

Point of Care Neonatal Ultrasound — Head, Lung, Gut and Line Localization

CHANDRA RATH AND *PRADEEP SURYAWANSHI

From Departments of Neonatology, Royal North Shore Hospital, Pacific High way, St Leonards, NSW, Australia; and *Bharati Vidyapeeth University Medical college, Pune, Maharashtra, India.

Correspondence to: Dr Pradeep Suryawanshi, Professor and Head, Department of Neonatology, Bharati Vidyapeeth University Medical College, Pune-Satara Road, Pune, Maharashtra 411 043, India. drpradeepsuryawanshi@gmail.com

Received: July 25, 2015; Accepted: June 11, 2016.

Context: Knowledge and skills of heart, head, lung, gut and basic abdominal ultrasound is of immense utility to clinicians in their day-to-day patient management, and in acute events, in the absence of specialist service back-up. This review examines the potential role of clinician-performed ultrasound in the neonatal intensive care unit.

Evidence Acquisition: The bibliographic search of English-language literature was performed electronically using PubMed and EMBASE databases for the different topics we have covered under this review.

Results: Bedside head ultrasound can be used to identify and screen for intraventricular hemorrhage, periventricular leukomalacia and post-hemorrhagic ventricular dilatation. It is also a useful adjuvant tool in the evaluation of hypoxic ischemic encephalopathy. The relatively new lung ultrasound technique is useful in identifying transient tachypnea, pneumonia, pneumothorax, fluid overload and pleural effusion. Gut ultrasound is useful in identifying necrotizing enterocolitis and probably is better than X-ray in prognostication. Ultrasound is also useful in identifying vascular line positions without radiation exposure.

Main conclusions: Ultrasound performed by the clinician has an extensive role in the neonatal intensive care unit. Basic ultrasound knowledge of head, lung and gut is a useful supplement to clinical decision-making.

Keywords: Decision-making, Evaluation, Neonatal intensive care unit, Investigations.

Published online: July 01, 2016. PII:S097475591600016

Ultrasonography (USG) is no longer the exclusive domain of radiologists and cardiologists. With appropriate training, clinician performed ultrasound (CPU) is now practised widely in obstetrics, emergency medicine and adult intensive care, and is the standard practice in neonatology in many developed countries [1]. Cardiologists and radiologists undoubtedly have an indispensable role to play in clinical care, but it is unrealistic to expect 24-hour specialist cover, even in a resource-rich setting. Neonatal intensive care is a dynamic process, involving frequent evaluation, some in real time, which makes dependence on radiologist or cardiologist impractical. CPU has already proven its mettle in day-to-day management and the information obtained has often resulted in a management change [2]. In this review, we shall discuss the practical use of ultrasound for imaging the head, lung, and gut, and for vascular line localization. We also discuss the application to clinical decision making in resource-poor settings.

CRANIAL ULTRASONOGRAPHY

Role of cranial ultrasonography in neonatal intensive care unit (NICU) is for:

- Preterm infants for evaluation of germinal matrix

hemorrhage-intraventricular hemorrhage (GMH-IVH) and follow-up.

- Unexplained cardiac failure (to rule out vascular abnormalities).
- Hypoxic ischemic encephalopathy (HIE).
- Congenital malformations.
- Neonatal seizures.
- Evaluation of suspected subgaleal hematoma
- Evaluation of antenatally detected abnormalities.

Preterm infants, especially those less than 32 weeks gestation, are at risk for GMH-IVH, and ischemic white matter injuries. Late preterm infants who are monochorionic twins, small for gestational age (SGA), and or have experienced events such as chorioamnionitis, fetal distress, acidosis, difficult delivery, or hypotension are also at risk for ischemic white matter injury. If these abnormalities are detected early via ultrasound, follow-up and early intervention can be planned appropriately. Serial ultrasounds may be necessary to detect white matter lesions, which may not be evident until 2 to 4 weeks after the ischemic event. There may be significant changes in USG findings between the first and second scan, possibly changing medical management and prognosis. Serial USG

is also important in identifying significant post-hemorrhagic hydrocephalus for early intervention.

Procedure

Cranial USG is done through the anterior and posterior fontanelle, the mastoid foramen and poorly ossified parts of the temporal bone. The mastoid, temporal and posterior fontanelle views are supplementary to the absolutely necessary anterior fontanelle view. Usually 5-10 Hz 2D curved or linear array transducers are useful for cranial USG. Frequency of the probe may be increased for optimal visualization of superficial structures like subcortical white matter and venous sinuses; this will increase resolution at the expense of penetration. Similarly, lower frequency may be used for visualization of deeper structures in the posterior fossa. The transducers used should fit perfectly on the anterior fontanelle, as a large footprint makes the contact and image quality suboptimal, and small footprints reduce the diagnostic ability.

In this review, we shall focus on hemorrhage, parenchymal changes and hydrocephalous evaluation, which are most frequently encountered in day-to-day practice.

Germinal Matrix-Intraventricular Hemorrhage

GMH-IVH is one of the most common ultrasound findings in NICU. Studies performed in the 1980s suggested that >90% IVH cases in very low birth weight (VLBW) infants occurred within postnatal days 4 to 5 [3]. Premature infants are relatively resistant to hemorrhage after this period, irrespective of the gestational age (GA) because of the shutdown in angiogenesis, making the vessels resistant to rupture despite fluctuation in the cerebral blood flow [4]. A recently published review [5] which included studies from the antenatal steroid and surfactant era, concluded that 48% of cases of IVH occurred in the first 6 hours of life in VLBW infants, and suggested that early cranial USG may have prognostic, preventive and medicolegal implications. A small percentage of GMH-IVH may occur up to third week of life.

Observational studies from the 1990s showed that in the first two weeks of life, 12-51% of infants <1,500 grams or gestational age of <33 weeks had abnormalities on ultrasound out of which 6-20% were major (such as grades 3 and 4 IVH or bilateral cystic periventricular leukomalacia) [3]. More severe IVH occurs in more premature infants. Although the American Academy of Neurology and the Practice Committee of the Child Neurology Society [3] suggested screening all preterms <30 weeks due to the incidence of severe IVH [3], infants

up to 34 weeks are also at increased risk of GMH-IVH. In a recent study by Ballardini, *et al.* [6] in late preterm infants (33-36 weeks), intracranial lesions were found in 13% of the neonates when ultrasound was undertaken within day 7 of life. The risk factors for detecting intracranial abnormalities were head circumference less than the 3rd percentile, the need for ventilation or surfactant, low Apgar score at fifth minute, and neurological abnormalities. However, severe grades of IVH and extreme periventricular leukomalacia (PVL) are rare in this gestational age [6]. In another study, Bhat, *et al.* [7] detected abnormal cranial ultrasonography in 6.8% preterm (30-34 weeks) newborns and recommended screening in infants born between 30 and 34 weeks of gestational age. They also detected severe intracranial anomalies in 1.5% of neonates in this gestational age group; however, inclusion of less than 40% of the eligible neonates [8], born during the study period makes this data a little less meaningful. Vanderwalt, *et al.* [8] in a cost analysis study, concluded that cranial ultrasound screening of infants >32 weeks is not cost-effective.

