Role of Point-of-care Ultrasound Imaging in Neonatal Sepsis

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ABSTRACT

Neonatal sepsis remains a very common cause of morbidity and mortality. Point-of-care ultrasound imaging is a useful tool to determine the site of infection, the progress of sepsis, and prognosis/outcomes of septic neonates. The organ system imaging that is useful includes cardiac ultrasound for delineation of the hemodynamics, cranial imaging for changes in meningitis/ventriculitis, lung ultrasound for early detection of effusions and consolidation, and gut imaging for gut perfusion and viability in necrotizing enterocolitis. We describe the role of ultrasound imaging in the early diagnosis and management of neonatal sepsis.

Keywords: Cardiac hemodynamics, Gut ultrasound, Lung ultrasound, Neonates, Point-of-care ultrasound, Sepsis.

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INTRODUCTION

Sepsis is one of the three most common causes of neonatal deaths globally.¹ In very low birth weight (VLBW) infants, sepsis has been an important cause of neonatal mortality and morbidity. More than one culture-proven sepsis episodes are seen in 25% of VLBW infants, and the overall mortality rate is 17%. Infection is the main cause of death in VLBW infants after second week of life which accounts for 40% of deaths.² Poor neurodevelopmental and growth in early childhood are seen in extremely low birth weight infants with neonatal infections.³ Therefore, early recognition and appropriate management is imperative for good outcomes. Point-of-care ultrasound imaging has become a useful tool in the hands of the clinician to determine the site of infection, the progress of sepsis, and prognosis/outcomes of neonates with sepsis.

We aim to describe the role of ultrasound imaging in neonates with signs of sepsis, including the alterations in cardiac hemodynamics and infective pathologies in the lung, gut, and cranial ultrasounds.

Role of Functional Echocardiography in Neonatal Sepsis/Septic Shock

There are developmental differences in the hemodynamic responses to sepsis in newborns, children, and adults. This has been well articulated by the "American College of Critical Care Medicine Clinical Practice Parameters for Hemodynamic Support of Pediatric and Neonatal Septic Shock" published in 2017⁴ and by other authors.^{5,6} Vasomotor paralysis is the predominant cause of mortality in "adult septic shock," leading to low systemic vascular resistance associated with myocardial dysfunction.^{4,5} "Pediatric septic shock" is typically associated with severe hypovolemia, and aggressive volume resuscitation is needed.⁴

At variance from adults and children, neonates are in the process of undergoing a physiologic transition from fetal to neonatal circulation. Initially, the ductus arteriosus can remain open and pulmonary artery pressures are elevated.^{4,5} "Neonatal septic shock" is, therefore, to be viewed alongside these physiological changes. The pulmonary artery pressures increase further due to sepsis-induced acidosis and hypoxia. This results in persistent pulmonary hypertension of the newborn (PPHN). There is increased right ventricular work, manifested as tricuspid regurgitation, right ventricular dysfunction, and hepatomegaly. The delivery of oxygen

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and nutrients to tissues is affected by myocardial dysfunction, abnormal peripheral vasoregulation, and hypovolemia. Therapies need to be directed at reversal of right ventricle failure through reduction of pulmonary artery pressures, augmentation of LV contractility, and volume support appropriately. The functional immaturity of the adrenal gland in premature infant results in relative adrenal insufficiency. This could lead to cardiovascular dysfunction.^{4,7}

The extra information on hemodynamic state in neonates with sepsis can be obtained by functional echocardiography which is probably the only diagnostic tool.⁸ This aids in choice of inotropic and vasopressor support.

Right Ventricle and Left Ventricle Cardiac Outputs (CO) and Superior Vena Cava (SVC) Blood Flow Measurements

Measurements of CO and SVC flows need to be done because it could be used to determine the type of inotropic and vasopressor support that may be needed in a clinical situation.⁹ The factors such as preload, contractility, and afterload affects the cardiac output and is a good marker of cardiac dysfunction in preterm infants. Cardiac Output (CO) = Heart Rate (HR) × Stroke Volume (SV); therefore, factors affecting SV like sepsis associated endotoxins reduce CO unless there is a compensatory tachycardia. An increase in HR by twofold from 70 to 140 beats/minute can compensate for the fall in SV in an adult. But a baby cannot increase the HR significantly, since baselines HR's are already high. So, while tachycardia is an

important response to maintain CO in neonates, this response (rise in HR) is likely to be inadequate and the CO falls.⁴ Therapies directed to increasing SV will often, reflexively, reduce HR and improve CO.

