Progress in Pediatric Cardiology 81 (2026) 101908

Contents lists available at ScienceDirect

Progress in Pediatric Cardiology

journal homepage: www.elsevier.com/locate/ppedcard

Flow acceleration and dynamic left ventricular outflow tract obstruction in neonatal warm shock: Echocardiographic phenotyping and management implications

Benjamim Ficial [a,b,*], Beatrice Allena [a], Ilaria Boffa [a], Arianna Zuccato [a], Daniele Mosolo [a], Martina Ciarcià [a], Federica Runfola [a], Silvia Nogara [a], Iuri Corsini [c], Willem P. de Boode [b]

[a] Neonatal Intensive Care Unit, University and Hospital Trust of Verona, P.le Stefani 1, 37126, Verona, Italy
[b] Radboud University Medical Center, Amalia Children's Hospital, Division of Neonatology, Department of Pediatrics, Geert Grooteplein zuid 10, 6525 GA, Nijmegen, the Netherlands
[c] Division of Neonatology, Careggi University Hospital of Florence, Largo Brambilla 3, 50134, Florence, Italy

ARTICLE INFO

Keywords:
Neonatology
Sepsis
Echocardiography
Neonatologist Performed Echocardiography
Shock

ABSTRACT

Echocardiography is increasingly used to assess hemodynamics in critically ill neonates. We describe a novel echocardiographic phenotype associated with warm shock in preterm neonates with late-onset sepsis, ranging from left ventricular outflow tract (LVOT) acceleration to dynamic LVOT obstruction, with systolic anterior motion (SAM) of the mitral valve and secondary mitral regurgitation (MR).

We retrospectively reviewed six consecutive preterm neonates with late-onset sepsis admitted between June 2024 and June 2025. All underwent neonatologist performed echocardiography (NPE) at the time of clinical deterioration.

All neonates presented with signs of warm shock, including tachycardia and hyperdynamic circulation. NPE demonstrated aliasing across the LVOT on color Doppler and a dagger-shaped spectral Doppler profile, indicative of LVOT acceleration. In three cases, dynamic LVOT obstruction with SAM and MR was demonstrated. Management included fluid boluses and vasopressors (norepinephrine); two cases required β-blockade (esmolol), resulting in resolution of obstruction and MR.

This case series describes a previously unreported echocardiographic phenotype associated with warm shock in preterm neonates, characterized by a spectrum ranging from LVOT flow acceleration to dynamic LVOT obstruction with SAM and secondary MR. NPE enabled early identification and pathophysiological understanding, and tailored management. Prospective studies are needed to validate these findings.

1. Introduction

Sepsis is a significant cause of morbidity and mortality in neonates, with cardiovascular compromise contributing to poor outcomes [1]. Sepsis-related cardiovascular dysfunction is traditionally classified into two main types: cold and warm shock and related to the degree of myocardial impairment and vasoregulatory failure, respectively [2]. A recent systematic review found that septic newborns commonly present with a warm shock physiology, characterized by reduced systemic vascular resistance (SVR) due to vasodilation, resulting in hypotension, normal or elevated cardiac output, rapid capillary refill, bounding pulses, and tachycardia [3]. Cold shock, in contrast, involves vasoconstriction, leading to pale or cold extremities, delayed capillary refill, generally decreased cardiac output, and weak pulses [4].

Although the classification appears straightforward, differentiating between warm and cold shock at the bedside remains challenging due to limitations in clinical assessment and the reliability of measurements such as blood pressure and capillary refill, particularly in preterm neonates [4]. Enhanced phenotyping through neonatologist-performed echocardiography (NPE) can improve diagnostic accuracy, uncover

Abbreviations: DoL, Day of Life; LVO, Left Ventricular Output; LVOT, Left Ventricular Outflow Tract; MR, Mitral Regurgitation; MV, Mitral Valve; NPE, Neonatologist Performed Echocardiography; SAM, Systolic Anterior Motion; SVR, Systemic Vascular Resistance.

* Corresponding author at: Neonatal Intensive Care Unit, University and Hospital Trust of Verona, P.le Stefani 1, 37126, Verona, Italy
E-mail address: benjamim.ficial@univr.it (B. Ficial).

https://doi.org/10.1016/j.ppedcard.2026.101908
Received 15 September 2025; Received in revised form 13 January 2026; Accepted 29 January 2026
Available online 30 January 2026

underlying pathophysiology, and guide targeted management strategies [5]. At our center, we routinely perform NPE using a structured protocol for image acquisition and quantitative measurements [6].