There is no consensus for the optimal timing for cranial ultrasonography. Based on the above discussion, we propose a screening schedule for preterm neonates (**Table I**). The incidence of severe intracranial abnormalities is low in neonates with gestational age greater than 30 weeks, and the proposed schedule may not be very cost-effective in resource-poor settings. IVH is often asymptomatic but the likelihood of signs increase with the severity of hemorrhage. Possible clinical signs are: tense anterior fontanelle, pallor and associated drop in hematocrit, unresponsiveness; tonic seizures and decerebrate posturing; these should warrant immediate bedside cranial USG.

Approximately 50-75% of preterm survivors with predominantly grade IV IVH develop cerebral palsy, intellectual disability, and/or hydrocephalus [10,11]. A recent Australian report on neurodevelopmental outcomes of extremely preterm infants revealed that grade I-II IVH, even in the absence of white matter injury or other late ultrasound abnormalities, is associated with adverse neurodevelopmental outcomes [12], supporting the use of routine cranial USG to identify all silent GMH-IVH. A grading system developed by Papile and Burstein [13] is still widely used for prognostication where grade I is a bleeding confined to germinal matrix and looks as echogenic as choroid plexus in USG. The caudothalamic groove acts as a convenient landmark: echogenicity anterior to the groove represents blood as the choroid finishes at the groove. Grade II is grade I with intraventricular extension where blood can be seen as white bright spots/lines in the ventricles separate from

TABLE I PROPOSED CRANIAL ULTRASONOGRAPHY SCANNING PROTOCOL FOR PRETERM INFANTS

< 28 weeks or birth weight < 1000g or 28-31+6 weeks and/or birth weight < 1500 g on life support.	28-31+6 weeks or birth weight 1000-1500g without life support	32-34 weeks with risk factors: Monochorionic twins, head circumference < 3 rd centile, ventilation and/or surfactant need, fetal distress, acidosis, 5 minute APGAR score of < 6, or hypotension
6 hours of age	Day 3 to 1 week	Day 5 to 1 week and then as indicated
Day 3 to 1 week	4 weeks	
4 weeks	TAE or discharge	
Term age equivalent (TAE) or discharge whichever occurs first		

One week after any "new" sick event such as sepsis, hypotension, necrotizing enterocolitis, etc. (If near term after the 1 week scan then as required) [9]. In case of IVH other than GMH alone, weekly scans are indicated. Cranial USG anytime in case of clinical suspicion of IVH.

choroid plexus, grade III is ventricle dilatation because of excessive blood inside it, and grade IV is extension of hemorrhage in to the parenchyma. It must be remembered here that Papile classification was originally developed using CT scan but there have been reports of its use in cranial USG with accuracy [14].

Grade IV is interpreted as the result of an extension of the hemorrhage from the ventricle into the adjacent white matter. However, it is now postulated that large blood clots in the germinal matrix and ventricles impair the flow of blood from the medullary veins (which drain the cerebral white matter) into the terminal vein leading to venous infarction and possibly hemorrhagic infarction i.e., periventricular hemorrhagic infarct (PVHI). Besides this compression theory, ependymal trauma and inflammation as a possible cause has also been proposed. Therefore, PVHI is not a simple extension of germinal matrix hemorrhage into adjacent brain parenchyma as assumed in the Papile classification [15]. PVHI is always associated with an ipsilateral GM-IVH. When GM-IVH is bilateral, it usually is larger on the side ipsilateral to the PVHI. A scoring system has been proposed using parameters like the extent of PVHI, midline shift and unilateral or bilateral PVHI. This scoring helps in better prognostication, as there is a strikingly significant relationship between high PVHI score and the likelihood to withdraw care, the development of early neonatal seizures, and abnormal neuromotor examination at 12 and 30 months of age [16,17]. Grade I-III IVH are easy to identify on cranial USG; however, clinicians may occasionally face difficulties in differentiating PVL from PVHI as both lesions are initially echogenic with later cystic evolution. PVHI is an echodense lesion in the periventricular white matter which is unilateral or, if bilateral, obviously asymmetric. PVHI is also associated with a GMH-IVH lesion, which is usually ipsilateral or larger on the ipsilateral side. PVL develops in the first week of life as

bilateral echo density at the lateral border of the lateral ventricle with minimal or no IVH. PVHI usually evolves into a single or few relatively large cysts, which communicate with the lateral ventricle where as PVL evolves in to multiple tiny cysts, which do not communicate with lateral ventricle. Bass, *et al.* [18] could differentiate between PVHI and PVL in 77% of their study subjects with cranial USG while 11% had mixed lesions [18].

Once the diagnosis of GMH-IVH is made, we should look for cerebellar bleeding, as the external layer of the cerebellum is also a germinal zone. Bleeding in and around the cerebellum may lead to poor future neurodevelopmental outcome. Early detection with the help of cranial USG through the mastoid foramen is important for prognostication and appropriate counselling of the family. Though small punctate cerebellar hemorrhages may not be seen well with cranial USG as compared to MRI, it remains a useful bedside tool [19].

Post-hemorrhagic Ventricular Dilatation

The risk of developing post-hemorrhagic ventricular dilatation (PHVD) is considerable after a severe hemorrhage (grade III/IV). PHVD is defined as ventricular enlargement ≥ 97 th centile for gestational age (GA) [20], and is recognized in about one-third of infants with GMH-IVH. About 35% of neonates who develop PHVD require some form of intervention [21]. Evaluation of progressive PHVD with clinical parameters such as serial measurement of head circumference, tense fontanelle, sunset phenomena of the eyes are not as reliable as serial cranial USG [20,22,23]. It is hard to distinguish post-hemorrhagic ventriculomegaly from atrophic ventriculomegaly resulting from white matter loss. However, regardless of the mechanism, the extent of white matter loss has a direct correlation with the motor outcome [24]. The

measurements commonly used in clinical practice (**Fig. 1**) are accurate compared to MRI [25]. Ventricular index is one of the most commonly used measurements and the reference value correlates well for term neonates. However, it may not increase during early hydrocephalus and the reference values for preterm infants show variation because of less representation of this population in the reference curves [20,26-28]. Another commonly used measurement, the anterior horn width (AHW), has the advantage of identifying early hydrocephalus [27] with a minimal variation with change in gestational age [27,29]. However, a recent study by Sondhi, *et al.* [30] demonstrated an evident increase in size with ongoing maturity. Thalamo-occipital distance (TOD), which essentially measures the occipital horn length of the lateral ventricle, may be a useful measurement and sometimes represent the only site of ventricular dilatation [30]. Absence of increased TOD is an important negative finding. However, difficult visualization, considerable variation in reference curves, and the presence of isolated dilation of the occipital horn in normal preterm infants makes this measurement clinically less meaningful [29-31]. Other measurements like ventricular height and frontal horn ratio are less valuable in clinical practice as no reference curves are available. Measurement of the 3rd and 4th ventricle may assist in differentiating communicating and non-communicating hydrocephalus; however, the absence of quality reference curves, inter-observer variability, and difficulties in measurements are the main drawbacks [29,30].