The mean arterial pressure = CO × SVR. Therefore, the hemodynamic variables that determine the blood pressure (BP) is combination of factors affecting CO and systemic vascular resistance (SVR). In a setting of sepsis, vasoconstriction to maintain BP is a the compensatory response to falling SV.⁴

In a study of 318 preterm neonates <34 weeks by de Waal¹⁰ et al., 4.7% ($n = 27$) had evidence of sepsis with hemodynamic compromise. The mean RVO, LVO, and SVC flows were 430, 300, and 90 mL/kg/minute, respectively, in stable preterm neonates <32 weeks' gestation with minimal respiratory support.^{10,11} de Waal¹⁰ et al. noted that preterm neonates with sepsis had relatively high left and right cardiac outputs and low systematic vascular resistance (SVR), after the initial volume support similar to the adult with sepsis. The mean RVO, LVO, and SVC flow at the first measurement was 555, 441, and 104 mL/kg/minute, respectively (Fig. 1). The mean (SD) calculated SVR was 0.08 (0.04) mm Hg/mL/kg/minute. There was a significant decrease in flow and an increase in SVR in the nonsurviving infants (Fig. 2). From the start till improvement within the sepsis episode among the survivors, there were no change in flow variables, while the SVR showed a mild increase (mean difference in SVR, 0.02 mm Hg/mL/kg/minute). Mortality risk was high when RVO or LVO decreased >50% compared to baseline.¹⁰

Similar findings have been replicated in other studies.^{12,13} Early in the course of pediatric septic shock, neonates have dysregulated cytokine and chemokine production. This leads to peripheral vasodilatation which is considered to be a major hemodynamic change.^{12,14} Therefore, persistent high-cardiac output and low SVR might benefit from increasing doses of potent vasopressors.¹⁰ Conversely, when flows or cardiac outputs are reduced, reducing vasopressor therapy and adding afterload reduction could prove beneficial (Table 1).

Deshpande et al.¹³ have shown that in gram-negative sepsis the mean RVO and LVO were significantly higher (338 and 378 mL/kg/minute, respectively), and in gram-positive sepsis they were in normal range (225 and 240 mL/kg/minute, respectively). There was no significant difference in the mean ventricular outputs comparing term and preterm infants with late-onset sepsis (p value = 0.422).¹³

Fig. 1: CO in sepsis survivors and non-survivors¹⁰ (permission from author obtained)

The effectiveness of shock therapies can be assessed by ultrasound-derived SVC blood flow measurement in VLBW infants. The SVC flow approximates blood flow from the brain. Improved neurologic outcomes and survival is seen when the value is greater than 40 mL/kg/minute.^{10,15,16}

Besides vasoregulatory failure leading to high LV output, myocardial dysfunction could coexist in neonates with septic shock leading to LVO <150 mL/kg/minute as was noted in three neonates in the study by Saini et al.¹²

Patency of Duct and Direction of Flow

A significantly higher proportion of neonates with septic shock have PDA. Saini et al.,¹² in their study of neonates with sepsis, determined that 60% of the septic neonates had a patent duct. Infection promotes patency of PDA by increased cyclooxygenase expression and prostaglandin levels.¹⁷ The flow patterns could be left to right or bidirectional (in case of associated pulmonary hypertension) (Figs 3 and 4).

Persistent Pulmonary Artery Hypertension (PPHN) of the Newborn in Sepsis

Mohsen and Amin¹⁸ have reported that in 43.7% of cases neonatal sepsis was the cause of pulmonary hypertension, which is a second most common cause of pulmonary hypertension. In neonatal sepsis, there is a vascular response to a cytokine storm, which causes markedly elevated pulmonary vascular resistance leading to instability in pulmonary vascular tone and thus causing persistent pulmonary hypertension of the newborn (PPHN). Pulmonary vascular resistance and artery pressure can increase with sepsis-induced acidosis and hypoxia. This maintains ductus arteriosus patent that results in PPHN and persistent fetal circulation.

While a comprehensive review of pulmonary hypertension is beyond the scope of this article, a functional echo assessment in sepsis-associated pulmonary hypertension includes measurement of Peak TR velocity to determine the pulmonary pressures (Fig. 5), pattern and flow of the ductal shunt, measurements of the acceleration time in the main pulmonary artery, position of the interventricular septum, direction of the atrial shunt, and cardiac contractility. Not all the parameters would be seen in each case.

