mmHg for MAP < 65 mmHg, n = 772) and underestimated it in the hypertensive stratum (bias −9.4 mmHg for MAP ≥ 95 mmHg, n = 2263). For systolic blood pressure, the pattern was even more pronounced, with a bias of +21.1 mmHg in the hypotensive stratum and −18.4 mmHg in the hypertensive stratum.

4.3.2. Trending Ability MAP

Four-quadrant analysis of beat-to-beat MAP changes is shown in Figure 8. Applying an exclusion zone of ±5 mmHg, concordance between FCM and PCA was 53.6% (n = 4281 delta-pairs), far below the Critchley threshold of ≥92%. Symmetric dispersion around the line of identity indicated that FCM failed to reliably detect the direction of invasive MAP changes on a beat-to-beat basis.

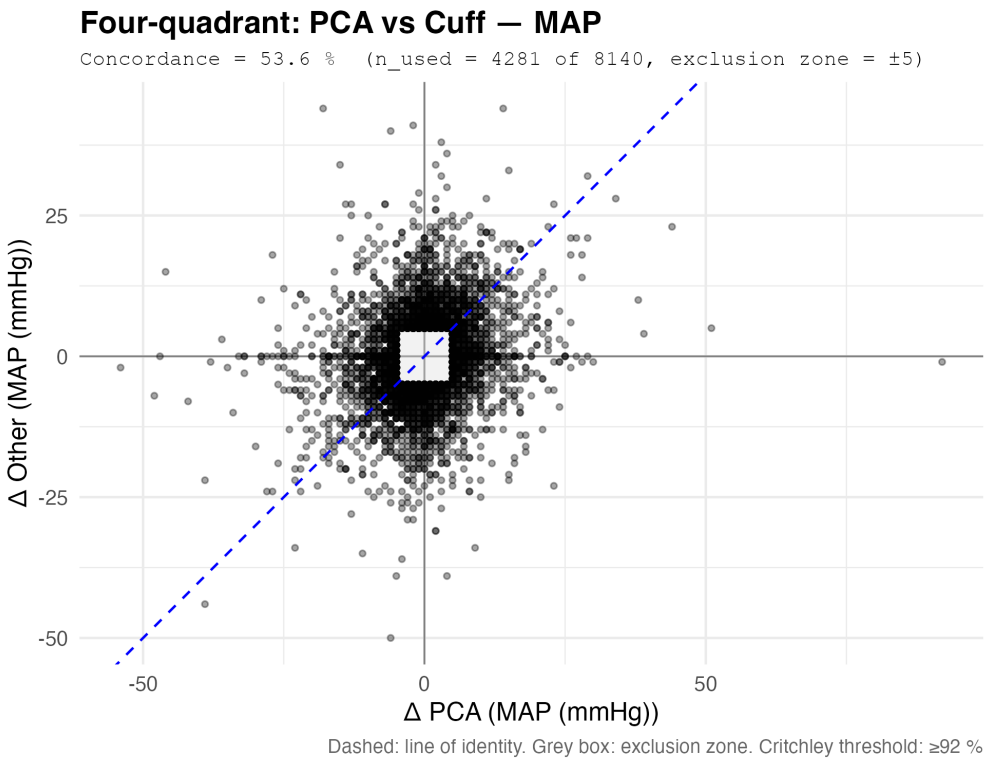


Figure 8: Four-quadrant plot for mean arterial pressure trending, PCA versus finger-cuff monitoring (n = 4281 delta-pairs). *Grey central box: exclusion zone ±5 mmHg; dashed blue line: line of identity. Critchley acceptability threshold: ≥ 92 %.*

4.3.3. Hypotension Exposure

To assess the clinical relevance of disagreement for intraoperative hypotension management, three patient-level summary measures of MAP below 65 mmHg were compared between FCM and PCA in the 22 paired cases with complete time-stamped MAP recordings. At the cohort level, no statistically significant differences were detected (all Wilcoxon signed-rank $p > 0.14$): median TWA-MAP<65 was 0.17 mmHg (FCM) versus 0.20 mmHg (PCA); median percentage of procedural time with MAP < 65 mmHg was 5.7% (FCM) versus 2.8% (PCA); and median AUC deficit was 9.5 mmHg·min (FCM) versus 13.1 mmHg·min (PCA).