The aim of the study was to review cases of sepsis in preterm neonates presenting with warm shock, to determine whether a distinct cardiac phenotype is present during warm shock, as assessed by NPE, and to describe their management implications.

In our unit, hemodynamic assessment is supported by a dedicated consultation service run by neonatologists with expertise in NPE, providing prompt and comprehensive cardiovascular evaluation. The NPE protocol is described in the Supplemental File 1. We retrospectively reviewed six cases of preterm neonates with late-onset sepsis presenting with signs of warm shock admitted between June 2024 and June 2025. Echocardiographic studies were triggered by clinical suspicion of sepsis, including signs such as tachycardia, hypotonia, hyporeactivity, and low blood pressure. In all cases, the first echocardiographic assessment was performed at the clinical onset of sepsis, prior to initiation of antibiotic therapy or any cardiovascular treatment. This was followed by repeat examinations according to an individualized management plan—typically approximately one hour after initiation of any cardiovascular treatment and subsequently every 24–48 h, depending on clinical evolution. Written informed consent was obtained from the parents or legal guardians of all participants.

2. Case

All cases demonstrated a consistent echocardiographic pattern characterized by high peak velocities in the left ventricular outflow tract (LVOT), indicated by color Doppler aliasing at the level of the LVOT with a characteristic late-peaking, dagger-shaped pressure gradient on Doppler imaging (Fig. 1, Supplementary Video 1). These high peak LVOT velocities were observed across cases with variable severity, ranging from isolated LVOT flow acceleration to dynamic LVOT obstruction with systolic anterior motion (SAM) of the mitral valve (MV) and concomitant mitral regurgitation (MR). In the apical five-chamber view, this pattern was visualized on color Doppler imaging as a blue jet with a characteristic "V-shaped" configuration. The base of the "V" corresponds to color aliasing at the LVOT, consistent with dynamic obstruction. The left arm of the "V" represents systolic flow directed toward the aorta, whereas the right arm reflects regurgitant flow through the MV into the left atrium (Fig. 1).

Table 1 summarizes the main echocardiographic parameters for each case. Given the descriptive nature of this retrospective case series and the limited sample size, no formal statistical analyses were performed.

2.1. Case 1

This case involves a female neonate born at 24 + 4 weeks' gestation, who developed sepsis on day 10 of life (DoL). She exhibited tachycardia, episodes of apnea, and desaturation, which prompted an NPE assessment. The echocardiogram revealed a left ventricular output (LVO) close to the upper limit of normal, and good left ventricular systolic function [7]. There was a mild LVOT acceleration without SAM of the MV and MR. [8] The neonate responded favorably to volume expansion (10 mL/