Ventricular index and AHW are the most widely studied and used measurements in clinical practice.

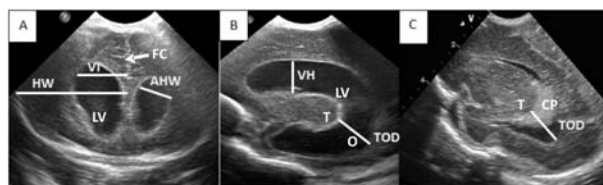


FIG. 1 Commonly used measurements used in evaluation of PHVD. A-Coronal plane Ventricular Index (VI)- Distance between the falx and the lateral wall of the anterior horn at the level of the third ventricle (4 mm above the 97th centile for GA is an indication for CSF drainage), FHR = VI/Hemispheric width (HW), AHW-Maximum diagonal width (values above 6mm significant). B- Sagittal plane TOD- Distance between the outermost point of the thalamus at its junction with the choroid plexus, to the outermost part of the occipital horn, Ventricular height (VH)- At the level of foramen of Monro. C- Sagittal plane-TOD in a non-dilated ventricle, LV- Lateral ventricle, T- Thalamus, CP- Choroid plexus, O- Occipital horn of the lateral ventricle, FC- Falx cerebri

Ventricular index greater by 4 mm of the 97th centile for gestational age is associated with a poor prognosis [21]. A normal AHW is less than 3 mm, with the 95th percentile curve reaching 2 mm at 36 weeks and 3 mm at 40 weeks. A size of more than 6 mm is considered abnormal. The implications of AHW between 3 and 5 mm is not clear. Cranial USG is useful in the identification of PVHD and should be undertaken at least twice weekly to identify progression; however interventional decisions are usually a combination of clinical findings, history and ultrasound findings. Indian data regarding these measurements are scarce [32,33].

Periventricular Leukomalacia

PVL, which occurs as a consequence of preterm brain ischemia and/or inflammation, is of great diagnostic importance because of its association with cerebral palsy and abnormal development. PVL usually occurs in preterm infants ≤ 32 weeks gestation as they have poorly vascularized white matter, which contains oligodendrocyte progenitors sensitive to ischemia and inflammation [34]. MRI has been reported to be a better modality than ultrasound in detecting white matter injury particularly in the diagnosis of punctate white matter lesion (PWML) and diffuse excessive high signal intensity [35]. However, serial USG has a definite role in evaluation of cystic PVL, a more severe form of white matter injury. The more extensive cysts tend to develop within 2-3 weeks following an insult, while the more localized cystic lesions may take as long as 3-6 weeks to develop [36]. Therefore, PVL diagnosed in the first week of life indicates an antenatal insult rather than a perinatal insult. Echogenicity in the brain equal to or greater than echogenicity in the choroid plexus, when persisting for more than 10-14 days, should alert the clinician about possible early PVL. Transient hyper-echoic lesions or periventricular halos might be seen in normal white matter of preterm infants. The pattern of distribution of PVL on ultrasound is typically dorsal and lateral to the external angles of the lateral ventricles. Any brain lesion, which causes brain parenchymal loss, may result in cyst formation. It has been suggested that PVHI and PVL can be differentiated by the location of the cysts. PVL has a predilection for periventricular arterial border zones, particularly in the region near the trigon of the lateral ventricles. PVHI is prominent more anteriorly with the lesion radiating from the periventricular region at the site of confluence of the medullary and terminal vein and assumes a triangular, fan-shaped appearance in the periventricular white matter [14]. The typical positions of various cystic lesions are depicted in **Fig. 2**. A classification for PVL has been suggested, though this is not widely accepted [37].

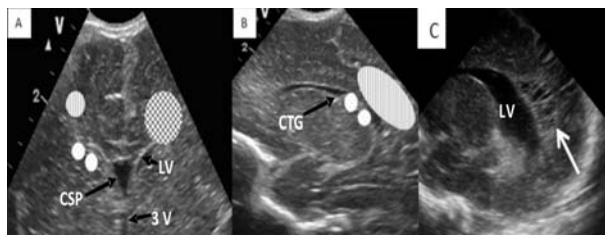


FIG. 2 A- Coronal view- Dotted area- Site for subependymal cyst, connatal cyst, Striped – Site for PVL, Chequerboard- Site for PVHI. B- Sagittal view- Striped- Site for PVL, Dotted area- site for choroid plexus cyst. C-White arrow showing cystic PVL. LV- Lateral ventricle, 3V-3rd Ventricle, CSP- Cavum septum pellucidum, CTG-Caudothalamic groove.

Doppler Evaluation

Doppler imaging of the anterior cerebral artery (ACA) and middle cerebral (MCA) is easily done through the anterior fontanelle in the sagittal plane and through the temporal window in the axial plane. The peak systolic velocity (PSV), end diastolic velocity (EDV), resistive index (RI) and pulsatility index (PI) are the most common measurements used for monitoring intracranial haemodynamics. Measuring PI is useful as it minimizes the effect of vessel angulation and correlates well with acute changes in intra-cerebral perfusion pressure [38]. Age-dependent reference values are available, and the normal range for the RI is 0.65 - 0.90. Values <0.5 or >0.9 are abnormal. An increase in diastolic flow results in a decrease in the RI, and conversely a decrease in diastolic flow results in an increase in the RI. Various factors can influence RI; for example, presence of a patent ductus arteriosus (PDA), scanning pressure on the anterior fontanelle, IVH, PVL, hydrocephalus, pneumothorax and low arterial carbon dioxide can increase the RI. Similarly RI is decreased in asphyxia, vascular malformation, tachycardia and decreased cardiac output [39]. RI <0.5 in asphyxiated newborns in the first few days of life is associated with both immediate and long term poor outcome [40-45]. Unfortunately, some full-term neonates with significant asphyxia may not show this decreased RI and may instead have a normal or increased RI which may be due to a relative decrease in diastolic flow velocity. This decrease in diastolic flow velocity may be because of the presence of a significant PDA, myocardial dysfunction (such as in transient myocardial ischemia), or hypervolemia. Mean cerebral blood flow is mainly determined from the diastolic flow. As intracranial pressure (ICP) rises, the arterial flow is more affected during diastole than during systole, resulting in an increase in RI as happens in hydrocephalus [46]. In individual infants, a tendency towards a correlation between ICP and flow variables was found when studied

longitudinally [47]. However, it is doubtful whether the RI can be used as an indicator for the timing of intervention, because it can vary widely between individual preterm infants and accuracy subjected to presence of other conditions, that may influence cerebral blood flow.