However, patient-level agreement remained limited: Lin's concordance correlation coefficient (CCC) ranged from 0.38 (AUC deficit) to 0.58 (mean MAP), and the 95% limits of agreement for the percentage of time below 65 mmHg spanned −17 to +24 percentage points. In individual patients, FCM-derived summary measures could therefore diverge from PCA-derived values by clinically meaningful margins, even when cohort medians were similar.

4.4. Postoperative Outcomes

No in-hospital mortality occurred (0/49). As per institutional protocol, all patients were admitted to the ICU or PACU postoperatively, with a median stay of 1.0 days [1.0; 2.0]. No patient required postoperative invasive ventilation. Median length of hospital stay was 4.0 days [3.0; 5.0].

Any complication was recorded in 18 patients (36.7%), and postoperative organ dysfunction in 13 (26.5%). The MACCE composite occurred in 13 patients (26.5%). Individual postoperative events included AV block III° in 11 patients (22.4%), new permanent pacemaker implantation in 9 (18.4%), paravalvular leak in 5 (10.2%), all of which were of trace or mild degree without haemodynamic relevance, access-site complications in 4 (8.2%), pneumonia in 2 (4.1%), acute ischaemic stroke in 1 (2.0%), acute kidney injury in 1 (2.0%), new-onset atrial fibrillation in 1 (2.0%), delirium in 1 (2.0%), and ventricular tachycardia in 1 (2.0%). No cases of STEMI, NSTEMI, cardiogenic shock, cardiac arrest, ventricular fibrillation, ARDS, pulmonary embolism, liver failure, or wound infection were observed. Full outcome data are provided in Table 3.

Table 3. Postoperative outcomes (n = 49)

Group	Outcome	Overall cohort (N = 49) [1]
Mortality and resource use	In-hospital mortality	0 (0.0%)
	ICU/PACU admission	49 (100.0%)
	Length of ICU stay [days], median [IQR]	1.0 [1.0; 2.0], range 1-10
	Postoperative mechanical ventilation	0 (0.0%)
	Length of hospital stay [days], median [IQR]	4.0 [3.0; 5.0], range 2-14
eCRF composite endpoints	Any complication (eCRF composite)	18 (36.7%)
	Postoperative organ dysfunction (eCRF composite)	13 (26.5%)
	MACCE (eCRF composite)	13 (26.5%)
Individual events	Paravalvular leak [2]	5 (10.2%)
	STEMI	0 (0.0%)
	NSTEMI	0 (0.0%)
	Cardiogenic shock	0 (0.0%)
	Cardiac arrest	0 (0.0%)
	AV block III°	11 (22.4%)
	New-onset atrial fibrillation	1 (2.0%)
	Ventricular tachycardia	1 (2.0%)
	Ventricular fibrillation	0 (0.0%)
	Bradyarrhythmia absoluta	1 (2.0%)
	New postoperative pacemaker	9 (18.4%)
	Acute ischaemic stroke	1 (2.0%)
	Delirium	1 (2.0%)
	ARDS	0 (0.0%)
	Pneumonia	2 (4.1%)
	Pulmonary embolism	0 (0.0%)
	Acute kidney injury	1 (2.0%)
	Liver failure	0 (0.0%)
	Thromboembolism	0 (0.0%)
	Access-site complications	4 (8.2%)
	Haematoma / minor bleeding	2 (4.1%)
	Aneurysma spurium	2 (4.1%)
	Conservative management	1 (2.0%)
	Surgical repair	1 (2.0%)
	Wound infection	0 (0.0%)
	Infection (any)	1 (2.0%)

[1] n (%); Median [Q1; Q3].

[2] All paravalvular leaks were of trace or mild degree; none required reintervention.