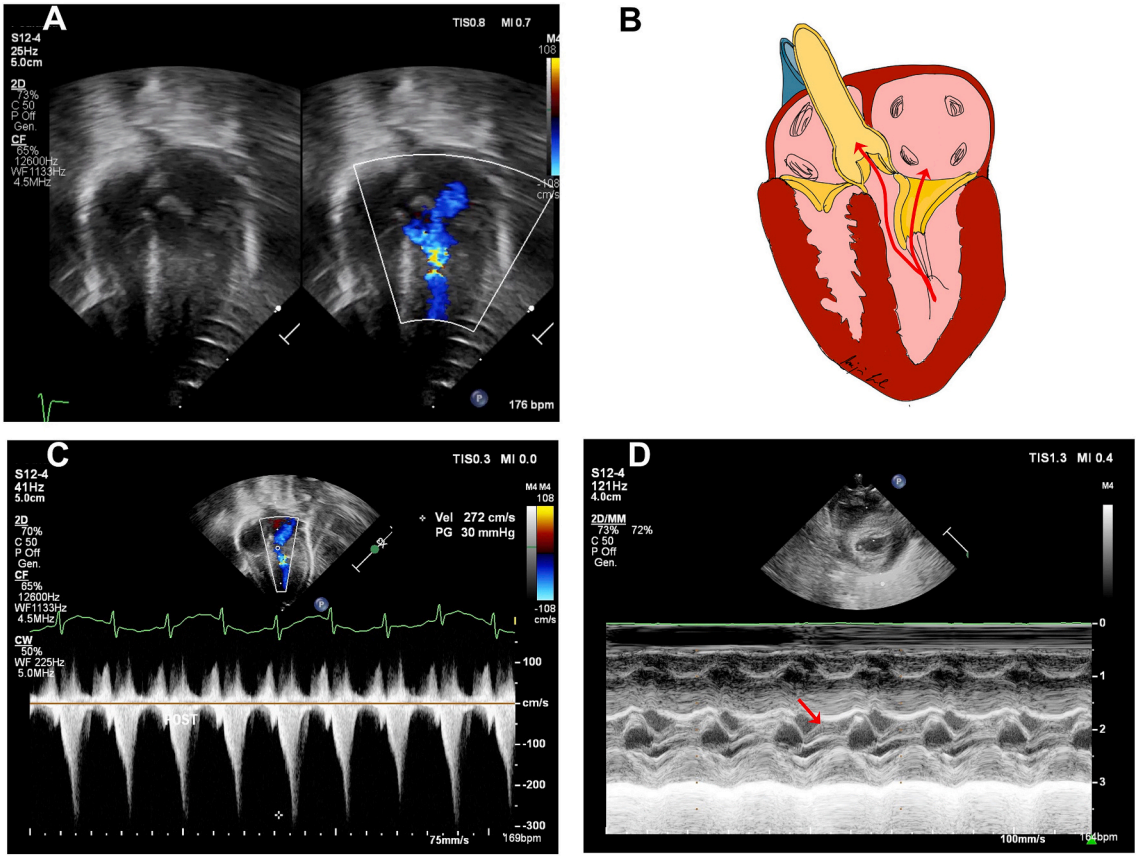

Fig. 1. Echocardiographic phenotype of warm shock. A) Apical 5-chamber view with color Doppler imaging demonstrating a blue jet with a characteristic "V" shape. At the base of the "V," aliasing is observed at the level of the left ventricular outflow tract (LVOT), consistent with dynamic obstruction. The left arm of the "V" represents flow directed toward the aorta, while the right arm indicates regurgitant flow through the mitral valve into the left atrium. B). Schematic of apical 5-chamber view showing dynamic LVOT obstruction due to systolic anterior motion (SAM) and associated mitral regurgitation. C) Continuous-wave Doppler tracing showing a characteristic dagger-shaped signal consistent with dynamic obstruction at the level of LVOT. D) M-mode echocardiography showing SAM of the mitral valve (red arrow). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 1
Clinical and echocardiographic data of the study neonates.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Clinical characteristics at birth						
Gestational age (week + day)	24 + 4	25 + 2	29 + 0	31 + 0	24 + 4	25 + 0
Birth weight (g)	755	875	580	1715	830	775
Sex	F	F	M	F	M	F
Mode of delivery	SVD	CS	CS	SVD	SVD	SVD
5' APGAR score	7	8	7	7	4	7
Antenatal steroids	yes	yes	yes	no	no	yes
Intrapartum antibiotics	no	yes	yes	no	no	yes
PROM >18 h	yes	no	no	no	no	no
Chorioamnionitis	yes	yes	no	no	no	yes
Postnatal steroids	no	no	no	no	no	no
Clinical characteristics at the onset of sepsis						
Day of Life	10	17	7	11	25	17
Postmenstrual age (week)	25 + 6	27 + 5	30 + 0	32 + 4	28 + 2	27 + 2
Body weight (g)	685	815	640	1640	960	880
Respiratory support	NIV	MV	NIV	MV	MV	MV
FiO_2 (%)	25	25	35	21	30	25
MAwP (cmH_2O)	6.9	6.8	9.3	9.1	15	12
preductal SpO_2	96	94	94	95	96	92
OSI	179	181	346	201	469	326
pH	7.30	7.45	7.16	7.28	7.29	7.37
CO_2 (mmHg)	30	55	55	56	63	36
BE (mmol/L)	−11.6	9.9	−9.5	−1.5	2	−3.9
lactate (mmol/L)	0.8	1.2	1.3	1.3	1.0	1.0
HR (bpm)	190	190	175	185	187	150
Blood pressure	Normotensive	Normotensive	Hypotensive	Hypotensive	Hypotensive	Normotensive
Systolic BP (mmHg)	75	74	50	55	44	55
Mean BP (mmHg)	50	48	28	30	20	35
Diastolic BP (mmHg)	37	35	21	25	15	27
Urine output (ml/kg/h - last 12 h)	4.7	2.5	3.0	4.5	5.2	3.9
Blood culture	*S. epidermidis*	*E. coli*	*S. warneri*	*K. pneumoniae*	*S. haemolyticus*	*E. coli*
CRP (mg/L)	7	15	10	163	7	49
PCT (mcg/L)	0.81	0.4	2.04	3.4	0.79	2.97
WBC (x 10^9 L)	42	26.15	2.9	7.0	18	23
Echocardiographic at the onset of sepsis/SIRS						
LVO (ml/kg/min)	280	233	265	260	349	287
RVO (ml/kg/min)	270	244	253	255	317	273
BP-EF (%)	55	50	65	64	63	69
GLS (%)	−18	−17	- 18	- 20	- 14.6	- 21
Mitral regurgitation, pressure gradient (mmHg)	none	100	48	none	69	none
Systolic anterior motion of the mitral valve (MV)	none	yes	yes	none	yes	none
Peak velocity of LVOT obstruction (m/s)	1.80	2.24	2.32	1.58	4.18	2.00
PDA	Closed	Closed	Closed	Closed	Closed	Closed
PAAT/RVET	0.3	0.3	0.35	0.3	0.4	0.3
EI	1.2	1.3	1.54	1.2	1.2	1.3
TAPSE (cm)	0.6	0.7	0.6	0.8	0.45	0.7
E/A (MV)	0.6	0.6	0.6	0.6	0.6	0.4
E/e' (MV)	11	15	12.3	7	NA	7.6
Follow-up						
Treatment	fluid bolus (10 mL/kg), norepinephrine	fluid bolus (10 mL/kg), increase in daily fluid intake (20 mL/kg/day), norepinephrine, esmolol	fluid boluses (20 mL/kg), increase in daily fluid intake (20 mL/kg/day), norepinephrine	fluid boluses (20 mL/kg), norepinephrine	fluid boluses (20 mL/kg), norepinephrine, esmolol	fluid boluses (20 mL/kg),
Outcome	Survived	Withdrawal of care	Died	Survived	Survived	Survived