Cranial USG has many other applications in term and preterm infants which is beyond the scope of this review. It is excellent for the detection of IVH, ventriculomegaly, perforator stroke, sinovenous thrombosis and cystic PVL, but MRI is superior in detecting cortical abnormalities, posterior fossa lesions, subtler white matter injury, early watershed infarct events, microabscesses and involvement of posterior limb of internal capsule. Reviews of the studies directly comparing cranial USG with MRI with cerebral palsy as the outcome show that utility of MRI tends to be similar or higher compared with cranial USG [48,49]. In a recently published study, serial cranial USG seems highly effective in diagnosing all common preterm brain injuries, but may miss cerebellar abnormalities [50]. However, it will be interesting to see neurodevelopmental prediction with early MRI in few of the upcoming studies.

LUNG ULTRASOUND

Clinical signs and radiographs are routinely used to diagnose neonatal lung disease albeit they have low specificity and sensitivity for many common clinical conditions. Lung ultrasound is being increasingly used in Neonatal Intensive Care Unit (NICU) and adult ICU because of its high sensitivity and specificity [51]. It is easy to learn and can be performed with a basic ultrasound machine. Lung and pleura being superficial structures, USG requires a high-frequency linear array probe (>7.5 MHz). Micro convex probe may be used, however, linear probe displays a wider field. A basic USG setting with 2D, M mode and occasional color Doppler is all that is required to do lung ultrasound. Normal USG of lung shows 'A lines', which are parallel to straight solid pleural lines, are reverberation artefacts, and are equidistant from each other. On the other hand B-lines occur when sound waves pass through the pleural line encountering a mixture of air and water as in pulmonary oedema. These are discrete laser-like vertical hyper echogenic lines that arise from the pleural line, extend to the bottom of the screen without fading, and move synchronously with lung sliding (**Fig. 3**). The pleural line slides from side to side with respiration and represents movement of the pleural surface with the respiratory cycle. This sign is known as sliding sign, a normal lung feature. Lung sliding can also be observed using time motion mode (M mode) where the fixed superficial chest wall structures give rise to an appearance of water and the

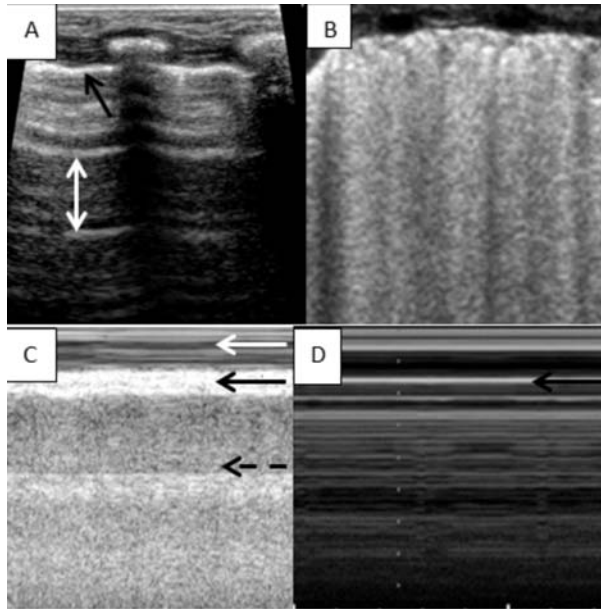


FIG. 3 (A) Normal lung ultrasound- Horizontal A lines shown by the white arrow equidistance from each other and the pleural line, black arrow showing the pleural line (PL). (B) Multiple vertical lines starting from the PL and almost coalescing with each other giving a white lung appearance. (C) Normal lung M mode- Seashore sign, black arrow denoting PL, white arrow showing the chest wall looking like water and the broken black arrow showing lung parenchyma looking like a sandy beach. (D) Stratosphere sign as seen in pneumothorax, there is no water and sandy part, it all looks like water.

constantly moving underlying lung gives rise to a sandy appearance known as seashore sign (**Fig. 3**). The 'lung pulse' refers to the rhythmic movement of the pleura in synchrony with the cardiac rhythm. As the heart beats the movement of the heart is transmitted through the medium of the lung, which is demonstrated in M-mode as a regular motion artefact through the seashore pattern to the level of the pleura. In normal well-aerated lung, the 'lung pulse' is not present, as lung sliding becomes dominant and resistant to cardiac vibrations. The lung pulse is easily identified when the baby is not breathing.

Pneumothorax

USG is an invaluable tool for the assessment of pneumothorax, with accuracy approaching CT, and far exceeding plain radiography in adults [52]. It is of immense value in emergencies such as tension pneumothorax, as it is readily available at the bedside and can be done in less than a minute. Features of pneumothorax such as the absence of lung sliding, presence of lung point (A point where seashore sign changes in to stratosphere sign), presence of stratosphere sign on M mode (**Fig. 3**), absence of B-lines and absence

of lung pulse are easy to identify with minimal training. In Stratosphere sign parallel horizontal lines above and below the pleural line is noted and it resembles a barcode. In contrast to the seashore sign which is a normal lung sign, in stratosphere sign the grainy shore below the pleural line is not seen (which is due to the movement of the lungs with respiration), rather only sea (parallel lines) is noted and this denotes a static lung which is not moving with respiration because of pneumothorax (**Fig. 3**). Though studies in neonates are lacking, Lichtenstein, *et al.* [53] from his experience in NICU suggested that neonatal signs are no different from adult lung signs.

Pneumonia

Lung ultrasound is a clinically useful tool in diagnosing pneumonia; however, consolidation that does not reach the pleura cannot be visualised. In adults, lung consolidation extends to the pleura in 98.5% of cases and can be seen on USG [52]. Lung mass is smaller in the newborn and extension to the pleura may be much more frequent. Coarse and/or irregular disrupted pleural line, hepatisation of the lung tissue (echogenicity similar to liver), hyperechoic area of varying size and shape in the same lung field, irregular margin around consolidation, presence of dynamic air-bronchogram, disappearance of lung sliding, mild pleural effusion and presence of lung pulse are few of the features which can be identified in pneumonia. The international consensus committee on lung ultrasound agreed that there is strong evidence that USG is an accurate tool in diagnosing lung consolidation when compared with chest radiography in pediatric age group [54]. Some neonatal studies have also shown USG to be a useful tool in recognizing neonatal pneumonia with good specificity and sensitivity [55,56].