Table 3: Postoperative outcomes of the analysis population (n = 49). *Categorical variables are presented as n (%); continuous variables as median [IQR]. ARDS, acute respiratory distress syndrome; eCRF, electronic case report form; ICU, intensive care unit; IQR, interquartile range; MACCE, major adverse cardiac and cerebrovascular events; NSTEMI, non-ST-elevation myocardial infarction; PACU, post-anaesthesia care unit; POD, postoperative day; STEMI, ST-elevation myocardial infarction.*

5. Discussion

The aim of this study was to provide insight into whether non-invasive haemodynamic monitoring might be an adequate substitute to the established invasive uncalibrated pulse contour analysis during TAVI procedures. For this, 49 consecutive patients undergoing TAVI were enrolled. Haemodynamic parameters were simultaneously collected using the ICON Monitor (EC), the Acumen IQ Cuff (FCM) and the Acumen IQ Sensor via radial cannulation (PCA). Neither non-invasive modality could provide sufficient concordance with the invasive reference. For both cardiac index and blood pressure measurements, accuracy as well as trending ability did not meet the predefined criteria for interchangeability.

In summary, the data do not support the use of either modality as a standalone substitute for invasive haemodynamic monitoring in the TAVI setting.

5.1. Accuracy and Precision of Non-Invasive Monitoring Under TAVI Conditions

Measurements for Cardiac Index was provided by both non-invasive modalities. The percentage error was 56.4% with FCM and 80.2% with EC. Both systems therefore exceed the 30% threshold for clinical interchangeability proposed by Critchley and Critchley [74]. In addition, variance-components Bland-Altman decomposition showed that most of the disagreement originated from within-patient beat-to-beat variability rather than from between-patient calibration errors. Inaccuracy therefore appears to be a dynamic, signal-level phenomenon rather than a structural one. This is consistent with the haemodynamic volatility typical during TAVI. The signal delays after rapid pacing sequences documented by Rittershofer et al. [41] add a further practical constraint during the most demanding procedural phases.

EC performed worse overall. FCM showed a basic concordance with the reference in the longitudinal trajectory around valve deployment. Both capturing a brief rise in CI before returning to baseline. EC, on the other hand, showed virtually no reaction to valve deployment with a persistent offset against PCA throughout the entire observation window. The bioimpedance-derived signal of the EC therefore appears to be especially insensitive to the acute afterload reduction that valve deployment produces. This may be due to the underlying principle of EC. During the phase of valve positioning and especially deployment, the delivery catheter hinders blood flow in the LVOT and aorta, possibly altering the bioimpedance signal and introducing further turbulences. This seems plausible, since severe aortic stenosis itself has been identified as a source of measurement error for electrical velocimetry-based output estimation [65]. The pre-existing inaccuracy under stenotic conditions may therefore further impair the ability of EC to detect the acute afterload reduction following valve deployment.

Blood pressure measurements were available only for FCM and PCA, but were equally unfavourable. FCM did not meet ANSI/AAMI/ISO 81060-2 criteria for MAP, systolic, or diastolic pressure. The reversal of the direction of the MAP bias is particularly interesting. The device overestimated in the hypotensive range and underestimated in the hypertensive range, a pattern that was even more prominent for systolic values. This reversal has not been reported in similar TAVI studies, probably because earlier studies did not break down agreement by pressure stratum. It may reflect the limitations of the Physiocal recalibration algorithm during rapid changes in arterial compliance and central haemodynamics that occur during TAVI procedures. Fluctuating dosage of vasopressors may affect FCM in particular, given the signal is acquired from the peripheral finger arteries especially affected by vasopressors. The algorithm used to reconstruct the brachial pressure waveform may additionally introduce error in this setting.

These findings are in line with those of similar previous studies. Stolte et al. reported percentage errors of 38 - 55% for blood pressure variables in a comparable TAVI cohort using the same finger-cuff device [42]. Rittershofer et al. similarly demonstrated limited agreement between non-invasive finger-cuff monitoring and invasive arterial pressure measurement during transfemoral TAVI [41]. Yahagi and Sasaki on the other hand reported acceptable agreement between finger-cuff and invasive arterial pressure in 30 TAVI patients under general anaesthesia [73]. However, their measurements were conducted during a stable 30-minute window following valve deployment, excluding the highly dynamic phases of rapid pacing, balloon valvuloplasty, and acute afterload change during valve deployment. Whereas the present dataset fully captured all disturbances during valve positioning. This methodological difference could explain the divergent results. An acceptable level of agreement under stable conditions does not necessarily reflect reliability during the critical phase of the procedure, in which haemodynamic monitoring is most needed.

