

Impact of acute cardiovascular collapse on cerebral electrical activity: importance of heart-brain interaction

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Abstract

A preterm infant experienced pericardial tamponade due to a deep central line. During the event the patient was on neurological monitoring, amplitude integrated electroencephalogram (aEEG), which demonstrated abnormal baseline activity during the point of cardiovascular collapse with improvement once intervention occurred with pericardiocentesis. The case highlights the value of neurological monitoring in acute hemodynamic instability.

Keywords

Pericardial effusion, low cardiac output state, PICC line, amplitude integrated electroencephalogram (aEEG), tamponade, neonate.

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Case

A male 33 week infant developed abdominal distension after birth. An exploratory laparotomy identified volvulus. A segment of ischemic bowel was resected, and a subclavian venous line was placed.

On post-operative day 1 the central line was noted to be in the right atrium and pulled back by 1 cm (**Fig. 1**). The patient was also placed on amplitude integrated electroencephalography (aEEG) due to altered level of consciousness, which demonstrated discontinuous normal voltage appropriate for gestation (**Fig. 2**). Sixteen hours later, the patient developed mottled skin, low pulse pressure, muffled heart sounds, hypotension,

elevated lactate and metabolic acidosis. The aEEG now demonstrated a burst suppression pattern. An emergent echocardiogram demonstrated a large pericardial effusion (**Fig. 3**). Bedside pericardiocentesis was performed after which there was immediate normalization of vital signs. Vital signs prior to, during and following the episode of pericardial tamponade are presented in **Tab. 1**. The aEEG returned to a normal pattern within 30 minutes (**Fig. 2**). There were no seizures and the neurological examination normalized.

This case demonstrates the impact of an acute drop in cardiac output on cerebral background electrical activity, the importance of longitudinal neurological monitoring and value of immediate access to bedside echocardiography in the NICU.

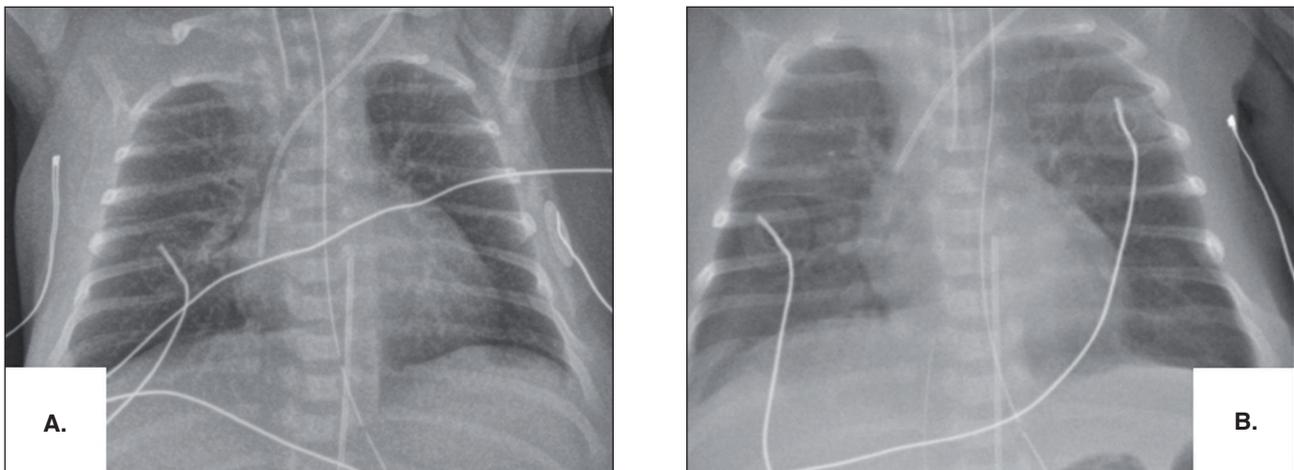


Figure 1. CXR during acute decompensation (**A**) and following pericardiocentesis (**B**).