Fig. 2: Systemic vascular resistance in sepsis survivors and non-survivors¹⁰ (permission from author obtained)

Table 1: Role of functional echocardiography in sepsis

S. no.	Variable measured	Parameter measured	Changes seen in sepsis
1.	Cardiac flows	Right ventricular output	Increased/low
		Left ventricular output	Increased/low
		Superior vena cava flows	Low
2.	Ductus arteriosus	Patency, size, flow pattern, velocities	Patent, bidirectional shunt seen in sepsis
3.	Pulmonary hypertension	TR jet, ductal flows	High pulmonary pressures
		Dicrotic notch	
		Interventricular septal position	
		PAAT: RVET	
4.	IVC distensibility	Diastolic function	Fluid responsiveness seen
		IVC diameters, distensibility, collapsibility	
5.	Ventricular diastolic function	Ventricular diastolic filling (E/A)	Impaired (<0.6–0.7)

Fig. 4: Bidirectional duct in sepsis. Bidirectional shunt. As pulmonary pressures rise, pressures during cardiac systole rise > than during diastole. Pulmonary pressure during systole exceeds systemic pressures but diastolic pressures are subsystolic. Hence there is a bidirectional shunt

Sepsis-associated hypoxia and acidosis cause an increase in pulmonary pressures and increased right ventricular work, which manifests as tricuspid regurgitation (TR). The TR jet is reportedly seen in 60–85% cases of PPHN.¹⁹

There is a change in RV outflow spectral Doppler pattern from a smooth round shape (Fig. 6A) to a triangular shape (Fig. 6B) in rising pulmonary artery pressure. Presence of a dicrotic notch suggests presence of pulmonary hypertension. As the pulmonary artery pressure rises, the 'Pulmonary Artery Acceleration Time (PAAT)' or 'Time to Peak Velocity' gets shorter.

Fig. 3: Left ventricular output (LVO). LVO Measurement: Suprasternal approach to ascending aortic doppler velocity signals. Flow profile should be laminar. This image shows 'hollow' signals on the spectral display indicating non-turbulent flow. Range of LVO = 170–300 mL/kg/minute

Fig. 5: Tricuspid regurgitation (TR) jet. Estimation of pulmonary artery pressures. TR jet velocity is used to derive the systolic pressure gradient between the RV and RA using Bernoulli's principle. Right ventricular systolic pressure (RVSP) = pressure gradient + RA pressure (5–10 mm Hg). In a normal heart (in the absence of pulmonary stenosis), RVSP = pulmonary artery systolic pressure

Pulmonary hypertension can be highly predicted in neonates when PAAT is <90 ms.²⁰ Since PAAT values are affected by heart rate, a ratio of PAAT: right ventricular ejection time is taken and values <0.31 are highly suggestive of PH.^{20,21}

The position of the interventricular septum may be determined by the pulmonary pressures. IVS can be flattened in pulmonary hypertension (PH), which makes the left ventricle appear as a D-shaped structure. The patients with sepsis and pulmonary hypertension may be classified as having an "O"-shaped (no PH), "D"-shaped (flat septum, moderate PH), or "Crescentic"-shaped LV (severe PH) (Figs 7A and B).^{15,23}

Tricuspid Annular Plane Systolic Excursion (TAPSE) (Fig. 8)

The systolic displacement of the tricuspid annulus toward the RV apex along the longitudinal axis can be measured by a TAPSe which is a sensitive measure of RV systolic function.^{19,20} The greater the descent of the basal annulus in systole, the better the RV systolic function. In term neonates, the mean value is 0.91 cm (0.68–1.15).¹⁹ The TAPSE values vary depending on the gestation age and weight of the neonate.

Figs 6A and B: (A) RV outflow doppler: smooth laminar flow; (B) Presence of dirotic notch

Figs 7A and B: (A) 'O' shaped IVS; (B) 'D' shaped IVS

Figs 8A and B: (A) TAPSE measurement—measure along free wall of tricuspid valve; (B) TAPSE measurement—'M Mode' excursion distance measured

Table 2: Inferior vena cava (IVC) diameter and variations in sepsis²¹

IVC diameter	IVC respiratory variation	Clinical status
<8 mm	>50% collapse	Fluid responsive
<8 mm	<50% collapse	May give fluids
>8 mm	<50% collapse	Normal
>8 mm	No inspiratory collapse	High fluid status (RV failure, high pulmonary pressures, hypervolemia)

In neonates with fluid-refractory shock and PPHN, the pulmonary artery pressures can be reduced by therapies directing at reversal of right ventricular failure.⁴

Inferior Vena Cava (IVC) Distensibility Variations (Table 2)

One of the challenges in management of sepsis is to know which neonate will respond to a fluid challenge. A quantitative method

is to measure the IVC diameter and determine the variation in diameter with respiration. In sepsis, this variation during respiration can assist in diagnosis of hypovolemia and guide fluid therapy.