5.2. Trending Ability Non-Invasive Monitoring Under TAVI Conditions

Four-quadrant concordance rates for CI were 43.9% for finger-cuff monitoring and 43.0% for EC, both falling below the 50% level expected from chance concordance alone and far below the 92% threshold proposed by Critchley et al. for clinical acceptability [75]. MAP concordance for FCM reached 53.6%, marginally above the level expected for chance concordance alone, but still below the acceptability threshold. Taken together, the ability of the non-invasive modalities to identify trends under these conditions seems of no practical use. The disagreement between non-invasive and invasive measurements is essentially random on a beat-to-beat basis, which fits with the within-patient variability pattern seen in the Bland-Altman analysis.

Several procedural factors may explain this random variability. Beat-to-beat fluctuations in stroke volume and ejection timing due to wire manipulation and rapid pacing may not be consistently captured by either modality. The highly dynamic haemodynamic environment of

TAVI, with abrupt phases of pacing-induced hypotension, valve deployment, and acute afterload reduction, generates signals fundamentally different from the stable conditions under which both devices were originally validated. Under these circumstances, the non-invasive signal is likely comprised of true haemodynamic change, possible motion artefacts due to conscious sedation, and device-specific processing latency.

The fact that FCM and EC performed almost identically despite their differences in underlying principle and signal processing, however, suggests that this might not primarily be a problem of the devices itself. The procedural environment itself appears to be the dominant limiting factor.

5.3. Limitations

Several limitations need to be addressed. Firstly, the reference method itself is a possible limitation. While invasive uncalibrated pulse contour analysis is widely used as the procedural standard during TAVI, it is not an absolute gold standard. The fact that it derives cardiac output from arterial waveform morphology without external calibration, makes it in principle also susceptible to disturbances such as rapid pacing, arrhythmias, and acute afterload changes. Even thermodilution, the gold standard most commonly used in validation studies of cardiac output, can exhibit a measurement variability of around 20% even under stable conditions [65, 66], and its limitations as a reference standard in the context of bioimpedance validation have been explicitly addressed [67]. Whilst PCA addresses some of these limitations, it is nevertheless susceptible to waveform distortion under haemodynamically unstable conditions [36]. The discrepancy observed could therefore at least in part reflect inaccuracy in the reference signal, rather than solely a failure of the non-invasive modalities.

Secondly, several study design characteristics limit the generalisability of the findings. As a single centre study, external validity may be restricted. The analysis population of 49 patients provided adequate power for the primary analyses but was underpowered for the planned subgroup analyses. For example, the low-flow/low-gradient subgroup included only three patients. All procedures were performed via transfemoral access and findings may not be directly applicable to other access routes.

While conscious sedation was the preferred and planed anaesthetic management, two patients required conversion to general anaesthesia. Additionally, depth of sedation was adjusted to patient need, resulting in a certain degree of heterogeneity in anaesthetic management. Since this also influences vasopressor requirements and therefore microcirculation and haemodynamic conditions, it may have a possible impact on device performance.

Finally, all devices were applied and monitored by the same trained study team, which ensured handling consistency across cases but did not enable blinded data acquisition. Any

systematic error introduced by device application or management would however be consistent across all cases.

5.4. Clinical Implications and Future Directions

The findings have direct implications for clinical practice. The data do not support using EC or FCM as standalone substitutes for invasive haemodynamic monitoring during TAVI. Clinical decisions made solely using either device would risk being based on unreliable data, particularly during the most haemodynamically demanding phases of the procedure.