Pleural Effusion

Opacities detected by conventional radiography can be differentiated as consolidation or effusion only by an ultrasound scan. For pleural effusions, USG has a sensitivity of 93% and specificity of 97% [57]. USG can also be used to differentiate between transudate and exudate [54]. Visualization of internal echoes, mobile particles or septa, is highly suggestive of exudate; however, in case of an anechoic effusion, the only way to differentiate between transudate and exudate is to use thoracoentesis.

Extravascular Fluid

Presence of vertical 'B lines' represents extravascular fluid in lungs. (**Fig. 3**). B lines can be used to monitor cardiac failure (systolic and diastolic), iatrogenic fluid overload (a sudden change from A to B line), or preload/afterload reduction therapy. However, B lines and white

lung in neonates should be considered in the clinical context of the disease. B lines and white lung can also be seen in respiratory distress syndrome (RDS), transient tachypnea of the newborn (TTN), consolidation and atelectasis of any cause, meconium aspiration syndrome and broncho-pulmonary dysplasia.

Respiratory Distress Syndrome and Transient Tachypnea of the Newborn

USG is a useful tool in the management of RDS and TTN with good interobserver agreement. In TTN, very compact B lines in the inferior pulmonary fields and not so compact B lines in the superior lung field gives a characteristic sign called the double lung point, a sign with which we may use to differentiate it from RDS. The double lung point sign is also useful in management and prognosis, particularly in a resource-poor setting. Co-existence of lung consolidation, abnormal pleural line (thickness of $>0.5\text{mm}$ or blurred), bilateral white lung and disappearance of A lines are constant ultrasonography features of RDS with a specificity and sensitivity of 100%. Other features like pleural effusion, lung pulse and uniform bilateral involvement are infrequent associations. The most important indicator of RDS is consolidation, which is seen in all RDS patients but the extent and scope of consolidation varies with severity of RDS. Consolidation in moderate RDS is sub pleural and focal in nature whereas consolidation in severe RDS is more widespread and deep. Similarly lung pulse was present in all grade 3 and 4 RDS while it was absent in all grade 2 RDS [58]. In term and near term infants, USG at 1-2 hours of life has been shown to anticipate the need for respiratory support and severe respiratory distress with 100% specificity and 77.7% sensitivity [59]. In a recent study, lung ultrasound predicted need for intubation after 2 hours of life in preterm babies with a positive predictive value of 100%, and negative predictive value of 94.7% [60]. Another recent study predicted the need for Surfactant administration on the basis of a scoring system which consists of oxygenation indices and lung ultrasound, with a sensitivity and specificity of 100% and 61% respectively [61]. The basic principle in all these studies is the abundance of B lines. A higher number of B lines appear as whiter lungs that need more support compared to a finding with more A lines.

USG is not yet completely ready to replace X-ray in neonatology. Few non-specific signs, paucity of neonatal research and publications are the drawbacks. However, this technology undoubtedly has the potential to replace X-ray as the most useful bedside lung disease diagnostic tool.

NECROTIZING ENTEROCOLITIS (NEC)

Identification and management of NEC is currently based on recommendations from the modified Bell's criteria [62]. Abdominal X-ray is the cornerstone in diagnosis and is able to detect bowel distension, bowel wall thickness, pneumatosis intestinalis, portal venous gas and free abdominal air. USG provides additional information about gut viability and free fluid in the abdomen. An 8-15 MHz linear probe should be used for bowel loop ultrasound.

Data on normal thickness of the bowel in preterm neonates is scarce; we suggest a thickness of 1.2 to 2 mm from personal experience. Normal term bowel wall thickness has been described as 1.1 to 2.6 mm. A normal bowel perfusion is 1–9 colour doppler signal dots per cm^2 (mean 3.8) in a setting of the lowest possible pulse repetition frequency and the highest Doppler gain settings without flash artefacts. The velocity was set at 0.029–0.11 m/sec [63]. Normal bowel wall is smooth with peristalsis.

A bowel wall thickness $>2\text{ mm}$ should be considered suspicious and conversely; a thickness $<1.0\text{ mm}$ indicates an abnormal thinning resulting from ischemia or necrosis. Increased bowel perfusion may present in different patterns such as ring-shape, Y-shaped and zebra-shaped. Absent bowel perfusion can be assumed when no color signal is detected at the slowest possible velocity (0.029 m/sec) and suggests a complete bowel wall necrosis with 100% sensitivity [63]. Intramural gas, a common finding though not pathognomonic of NEC, can be identified as highly echogenic dots in the bowel wall and may involve the whole circumference, in which case it is called the "circle sign" (**Fig. 4**). Intramural gas must be differentiated from intraluminal gas, which moves with compression of the abdomen with the ultrasound probe. The amount of intramural gas present does not always relate to the clinical severity of NEC and its disappearance does not correlate with clinical improvement [64]. In the absence of NEC, the commonest cause of portal venous gas is the passage

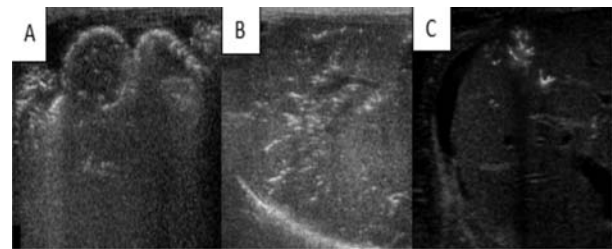


FIG. 4 (A) Extensive pneumatosis intestinalis (white dots) white arrow- Bowel wall, (B) Extensive portal venous air in the liver USG (White dots), (C) Portal venous gas (White dots).

small amounts of gas through an umbilical venous catheter. Neither is the presence portal venous gas fatal, nor does its disappearance always herald clinical improvement. Portal venous gas has been reported in only 30% of the neonates with NEC, and is detected by ultrasound much earlier than it appears on X-ray [65,66]. Free abdominal gas secondary to perforation can be seen as a bright white hyper echogenicity between the diaphragm and liver which moves with abdominal compression. Detection of intra peritoneal fluid and or a mass may help in diagnosing perforated NEC. In a study by Silva, *et al.* [67], 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, there was a sensitivity of 0.82 and a specificity of 0.78 for poor outcome.