Abbreviations: BP = blood pressure; BP-EF = Simpson's biplane ejection fraction; CRP = C-reactive protein; CS = cesarean section; EI = eccentricity index; F = female; GLS = global longitudinal strain; HR = heart rate; LVO = left ventricular output; LVOT = left ventricular outflow tract; M = male; MAwP = mean airway pressure; MV = mechanical invasive ventilation; NEC = necrotizing enterocolitis; NIV = non-invasive ventilation; OSI = oxygen saturation index; PAAT/RVET = pulmonary artery acceleration time/ right ventricle ejection time; PCT = procalcitonin; PDA = patent ductus arteriosus; PROM = premature rupture of membranes; RVO = right ventricular output; SVD = spontaneous vaginal delivery; TAPSE = tricuspid annular plane systolic excursion; WBC = white blood cell.

kg) and norepinephrine infusion, with a subsequent reduction in heart rate and improvement of the dynamic LVOT obstruction. Blood cultures were positive, and laboratory results were consistent with sepsis. The echocardiographic anomalies resolved within 48 h.

2.2. Case 2

A female neonate born at 25 + 2 weeks' gestation presented with sepsis on DoL 17. She exhibited clinical deterioration, characterized by tachycardia, abdominal distension, and pallor/mottled skin. Blood cultures and lumbar cerebrospinal fluid tests were positive for *E. coli*. The initial echocardiogram revealed a normal LVO with good systolic function. There was a mild LVOT obstruction, accompanied by SAM and MR. A fluid bolus of 10 mL/kg and an increase in daily fluid intake by 20 mL/kg/day were administered, along with initiation of norepinephrine. However, the dynamic obstruction and MR persisted, leading to the initiation of continuous esmolol infusion. Both the obstruction and the MR improved rapidly and resolved completely within 10 days, but due to severe neurological sequelae, care was ultimately withdrawn.