The hypotension exposure analysis shows another important consideration. While cohort-level summaries of MAP below 65 mmHg were broadly comparable between finger-cuff monitoring and invasive PCA, patient-level agreement remained limited. This distinction is relevant, as adequate performance at the cohort level does not guarantee reliable measurement in the individual patient. Especially in the context of TAVI, where significant hypotension occurs regularly and abruptly, devices that underestimate hypotension or provide data with significant delay are not well suited to inform clinical decision-making.

However, these results should be interpreted in context of a medical field that continues to evolve rapidly. Ongoing advances in valve systems and delivery techniques have significantly reduced the procedural complexity, rapid pacing duration, and the extent of haemodynamic impairment associated with valve delivery. As the impact of the procedural conditions on haemodynamics becomes less extreme, the performance of non-invasive monitoring may improve correspondingly.

Parallel advances in device algorithms and signal processing are likely to reduce the performance gap between non-invasive and invasive monitoring. The inaccuracies shown here reflect the current generation of signal processing in a very specific context and do not necessarily represent the absolute limits of what can be achieved with non-invasive monitoring. Future models, potentially incorporating machine learning based waveform analysis or better adaptive recalibration strategies, may offer greater robustness under the dynamic conditions inherent to structural heart interventions.

Both TAVI and device development are pushing toward even more minimalist practice. Prospectively, non-invasive monitoring could support invasive monitoring in selected patients or serve as a primary option for low-complexity procedures.

The present study provides a reference point against which that progress can be assessed.

6. Conclusion

In this prospective single-centre study neither Electrical Cardiometry nor finger-cuff monitoring achieved clinically acceptable agreement with invasive uncalibrated pulse contour analysis during transfemoral TAVI. Cardiac index measurements did not meet the interchangeability threshold for both devices. Blood pressure measurements did not meet ANSI/AAMI/ISO 81060-2 standards. Four-quadrant concordance rates for both cardiac index and mean arterial pressure were around the level expected for chance concordance alone. Cohort hypotension exposure was comparable, but individual agreement was insufficient for clinical use. In their current form, neither device is suitable as a standalone replacement for invasive haemodynamic monitoring during TAVI.

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8. Appendix

8.1. List of Figures

Figure 1: Overview of diagnostic and therapeutic decision-making for aortic stenosis. Modified from: Praz F et al. 2025 ESC/EACTS Guidelines for the management of valvular heart disease [2]. AS = aortic stenosis; AU = Agatston units; AVA = aortic valve area; AVAi = AVA indexed to body surface area; AVCS = aortic valve calcium score; BP = blood pressure; BNP = B-type natriuretic peptide; DSE = dobutamine stress echocardiography; HF = heart failure; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-BNP; PG = pressure gradient; SAVR = surgical aortic valve replacement; SVi = stroke volume index; TAVI = transcatheter aortic valve implantation; Vmax = peak transvalvular velocity.

Figure 2: Schematic illustration of haemodynamic changes across the aortic valve before and after TAVI. Original schematic; pressure waveforms adapted from Graziani et al. [35]. (A) Severe aortic stenosis: turbulent, high-velocity jet (V_{max} ≥ 4 m/s) through the calcified, stenotic valve with markedly elevated left ventricular (LV) systolic pressure and a large pressure gradient (ΔP) between LV and aorta (Ao). (B) After transcatheter aortic valve implantation (TAVI): laminar flow through the prosthetic valve (V_{max} < 1.5 m/s), normalisation of LV peak pressure, and near-equalisation of the transvalvular pressure gradient. Blue, LV pressure; green, aortic pressure.

Figure 3: Schematic illustration of the simultaneous periprocedural monitoring setup. Invasive pulse contour analysis was performed via an arterial cannula placed in the left radial artery, connected to the Acumen HemoSphere monitor (Hemosphere 1; Edwards Lifesciences). Finger-cuff monitoring was conducted using the Acumen IQ Cuff applied to the second or third finger of the left hand, connected to a second Acumen HemoSphere monitor (Hemosphere 2; Edwards Lifesciences). Electrical Cardiometry was measured via four electrodes positioned at the left side of the neck and left thorax according to the manufacturer's specifications, connected to the ICON monitor (Osypka Medical). All three modalities acquired data simultaneously and continuously throughout the procedure. Device depictions are representative only. EC, Electrical Cardiometry; FCM, finger-cuff monitoring; PCA, pulse contour analysis.