The chest volume expands during inhalation as the dome of the diaphragm is lowered (flattened). This increases abdominal pressure resulting in compression of the wall of the inferior vena cava, thus creating the smaller dimension of the vessel. This is called the "inspiratory collapse."

There are two measures that are taken into consideration: the IVC collapsibility index and the IVC distensibility index (Fig. 9).²⁰

$$\text{IVC Collapsibility Index} = \frac{D_{\max} - D_{\min}}{D_{\max}} \times 100\% \quad (D = \text{diameter of IVC})$$

$$\text{IVC Distensibility Index} = \frac{D_{\max} - D_{\min}}{D_{\min}} \times 100\%$$

While an IVC Collapsibility index >55% is predictive of fluid responsiveness, an IVC distensibility index >18% is predictive of fluid responsiveness. However, these values need to be interpreted

Fig. 9: Inferior vena cava status.²¹ Key findings in favor of hypovolemia on 2D imaging. LV and RV of small dimensions and hyperkinetic. Small LV end-systolic and end-diastolic areas. Increased LV ejection fraction ($>70\%$). Small IVC diameter with wide respiratory variation

Figs 10A and B: Assessment of diastolic function (E/A ratio). Diastolic function: 'E wave': phase of passive ventricular filling. 'A wave': atrial contraction phase. Normal 1:1 ratio. Diastolic filling abnormality: E/A $<0.6-0.7:1$ (Fig. 7B)

with caution, since studies have validated these in self-breathing adults.²⁰

Impaired Ventricular Diastolic Function

Babies with neonatal sepsis may also have an impaired diastolic filling although this may not be a universally recognized feature. Relaxation of the myocardium is an active process. Transmittal flow occurs in two phases: majority of the filling occurs ('E' wave) in an early phase of passive flow across AV valve. The remaining third of the venous return to the ventricle is delivered ('A' wave) in the late atrial contraction phase. The comparison of maximum E and A wave velocities are done as ratios.

The majority of filling occurs during the atrial phase in diastolic dysfunction, as the passive early flow across mitral valve is prevented by the stiff ventricular wall. This biphasic pattern of transmittal flow can be examined by pulsed wave Doppler. In normal children, E filling is greater than A. In neonatal period, mean normal ratio is 1:1 in preterm and 1.1:1 in full-term neonates. A ratio of $<0.6:1$ in preterm and $<0.7:1$ in term babies is highly suggestive of diastolic filling abnormality (Fig. 10).²²

In a study on LV functions in septic and nonseptic neonates ($n = 60$), Tomerak et al.²³ noted significantly lower E/A ratio in the septic than in the nonseptic newborns, suggesting LV diastolic dysfunction. No difference was noticed in the LV systolic function between septic and nonseptic neonates. Impaired diastolic function is also seen as a characteristic feature in the immature fetal and preterm myocardium, and therefore, this may not be diagnostic in neonatal sepsis.

Role of Cranial Ultrasound in Sepsis^{24,25}

The common manifestation of late-onset neonatal sepsis is neonatal bacterial meningitis. The assessment of infants with clinical suspicion of bacterial meningitis, and follow-up of its complications can be done by cranial ultrasonography (CUS). The intraventricular contents, especially debris and intraventricular septations, can be evaluated by CUS. It is superior to computed tomography and magnetic resonance imaging as an imaging modality due to absent need for sedation and lack of ionizing radiation, speed of imaging, low cost, and easy portability.

Infection of cerebrospinal fluid (CSF) and subsequently ventriculitis result from seeding of choroid plexus caused by bacteremia. Pia and arachnoid mater including the vessels traversing the subarachnoid space are also infected. The permeability of membrane and vessel wall is increased by the inflammatory response and thus causing accumulation of inflammatory exudates.