USG for NEC is not without drawbacks; inter-observer variability, large amount of bowel gas and tender unstable abdomen may hamper good USG evaluation. However, USG has an obvious advantage over routine X-ray in diagnosis and prognostication of NEC [68], especially in neonates with clinical deterioration without X-ray changes.

LINE-LOCALIZATION

Though central line placement in NICU is a necessity, it is not without complication. Identification of central line tip location may help in reducing the complications, and USG is one of the easiest bedside modality to do so. A recently published review [69] suggested considering USG as a potential alternative to X-ray in central line tip location in neonates. Two recent studies [70,71] could identify around 25% of the cases with abnormal tip position, which were reported to be normal in X-ray reporting. Ultrasound-guided umbilical catheter placement is a faster method to place catheters requiring fewer manipulations and X-rays when compared with conventional catheter placement [72].

TRAINING AND MEDICO-LEGAL IMPLICATIONS

It is important to have a structured training program for clinicians in order to make them ultrasound literate. Few developed countries in the world have a structured training program for bedside echocardiography and fewer have it for bedside cranial ultrasound [1,73]. Most of these training programs require the clinician to undertake 75-250 studies under the guidance of the experts in an accredited center, and which might take anytime between 6 months to 24 months to complete. The course also includes hands on basic, advanced training courses and an online physics course.

However, issues like different clinical needs, misdiagnosis, medicolegal liability and financial return for examination, need further discussion. Clinical need is a pertinent issue in the Indian scenario, as there are very few hospitals around the country catering to newborns that have in-house radiological and pediatric cardiology services. In reality, 24-hour presence of specialists to provide ultrasound services in the NICU is not achievable. It is here where clinician-performed ultrasound can be handy. However, the risk of misdiagnosis is a real and important concern, and some of this can be resolved by guidelines about when consultative referral should be mandatory. The other important step, which can reduce misdiagnosis, is structured training and accreditation. There is a medicolegal vacuum as far as clinician-performed ultrasound is concerned. If a registered medical practitioner with six months training or one year experience in sonography or a gynecologist with experience are allowed to do ultrasound, we do not see any reason why adequately trained clinicians cannot do bedside USG for better patient management. The motivation to acquire point of care ultrasound skill should be to assist in clinical decision-making and clinicians should be careful in practicing outside the limits of their skills. Neonatologists with adequate training should be able to report his/her USG findings in the progress sheet for day-to-day clinical decision-making. It is important to mention in the report, whether clinician or an imaging specialist performs bedside ultrasound.

CONCLUSION

Head, lung and abdomen ultrasound are useful bedside clinical tools, which can be used as frequently as required without the risk of radiation exposure. Cranial USG is most commonly used to identify IVH, PVHD and cystic PVL with good efficacy. Lung ultrasound is useful in identifying pneumothorax, pleural effusion, pneumonia and plays a supportive role in the management of RDS. Bedside USG for NEC should be supplementary to usual management. Bedside USG has a definite role in line localization. Use of bedside USG in neonatology is on the rise with frequent new utility additions like endotracheal tube tip localization and is becoming an obligatory screening and diagnostic tool.

It must be emphasized here that clinician-performed USG is not here to replace the role of pediatric cardiologists and radiologists in neonatal practice. However, ultrasound-literate clinicians should be able to do USG in an acute clinical setting, document it and do appropriate intervention in absence of specialist expertise.

Contributors: Both authors contributed to literature search, manuscript writing and its approval.

Funding: None; *Competing interest:* None stated.