2.3. Case 3

The patient was a male infant born at 29 + 0 weeks' gestation, who developed clinical signs consistent with sepsis, including tachycardia, hypotension, and acute respiratory deterioration requiring mechanical ventilation on DoL 7. Echocardiography showed a mild LVOT obstruction and a mild secondary MR. These findings suggested a relative hypovolemia and excessive catecholamine stimulation, which exacerbated the LVOT obstruction. The accompanying MR caused elevated left atrial pressure, supported by the presence of a left-to-right atrial shunt, and may have contributed to the development of pulmonary edema. Initial management included fluid boluses totaling 20 mL/kg, followed by a further increase in fluid intake of 20 mL/kg/day, and vasopressor support using noradrenaline. Despite these interventions, the infant developed refractory shock with progressive circulatory collapse.

2.4. Case 4

A female infant born at 31 + 0 weeks' gestation presented on DoL 11 with feeding intolerance, abdominal distension, and respiratory deterioration. Echocardiography revealed a good systolic function and a mild LVOT acceleration, which resolved after initiation of fluids and norepinephrine.

2.5. Case 5

A male infant born at 24 + 4 weeks' gestation presented on DoL 25 with respiratory deterioration requiring high frequency ventilation, tachycardia, and biochemical evidence of late-onset sepsis. Echocardiography revealed severe LVOT obstruction without anatomical abnormalities, associated with SAM and severe MR, likely causing increased atrial pressure and pulmonary venous congestion. Despite fluid boluses totaling 20 mL/kg and norepinephrine, there was no initial improvement. During the clinical course, the infant rapidly developed left ventricular hypertrophy, which was hypothesized to be secondary to persistent dynamic LVOT obstruction, as no postnatal corticosteroids or insulin exposure occurred. Significant clinical and echocardiographic improvement occurred following initiation of esmolol after 48 h, which sharply reduced the LVOT obstruction. On this basis, beta-blockade was maintained to mitigate hypercontractility and prevent recurrence of obstruction. Esmolol was progressively weaned and transitioned to oral propranolol, which was discontinued on DoL 60.

2.6. Case 6

A female infant born at 25 + 0 weeks' gestation developed sepsis on DoL 17, presenting with respiratory deterioration. Echocardiography revealed a mild LVOT acceleration that disappeared with fluid boluses totaling 20 mL/kg.

3. Discussion

To the best of our knowledge, this is the first reported association between sepsis, LVOT acceleration, and dynamic LVOT obstruction in preterm neonates with a clinical phenotype of warm shock. Recognition of this pattern in neonates is critical, as it helps individualize the approach based on the underlying pathophysiology.

All cases in our neonatal series were characterized by the onset of warm shock. The initial trigger was vasoplegia due to sepsis, leading to low afterload, loss of intravascular volume from capillary leak into the "third space," and relative hypovolemia. These changes activated a compensatory sympathetic response, resulting in tachycardia and increased myocardial contractility—a classic hallmark of warm shock physiology [3,4].

The echocardiographic hallmark of this response was a blue jet with color Doppler aliasing at the LVOT with a characteristic late-peaking, dagger-shaped pressure gradient on Doppler imaging, both indicative of dynamic LVOT acceleration (Fig. 1, Supplementary Video 1). High peak LVOT velocities, commonly observed in hypertrophic cardiomyopathy, can also be observed in healthy children and adolescents without left ventricular hypertrophy, particularly after exercise. In these settings, the underlying physiology remains incompletely understood but is thought to reflect dynamic intracavitary narrowing rather than true obstruction at the LVOT [9]. This intracavitary flow acceleration is likely multifactorial and may be related to the combination of hyperdynamic systolic function, reduced preload, and reduced afterload. Therefore, although these findings are not specific to warm shock, they are frequently encountered in this clinical scenario [9].

This pattern was observed in Cases 1, 4, and 6, in which only mild LVOT acceleration was detected (Aortic $V_{max} < 2$ m/s), without evidence of systolic anterior motion (SAM). These findings likely represent the milder end of a physiological spectrum of hyperdynamic flow rather than pathological obstruction [8].

At the opposite end of the spectrum, Cases 2, 3, and 5 demonstrated clear dynamic left ventricular outflow tract obstruction, characterized by high Doppler-derived gradients, the presence of SAM of the MV, and secondary MR, with severity graded according to Doppler-derived peak aortic velocities (mild: Vmax 2.0–2.9 m/s; moderate: Vmax 3.0–3.9 m/s; severe: Vmax $\geq$4.0 m/s), as reported in recent guidelines [8]. This pattern closely resembles dynamic LVOT obstruction described in hypercontractile states such as hypertrophic cardiomyopathy and adult septic shock [10–12].