Figure 4: Longitudinal cardiac index ($L \cdot min^{-1} \cdot m^{-2}$) around the aortic valve implantation pacing marker. Pulse contour analysis (PCA) versus finger-cuff monitoring (FCM) versus electrical cardiometry (ICON). Lines: cross-case median; ribbons: interquartile range. AVI window ±30 s; bin width 20 s; plot window ±1000 s.

Figure 5: Bland–Altman plots for cardiac index. (A) PCA versus finger-cuff monitoring (n = 8190). (B) PCA versus electrical cardiometry (n = 3819). Solid line: mean bias; dashed black lines: classical 95 % limits of agreement (LOA); dotted red lines: variance-components LOA accounting for repeated measurements. The striations visible in panel B reflect the 0.1 $L \cdot min^{-1} \cdot m^{-2}$ output resolution of the ICON device.

Figure 6: Four-quadrant plots for cardiac index trending. (A) PCA versus finger-cuff monitoring. (B) PCA versus electrical cardiometry. Each point represents a pair of consecutive within-patient changes (Δ PCA, Δ other method). The grey central box indicates the exclusion zone (±0.3 $L \cdot min^{-1} \cdot m^{-2}$); dashed blue line: line of identity. Critchley acceptability threshold for concordance: ≥ 92 %.

Figure 7: Agreement of finger-cuff blood-pressure measurements with invasive pulse contour analysis according to ANSI/AAMI/ISO 81060-2 criteria, stratified by clinically relevant pressure ranges. Points: mean bias (Cuff − PCA); horizontal bars: 95 % limits of agreement. Red dashed vertical lines: AAMI bias criterion (±5 mmHg). Combined AAMI pass requires | bias| ≤ 5 mmHg and SD of differences ≤ 8 mmHg; no stratum met both criteria (all shown in red = Fail).

Figure 8: Four-quadrant plot for mean arterial pressure trending, PCA versus finger-cuff monitoring (n = 4281 delta-pairs). Grey central box: exclusion zone ±5 mmHg; dashed blue line: line of identity. Critchley acceptability threshold: ≥ 92 %.

8.2. List of Tables

Table 1: Discordant subtypes of AS according to the 2025 ESC/EACTS Guidelines for the management of valvular heart disease [2]. LF/LG. low-flow/low-gradient; NF/LG, normal-flow/ low-gradient; AS, aortic stenosis; PGmean, mean pressure gradient; AVA, aortic valve area; SVi, stroke volume index; LVEF, left ventricular ejection fraction.

Table 2: Baseline demographic, clinical, and procedural characteristics of the analysis population (n = 49). Continuous variables are presented as median [interquartile range]; categorical variables as n (%). ASA, American Society of Anesthesiologists; BMI, body mass index; COPD, chronic obstructive pulmonary disease; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; IQR, interquartile range; LVEF, left ventricular ejection fraction.

Table 3: Postoperative outcomes of the analysis population (n = 49). Categorical variables are presented as n (%); continuous variables as median [IQR]. ARDS, acute respiratory distress syndrome; eCRF, electronic case report form; ICU, intensive care unit; IQR, interquartile range; MACCE, major adverse cardiac and cerebrovascular events; NSTEMI, non-ST-elevation myocardial infarction; PACU, post-anaesthesia care unit; POD, postoperative day; STEMI, ST-elevation myocardial infarction.

8.3. Documentation of AI Use

Area of Application	AI Tool Used	Purpose	Validation
Literature research and review	Claude (Sonnet 4.6)	Content screening and extraction	Sources and content manually verified by the author
Language optimisation and phrasing	Claude (Sonnet 4.6)	Improvement of readability and grammar	Final correction by the author
Translation assistance	DeepL	German-English translations	Final correction by the author
Figures and tables	Claude (Sonnet 4.6), ChatGPT-5.5	Visual enhancement and structuring	Content reviewed and modified by the author