REFERENCES

1. Evans N, Gournay V, Cabanas F, Kluckow M, Leone T, Groves A, *et al.* Point-of-care ultrasound in the neonatal intensive care unit: international perspectives. *2011*;16:61-8.
2. El-Khuffash A, Herbozo C, Jain A, Lapointe A, McNamara PJ. Targeted neonatal echocardiography (TnECHO) service in a Canadian neonatal intensive care unit: a 4-year experience. *J Perinatol.* 2013;33:687-90.
3. Ment LR, Bada HS, Barnes P, Grant PE, Hirtz D, Papile LA, *et al.* Practice parameter: neuroimaging of the neonate: report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology.* 2002;58:1726-38.
4. Praveen B. Pathogenesis and prevention of intraventricular hemorrhage. *Clin Perinatol.* 2014;41:47-67.
5. Al-Abdi SY, Al-Aamri MA. A systematic review and meta-analysis of the timing of early intraventricular hemorrhage in preterm neonates: Clinical and research implications. *J Clin Neonatol.* 2014;3:76-88.
6. Ballardini E, Tarocco A, Baldan A, (Antoniazzi E, Garani G, Borgna-Pignatti C. Universal cranial ultrasound screening in preterm infants with gestational age 33-36 weeks. A retrospective analysis of 724 newborns. *Pediatr Neurol.* 2014;51:790-4.
7. Bhat V, Karam M, Saslow J, Taylor H, Pyon K, Kemble N, *et al.* Utility of performing routine head ultrasound in preterm infants with gestational age 30-34 weeks. *J Matern Fetal Neonatal Med.* 2012;25:116-9.
8. Van der walt C, Vazzalwar R, Schweig L, Donovan R. IVH Screening By Cranial Ultrasound for All Preterm Infants ≥ 30 Weeks Is Not Cost Effective. *In: Proceedings of the AAP Experience National Conference & Exhibition: Perinatal Pediatrics Scientific Posters Presentations; 2013 October 25; Orlando. Florida; 2013.* Available from: https://aap.confex.com/aap/2013/webprogram/Paper_21331.html. Accessed July 15, 2015.
9. Andre P, Thebaud B, Delavaucoupet J, Zupan V, Blanc N, d'Allest AM, *et al.* Late-onset cystic periventricular leukomalacia in premature infants: a threat until term. *Am J Perinatol.* 2001;18:79-86.
10. Sherlock RL, Anderson PJ, Doyle LW. Neurodevelopmental sequelae of intraventricular haemorrhage at 8 years of age in a regional cohort of ELBW/very preterm infants. *Early Hum Dev.* 2005;81:909-16.
11. Luu TM, Ment LR, Schneider KC, Katz KH, Alan WC, Vohr BR. Lasting effects of preterm birth and neonatal brain hemorrhage at 12 years of age. *Pediatrics* 2009;123:1037-44.
12. Bolisetty S, Dhawan A, Abdel-Latif M, Bajuk B, Stack J, Lui K. Intraventricular hemorrhage and neurodevelopmental outcomes in extreme preterm infants. *Pediatrics.* 2014;133:55-62.
13. Burstein J, Papile LA, Burstein R. Intraventricular hemorrhage and hydrocephalus in premature newborns: A prospective study with CT. *Am J Roentgenol.* 1979;132:631-5.
14. Khan IA, Wahab S, Khan RA, Ullah E, Ali M. Neonatal intracranial ischemia and hemorrhage: Role of cranial sonography and CT scanning. *J Korean Neurosurg Soc.* 2010;47:89-94.
15. Volpe JJ. Intracranial Hemorrhage. *In: Volpe JJ. Neurology of the Newborn.* 5th Ed. Saunders; Philadelphia. 2008;11:517-88.
16. Bassan H, Benson CB, Limperopoulos C, Feldman HA, Ringer SA, Veracruz E, *et al.* Ultrasonographic features and severity scoring of periventricular hemorrhagic infarction in relation to risk factors and outcome. *Pediatrics.* 2006;117:2111-8.
17. Bassan H, Limperopoulos C, Visconti K, Mayer DA, Feldman HA, Avery L, *et al.* Neurodevelopmental outcome in survivors of periventricular hemorrhagic infarction. *Pediatrics.* 2007;120:785-92.
18. Bass WT, Jones MA, White LE, Montgomery TR, Karłowicz MG. Ultrasonographic differential diagnosis and neurodevelopmental outcome of cerebral white matter lesions in premature infants. *J Perinatol.* 199;19:330-6.
19. Steggerda SJ, Leijser LM, Wiggers-de Bruïne FT, van der Grond J, Walther FJ, van Wezel-Meijler G. Cerebellar injury in preterm infants: incidence and findings on US and MR images. *Radiology.* 2009;252:190-9.
20. Levene MI. Measurement of the growth of the lateral ventricles in preterm infants with real-time ultrasound. *Arch Dis Child.* 1981;56:900-4.
21. De Vries LS, Liem KD, van Dijk K, Smit BJ, Sie L, Rademaker KJ, *et al.* Early versus late treatment of posthaemorrhagic ventricular dilatation: results of a retrospective study from five neonatal intensive care units in The Netherlands. *Acta Paediatr.* 2002;91:212-7.
22. Ingram MC, Huguenard AL, Miller BA, Chern JJ. Poor correlation between head circumference and cranial ultrasound findings in premature infants with intraventricular hemorrhage. *J Neurosurg Pediatr.* 2014;14:184-9.
23. Muller WD, Urlesberger B. Correlation of ventricular size and head circumference after severe intra-periventricular haemorrhage in preterm infants. *Childs Nerv Syst.* 1992;8:33-5.
24. Brouwer A, Groenendaal F, van Haastert I, Rademaker K, Hanlo P, de Vries LS. Neurodevelopmental outcome of preterm infants with severe intraventricular hemorrhage and therapy for post-hemorrhagic ventricular dilatation. *J Pediatr* 2008;152:648-54.
25. Leijser LM, Srinivasan L, Rutherford MA, Counsell SJ, Allsop JM, Cowan FM. Structural linear measurements in the newborn brain: accuracy of cranial ultrasound compared to MRI. *Pediatr Radiol.* 2007;37:640-8.
26. Grasby DC, Esterman A, Marshall P. Ultrasound grading of cerebral ventricular dilatation in preterm neonates. *J Paediatr Child Health.* 2003;39:86-90.
27. Liao MF, Chaou WT, Tsao LY, Nishida H, Sakanoue M. Ultrasound measurement of the ventricular size in newborn infants. *Brain Dev.* 1986;8:262-8.
28. Brouwer MJ, de Vries LS, Groenendaal F, Koopman C, Pistorius LR, Mulder EJJ, *et al.* New reference values for