This obstruction is likely due to a combination of reduced left ventricular filling, hypercontractility, and reduced afterload, which together create the ideal conditions for promoting systolic anterior motion (SAM) of the MV [10–12]. The anterior mitral leaflet moves toward the interventricular septum during systole, narrowing the LVOT. This is, at least in part, driven by the Venturi effect, where high-velocity blood flow through the narrowed outflow tract creates a low-pressure zone, drawing the mitral leaflet into the LVOT and causing a dynamic obstruction [13].

MR may occur secondary to SAM when the anterior displacement of the mitral leaflet disrupts leaflet coaptation, allowing regurgitant flow into the left atrium. The presence of SAM and MR may depend on multiple factors, including blood flow velocity in the LVOT, interventricular septal hypertrophy, and the intrinsic anatomy of the MV apparatus, including the leaflets, papillary muscles, and chordae tendineae [13]. When MR was present, it likely contributed to elevated left atrial pressure, pulmonary venous congestion, and subsequent pulmonary edema, providing a mechanistic explanation for the observed respiratory deterioration in these neonates [10,14].

In this setting, fluid resuscitation was triggered by the presence of

dynamic LVOT obstruction, with the aim of increasing preload and alleviating the obstruction. Fluid boluses and/or an increase in daily fluid intake were therefore considered key components of management to increase left ventricular end-diastolic volume, even in the presence of MR. Importantly, the MR was interpreted as functional and related to SAM of the MV rather than to annular dilation or structural valve disease. Accordingly, volume expansion was directed toward optimizing left ventricular filling to mitigate LVOT obstruction, with the intention of secondarily reducing SAM and MR, while carefully monitoring for potential pulmonary congestion with serial lung ultrasound assessments.

Another cornerstone of management in this physiological context is appropriate vasopressor selection. Avoiding agents with mixed α- and β-adrenergic effects (such as dopamine or epinephrine) in favor of predominantly α-adrenergic agonists (e.g., norepinephrine) may improve cardiac filling by increasing systemic vascular resistance and arterial pressure, thereby reducing reflex tachycardia and limiting dynamic LVOT obstruction without a marked increase in contractility. Vasopressin may represent an attractive alternative in this setting, as it provides vasoconstriction without β-adrenergic effects; however, current evidence in neonates remains limited, and further studies are required [10].

Finally, as demonstrated in two of our cases, there was a physiological rationale for the use of β-blockade in the specific setting of dynamic LVOT obstruction. β-blockers reduced heart rate, increased diastolic filling time, and mitigated hypercontractility, thereby decreasing LVOT obstruction and secondary MR. Although potential benefits of β-blockade have been reported in adult patients with warm septic shock, its use remains controversial [15]. Importantly, β-blockade is not a conventional therapy in neonates, and clinicians should be fully aware of its potential complications, including bradycardia, hypotension, and reduced cardiac output. Its use should therefore be considered only in carefully selected cases with echocardiographically confirmed dynamic LVOT obstruction and under close monitoring.

3.1. Limitations

We acknowledge that this study is a retrospective case series with a small sample size, conducted at a single center, which limits the generalizability of our findings. The retrospective design may be subject to incomplete data capture and unmeasured confounders, and the single-center experience reflects local clinical practices that may not be universally applicable. In addition, potential selection bias cannot be excluded, as echocardiography was performed based on clinical indication in critically ill neonates. As such, our findings should be considered hypothesis-generating and intended to stimulate further prospective and larger-scale studies to validate this echocardiographic phenotype and its clinical implications.

4. Conclusions

This case series describes a previously unreported echocardiographic phenotype associated with warm shock in preterm neonates, characterized by a spectrum ranging from LVOT flow acceleration to dynamic LVOT obstruction with SAM of the MV and secondary MR. These findings highlight the complexity of cardiovascular responses during neonatal sepsis and underscore the potential value of echocardiography in improving pathophysiological understanding of warm shock. Further multicenter, prospective studies are required to determine the prevalence, clinical significance, and prognostic implications of this phenotype, as well as its potential impact on management strategies and outcomes.