The common sonographic findings include (Table 3):

- Echogenic sulcus: it is the most common finding and seen as the earliest finding. Sulcal thickness should not normally be >2 mm as this includes pia and arachnoid mater. Accumulation of inflammatory exudates can result in widening and increased echogenicity (Fig. 11).
- Ventriculomegaly occurs due to increased production and decreased CSF absorption during the acute phase of bacterial meningitis (Fig. 12).
- Ventriculitis: it is identified as increased thickness, irregularity, and increased echogenicity of the ependyma. This occurs because ventricles and choroid plexus act as reservoirs of infection. This can cause it to harbor bacteria, but the lumbar puncture can yield sterile cultures. Inflammatory exudate may undergo organization into strands and fibrous septae in long-standing chronic cases (Figs 13A and B).
- Abnormal resistive index (RI) (Fig. 14): There is a paucity of literature explaining the effect of sepsis on cerebral blood flow through early cerebrovascular alteration. In early sepsis, cerebral vasodilation occurs because of production of cytokines and interleukins, thus increasing cerebral blood flow causing decreased RI.²⁶ However, decreased cerebral blood flow with increased RI can be caused by sepsis by unknown mechanisms, likely due to vasoconstriction of cerebral resistance arterioles.²⁷ Decreased compliance of the

Table 3: Sonographic findings in neonatal bacterial meningitis²⁴

Findings	Description	Significance
Echogenic sulci	Sulcal thickness >2 mm	Often transient
Ventriculitis	Echogenic debris within ventricular cavity; late stages: septae formation, compartmentalization, intraventricular cysts; choroid plexitis: ↑echogenicity and irregularity of choroid plexus	Causes lack of response to antibiotics Choroid plexus infections cause recurrent infections Requires long-term follow-up for hydrocephalus
Ventriculomegaly/hydrocephalus	Lateral ventricle width >11 mm at level of body	Acute stage: due to CSF production and decreased absorption Chronic stage: fibrotic obstruction
Miscellaneous	Periventricular hypoechoogenicity due to periventricular ooze Intraventricular hemorrhage: grade II–III with/without post-hemorrhagic ventricular dilatation Echodensities in thalamus, basal ganglia	Parenchymal atrophy Long-term neurodevelopment may be affected

Fig. 11: Widened echogenic cerebral sulci

Fig. 12: Dilated frontal horns of lateral ventricle

Figs 13A and B: Coronal view of AF with fibrous strands (A) and septae (B) suggesting ventriculitis

vascular bed results in increased RI, and it is associated with poor neurological outcomes.

- Brain abscess: This is a rarely reported complication in neonates (Fig. 15). Clinical data along with serial CUS examinations may differentiate between an early phase abscess and an ischemic process, thus helping in making a correct diagnosis.

Role of Lung Ultrasound (LUS) in Detection of Infective Pathologies

The two infective pathologies, that LUS would be useful in, are lung consolidation and pleural effusions. In pediatric age-group, lung consolidation can be accurately diagnosed by LUS when compared to chest radiography in pediatric age-group, and this is backed by a strong evidence.²⁹

Lung Consolidation

A positive LUS can confirm a clinical suspicion of pneumonia and can exclude the need for CXR. If the consolidation does not reach the pleura, the LUS may not detect the consolidation. The extension to the pleura may occur more frequently as the

newborn lung mass is smaller.²⁹ The echogenicity and echotexture of the consolidated airless lung are similar to liver, hence it appears as hepatization (Fig. 16). The air bronchogram visible on standard chest radiographs appears as multiple bright dot-like and branching linear structures in the solid-appearing area of echogenicity that represents air in the bronchi (Fig. 17).³⁰

The “Shred Sign” is a sonographic sign seen in neonates with lung consolidation (Fig. 18). The area of contact between the consolidated lung and the deeper aerated lung appears shredded and irregular. In massive consolidation, the sign is not well seen, since it is more difficult to visualize very deep structures.

Pleural Effusion

It is visualized as Quad Sign, which is an anechoic space between the parietal and visceral pleura (Fig. 19). The nature of the effusion can be diagnosed by lung ultrasound. It is considered as exudate (Fig. 20) or hemothorax when there is visualization of internal echoes, either of mobile particles or septa. However, when there is an anechoic effusion (Fig. 20), the transudate and exudate can be differentiated by use of thoracentesis or alternatively can be evaluated in the clinical context.