- the neonatal cerebral ventricles. *Radiology*. 2012;262:224-33.
29. Davies MW, Swaminathan M, Chuang SL, Betheras FR. Reference ranges for the linear dimensions of the intracranial ventricles in preterm neonates. *Arch Dis Child Fetal Neonatal Ed*. 2000;82:F218-23.
 30. Sondhi V, Gupta G, Gupta PK, Patnaik SK, Tshering K. Establishment of nomograms and reference ranges for intracranial ventricular dimensions and ventriculo-hemispheric ratio in newborns by ultrasonography. *Acta Paediatr*. 2008;97:738-44.
 31. Reeder JD, Kaude JV, Setzer ES. The occipital horn of the lateral ventricles in premature infants. An ultrasonographic study. *Eur J Radiol*. 1983;3:148-50.
 32. Chowdhary V, Culati P, Arora S, Thirupuram S. Cranial sonography in preterm infants. *Indian Pediatr*. 1992;27:411-5.
 33. Soni JP, Gupta BD, Soni M, Singh RN, Purohit NN, Gupta M, *et al*. Normal parameters of ventricular system in healthy infants. *Indian Pediatr*. 1995;32:549-55.
 34. Blumenthal I. Periventricular leucomalacia: A review. *Eur J Pediatr*. 2004;163:435-42.
 35. Hart AR, Whitby EW, Griffiths PD, Smith MF. Magnetic resonance imaging and developmental outcome following preterm birth: review of current evidence. *Dev Med and Child Neurol*. 2008;50:655-63.
 36. De Vries LS, van Haastert IL, Rademaker KJ, Koopman C, Groe-nendaal F. Ultrasound abnormalities preceding cerebral palsy in high-risk preterm infants. *J Pediatr*. 2004;144:815-20.
 37. De Vries LS, Eken P, Dubowitz LM. The spectrum of leukomalacia using cranial ultrasound. *Behav Brain Res*. 1992;49:1-6.
 38. Seibert JJ, McCowan TC, Chaddock WM, Adametz JR, Glasier CM, Williamson SL, *et al*. Duplex pulsed doppler US versus intracranial pressure in the neonate. Clinical and experimental studies. *Radiology*. 1989;171:155-60.
 39. Bulas DI. Transcranial doppler: Applications in neonates and children. *Ultrasound Clin*. 2009;4:533-51.
 40. Liu J, Cao HY, Huang XH, Wang Q. The pattern and early diagnostic value of Doppler ultrasound for neonatal hypoxic-ischemic encephalopathy. *J Trop Pediatr*. 2007;53:351-4.
 41. Nishimaki S, Iwasaki S, Minamisawa S, Seki K, Yokota S. Blood flow velocities in the anterior cerebral artery and basilar artery in asphyxiated infants. *J Ultrasound Med*. 2008;27:955-60.
 42. Argollo N, Lessa I, Ribeiro S. Cranial Doppler resistance index measurement in preterm newborns with cerebral white matter lesion. *J Pediatr (Rio J)*. 2006;82:221-6.
 43. Ilves P, Lintrop M, Metsvaht T, Vaher U, Talvik T. Cerebral blood-flow velocities in predicting outcome of asphyxiated newborn infants. *Acta Paediatr* 2004;93:523-8.
 44. Kirimi E, Tuncer O, Atas B, Sakarya ME, Ceylan A. Clinical value of color doppler ultrasonography measurements of full-term newborns with perinatal asphyxia and hypoxic ischemic encephalopathy in the first 12 hours of life and long-term prognosis. *Tohoku J Exp Med*. 2002;197:27-33.
 45. Ilves P, Talvik R, Talvik T. Changes in doppler ultrasonography in asphyxiated term infants with hypoxic-ischaemic encephalopathy. *Acta Paediatr*. 1998;87:680-4.
 46. Mackamee LR, Gonzales JI, Chance GW. Cerebral blood flow velocity profiles in intraventricular haemorrhage progressing to hydrocephalus. *Pediatr Res*. 1998;43:224A.
 47. Maertzdorf WJ, Vles JSH, Beuls E, Mulder ALM, Blanco CE. Intracranial pressure and cerebral blood flow velocity in preterm infants with post-haemorrhagic ventricular dilatation. *Arch Dis Child Fetal Neonatal Ed*. 2002;87:3 F185-8.
 48. Soo HK, Lana V, Laura RM, Petra SH. The role of neuroimaging in predicting neurodevelopmental outcomes of preterm neonates. *Clin Perinatol*. 2014;41:257-83.
 49. deVries LS, Benders MJ, Groenendaal F. Imaging the premature brain: ultra- sound or MRI? *Neuroradiology*. 2013;55:13-22.
 50. Plaisier A, (Raets MMA, Ecury-(Goossen GM, Govaert P, Feijen-Roon M, Reiss IK, *et al*. Serial cranial ultrasonography or early MRI for detecting preterm brain injury? *Arch Dis Child Fetal Neonatal Ed*. 2015;100:F293-F300.
 51. Lichtenstein DA, Mauriat P. Lung ultrasound in the critically ill neonate. *Curr Pediatr Rev*. 2012;8:217-23.
 52. Lichtenstein D. Lung ultrasound in the critically ill. *Clin Intensive Care*. 2005;16:79-87.
 53. Lichtenstein DA. Ultrasound examination of the lungs in the intensive care unit. *Pediatr Crit Care Med*. 2009;10:693-8.
 54. Volpicelli G, Elbarbary M, Blaivas M, Lichtenstein DA, Mathis G, Kirpatrick AW, *et al*. International Liaison Committee on Lung Ultrasound (ILC-LUS) for International Consensus Conference on Lung Ultrasound (ICC- LUS). International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med*. 2012;38:577-91.
 55. Hadeel M, Seif El Dien, Dalia AK ElLatif A. The value of bedside Lung Ultrasonography in diagnosis of neonatal pneumonia. *Egyptian J Radiol Nuclear Med*. 2013;44:339-47.
 56. Liu J, Liu F, Liu Y, Wang HW, Feng ZC. Lung Ultrasonography for the diagnosis of severe neonatal pneumonia. *Chest*. 2014;146:383-8.
 57. Lichtenstein D, Goldstein I, Mourgeon E, Cluzel P, Grenier P, Rouby JJ. Comparative diagnostic performances of auscultation, chest radiography and lung ultrasonography in ARDS. *Anesthesiology*. 2004;100:9-15.
 58. Liu J, Cao HI, Wang HW, Kong XY. Role of lung ultrasound in diagnosis of respiratory syndrome in newborn infants. *Iran J Pediatr*. 2015;25:e323.
 59. Raimondi F, Migliaro F, Sodano A, Umbaldo A, Romano A, Vallone G, *et al*. Can neonatal lung ultrasound monitor fluid clearance and predict the need of respiratory support? *Crit Care*. 2012;16:R220.
 60. Raimondi F, Migliaro F, Sodano A, Ferrara T, Lama S, Vallone G, *et al*. Use of neonatal chest ultrasound to predict noninvasive ventilation failure. *Pediatrics*. 2014;134:e1089-94.

61. Brat R, Yousef N, Klifa R, Reynaud S, Aguilera SS, De Luca D. Lung ultrasonography score to evaluate oxygenation and surfactant need in neonates treated with continuous positive airway pressure. *JAMA Pediatr.* 2015;169:e151797.
 62. Walsh MC, Kliegman RM. Necrotizing enterocolitis: treatment based on staging criteria. *Pediatr Clin North Am.* 1986;33:179-201.
 63. Faingold R, Daneman A, Tomlinson G, Babyn PS, Manson DE, Mohanta A, *et al.* Necrotizing enterocolitis: assessment of bowel viability with color doppler US. *Radiology.* 2005;235:587-94.
 64. Leonidas JC, Krasna IH, Fox HA, Broder MS. Peritoneal fluid in necrotizing enterocolitis: a radiologic sign of clinical deterioration. *J Pediatr.* 1973;82:672-5.
 65. Kim WY, Kim WS, Kim IO, Kwon TH, Chang W, Lee EK, *et al.* Sonographic evaluation of neonates with early-stage necrotizing enterocolitis. *Pediatr Radiol.* 2005;35:1056-61.
 66. Kirks DR, O'Byrne SA. The value of the lateral abdominal roentgenogram in the diagnosis of neonatal hepatic portal venous gas (HPVG). *Am J Roentgenol Radium Ther Nucl Med.* 1974;122:153-8.
 67. Silva CT, Danemann A, Navarro OM, Moore AM, Moineddin R, Gerstle JT, *et al.* Correlation of sonographic findings and outcome in necrotizing enterocolitis. *Pediatr Radiol.* 2007;37:274-82.
 68. Garbi Gautel A, Brevaut Malaty V, Panual M, Michel F, Merrot T, Gire C. Prognostic value of abdominal sonography in necrotizing enterocolitis of premature infants born before 33 weeks gestational age. *J Pediatr Surg.* 2014;49:508-13.
 69. Perin G, Scarpa MG. Defining central venous line position in children: tips for the tip. *J Vasc Access.* 2015;16:77-86.
 70. Jain A, McNamara PJ, Ng E, El-Khuffash A. The use of targeted neonatal echocardiography to confirm placement of periph-erally inserted central catheters in neonates. *Am J Perinatol.* 2012;29:101-6.
 71. Tauzin L, Sigur N, Joubert C, Parra J, Hassid S, Moulies ME. Echocardiography allows more accurate placement of periph-erally inserted central catheters in low birthweight infants. *Acta Paediatr.* 2013;102:703-6.
 72. Fleming SE, Kim JH. Ultrasound-guided umbilical catheter insertion in neonates. *J Perinatol.* 2011;31:344-9.
 73. Stanojevic M. Training of ultrasound in neonatology: Global or local? *Donald School J Ultrasound Obstet Gynecol.* 2013;7:338-45.
-