CRediT authorship contribution statement

Benjamim Ficial: Writing – review & editing, Writing – original draft, Visualization, Investigation, Formal analysis, Conceptualization. **Beatrice Allena:** Writing – review & editing, Writing – original draft, Data curation. **Ilaria Boffa:** Writing – review & editing, Data curation. **Arianna Zuccato:** Writing – review & editing, Data curation. **Daniele Mosolo:** Writing – review & editing, Data curation. **Martina Ciarcià:** Writing – review & editing, Formal analysis. **Federica Runfola:** Writing – review & editing, Formal analysis. **Silvia Nogara:** Writing – review & editing, Formal analysis. **Iuri Corsini:** Writing – review & editing, Conceptualization. **Willem P. de Boode:** Writing – review & editing, Supervision.

Informed consent

The parents or guardians of babies enrolled gave their written informed consent. The authors affirm that human research participants provided informed consent for publication of the images in Fig. 1 and Supplemental Video 1.

Ethics approval

The study was approved by the Research Ethics committee of the University Hospital of Verona, no. 2132CESC. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

None declared.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ppedcard.2026.101908.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

References

[1] Fleischmann-Struzek C, Goldfarb DM, Schlattmann P, Schlapbach LJ, Reinhart K, Kissoon N. The global burden of paediatric and neonatal sepsis: a systematic review. Lancet Respir Med 2018;6:223–30.
[2] Duignan SM, Lakshminrusimha S, Armstrong K, de Boode WP, El-Khuffash A, Franklin O, et al. Neonatal sepsis and cardiovascular dysfunction I: mechanisms and pathophysiology. Pediatr Res 2024;95:1207–16.
[3] Pugnaloni F, De Rose DU, Kipfmueller F, Patel N, Ronchetti MP, Dotta A, et al. Assessment of hemodynamic dysfunction in septic newborns by functional echocardiography: a systematic review. Pediatr Res 2024;95:1422–31.
[4] Kharrat A, Jain A. Hemodynamic dysfunction in neonatal sepsis. Pediatr Res 2022;91:413–24.
[5] de Boode WP. Individualized Hemodynamic Management in Newborns. Front Pediatr 2020;8:580470.
[6] Ficial B, Corsini I, Clemente M, Cappelleri A, Remaschi G, Quer L, et al. Feasibility, reproducibility and reference ranges of left atrial strain in preterm and term neonates in the first 48 h of life. Diagnostics (Basel) 2022;12.
[7] McNamara PJ, Jain A, El-Khuffash A, Giesinger R, Weisz D, Freud L, et al. Guidelines and recommendations for targeted neonatal echocardiography and cardiac point-of-care ultrasound in the neonatal intensive care unit: an update from the American Society of Echocardiography. J Am Soc Echocardiogr 2024;37:171–215.
[8] Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a

report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Thorac Cardiovasc Surg 2021;162:e183–353.

[9] Wittlieb-Weber CA, Cohen MS, McBride MG, Paridon SM, Morrow R, Wasserman M, et al. Elevated left ventricular outflow tract velocities on exercise stress echocardiography may be a normal physiologic response in healthy youth. J Am Soc Echocardiogr 2013;26:1372–8.

[10] Dugar S, Latifi M, Moghekar A, Duggal A. All shock states are not the same. systolic anterior motion of mitral valve causing left ventricular outflow tract obstruction in septic shock. Ann Am Thorac Soc 2016;13:1851–5.

[11] Auer J, Berent R, Weber T, Lamm G, Eber B. Catecholamine therapy inducing dynamic left ventricular outflow tract obstruction. Int J Cardiol 2005;101:325–8.

[12] Yang JH, Park SW, Cho SW, Kim HS, Choi KA, Kim HJ. Dynamic left ventricular outflow tract obstruction without basal septal hypertrophy, caused by catecholamine therapy and volume depletion. Korean J Intern Med 2008;23:106–9.

[13] Guigui SA, Torres C, Escolar E, Mihos CG. Systolic anterior motion of the mitral valve in hypertrophic cardiomyopathy: a narrative review. J Thorac Dis 2022;14:2309–25.

[14] Ficial B, Dolce P, Petoello E, Flore AI, Nogara S, Ciarcià M, et al. Left atrial strain assessment unveils left ventricular diastolic dysfunction in neonates with transient tachypnea of the newborn: a prospective observational study. Pediatr Pulmonol 2024;59:2910–21.

[15] Alexandru MG, Niewald P, Krüger S, Borgstedt R, Whitehouse T, Singer M, et al. Mortality in septic patients treated with short-acting betablockers: a comprehensive meta-analysis of randomized controlled trials. Crit Care 2024;28:392.