Fig. 14: High RI with reversal of diastolic flow in anterior cerebral artery

Fig. 15: Coronal view at the ant horn level—hyperechoic area in left temporal lobe—suggestive of brain abscess²⁸

Fig. 16: Hepatization of the subpleural lung tissue

Fig. 17: Multiple bright dot-like, branching linear structures, represent air in bronchi

Figs 18A and B: Shred sign: Specific to lung consolidation, white arrows indicate pleural line. (A) Pleural line intact; (B) Pleural line is shredded

Figs 19A and B: Quad sign. (Minimum pleural effusion) Quadriateral space between the parietal and the visceral pleura (indicated by white arrows in (A), and the shadow of rib margins laterally

Role of US in Necrotizing Enterocolitis (NEC)^{31,32}

The physiology and tissue architecture of the gut is disrupted by a severe inflammatory condition of the bowel in preterms called as NEC. Abdominal X-rays have remained the gold standard so far, but there is an increasing role for abdominal US in diagnosing NEC. The static physical characteristics, such as bowel wall thickness, wall diameter, dilatation, and echogenicity, as well as dynamic characteristics of peristalsis and perfusion, can be determined by US imaging in NEC. Since preterm and term neonates are small, and have thin abdominal walls, bowel US is easy to perform.

Physical Characteristics

These include bowel wall thickness, wall diameter, dilatation, and echogenicity. It is described that the bowel wall thickness in a normal term neonate is 1.1–2.6 mm, while it is estimated to be 1.2–2 mm in preterms babies. It is likely to be NEC when the bowel wall thickness is >2 mm; abnormal thinning with a thickness of <1.0 mm results from ischemia or necrosis.³³

Blood Flow

Normal bowel wall is smooth with peristalsis, and it is said to normally perfused when there are 1–9 color Doppler signal dots per cm² (mean 3.8). In early NEC, there is an increased bowel perfusion that can appear with different patterns such as ring-shape, Y-shaped, and zebra-shaped (Fig. 21).

Intramural Gas

This is seen as highly echogenic dots in the bowel wall, called as "circle sign," as it may involve the whole circumference (Fig. 22). The clinical severity of NEC cannot be determined by the amount of intramural gas present, and its disappearance does not correlate with clinical improvement.³⁴ BUS detects portal venous gas better than radiographs due to the high echogenic signal air produced.³⁵

In a study by Silva³⁶ et al., there was a sensitivity of 0.82 and a specificity of 0.78 for poor outcome when three of the seven USG features (portal venous gas, intramural gas, increased wall echogenicity, bowel wall thickening or thinning, absent perfusion, free echogenic fluid) were present. The early diagnosis and monitoring of patients with NEC can be done with bowel sonography.

Ultrasound imaging has a role in diagnosis of deep seated sites of infection such as liver abscesses, perinephric collection of fluid, and suprapubic urinary bladder urine collection for culture.

SUMMARY—ROLE OF POINT-OF-CARE ULTRASOUND IN NEONATAL SEPSIS

S. No.	Imaging modality	Variable measured
1.	Functional echocardiography	Cardiac flows—right, left ventricles and SVC flows Presence of ductus arteriosus Presence of pulmonary hypertension IVC distensibility, collapsibility Ventricular diastolic function
2.	Cranial ultrasound	Echogenic sulci Ventriculitis Hydrocephalus/ventriculomegaly Intra-ventricular hemorrhage Post-ventricular hemorrhagic dilatation Echodensities in basal ganglia Abnormal resistive index Brain abscess
3.	Lung ultrasound	Lung consolidation Pleural effusion

Contd...

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S. No.	Imaging modality	Variable measured
4.	Bowel imaging	Bowel wall thickness, diameter Ileus Echogenicities Blood flows Intramural gas Ascites

KEY MESSAGE

- Point-of-care ultrasound imaging has become a useful tool in the hands of the clinician to determine the site of infection, the progress of sepsis, and prognosis/outcomes of neonates with sepsis.
- Ultrasound imaging can determine cardiac hemodynamics and aid the clinician in appropriate use of inotropes and vasopressors.

Figs 20A to C: Pleural effusion from the subcostal view:²⁴ (A) Normal view; (B) Large pleural effusion; (C) Fibrous septae

Figs 21A to C: Bowel wall perfusion—normal perfusion and hyperemia: (A) Normal perfusion; (B) Zebra pattern; (C) Y-shaped pattern

Figs 22A and B: (A) Echo shadows in the bowel wall; (B) Bowel wall edema—pneumatosis intestinalis

- Lung ultrasound has a significant role in urgent bedside evaluation of the neonatal lung disorders such as consolidation and pleural effusions.
- Bowel sonography can be helpful for the early diagnosis and monitoring of patients with NEC.

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