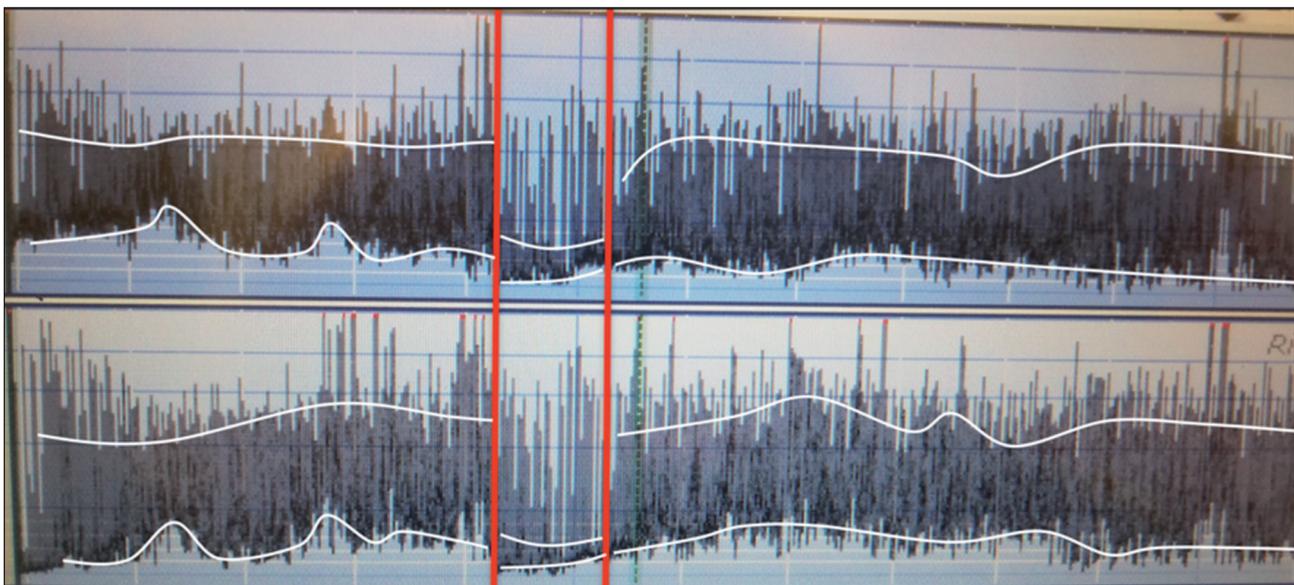


Figure 2. Discontinuous normal voltage with burst suppression (between red lines) seen on amplitude integrated electroencephalogram; the white lines denote the upper and lower margins.

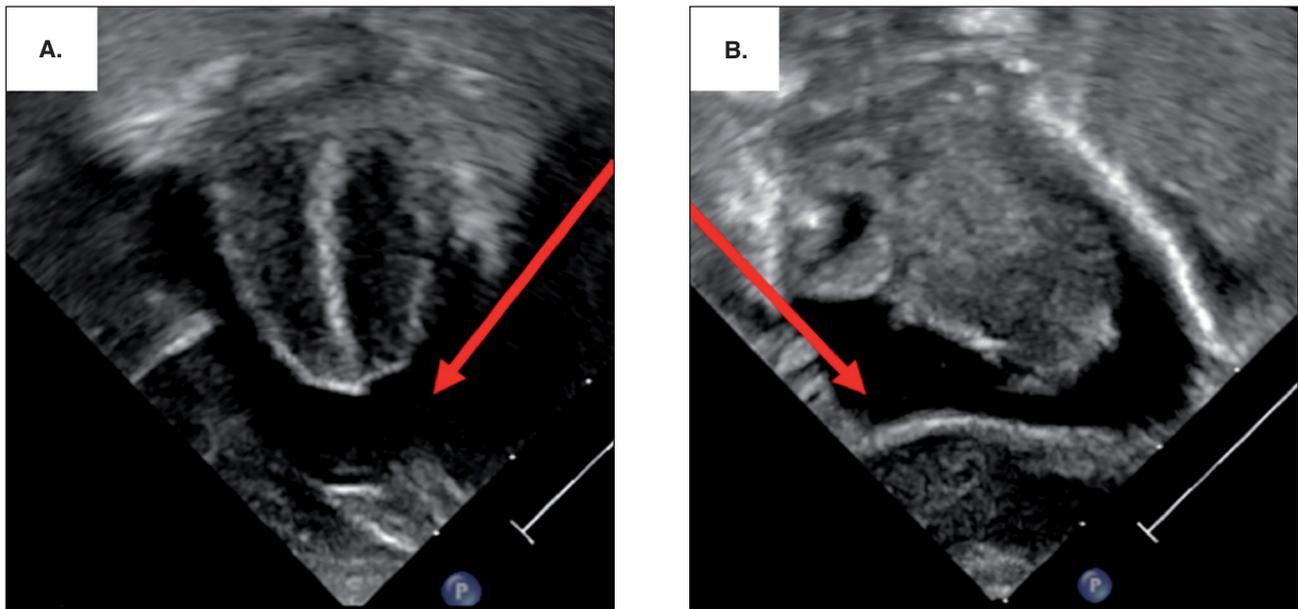


Figure 3. Apical 4 chamber view (A) and subcostal long axis (B) demonstrating pericardial effusion on echocardiogram (indicated by red arrows).

Table 1. Vital signs prior to, during and following the episode of pericardial tamponade.

| Time | HR (bpm) | SAP (mmHg) | DAP (mmHg) | PP | MAP (mmHg) | FiO ₂ |
|--|----------|------------|------------|----|------------|------------------|
| Prior to cardiopulmonary collapse | | | | | | |
| T -4 hours | 164 | 69 | 45 | 24 | 56 | 0.21 |
| T -2 hours | 149 | 54 | 42 | 12 | 47 | 0.21 |
| Event (cardiopulmonary collapse) | | | | | | |
| T 0 min | 120 | 34 | 23 | 11 | 27 | 0.27 |
| T 30 min | 117 | 30 | 21 | 9 | 24 | 0.27 |
| T 60 min | 157 | 37 | 28 | 9 | 32 | 0.75 |
| T 90 min | 99 | 23 | 17 | 6 | 19 | 0.75 |
| Time of intervention (pericardiocentesis) | | | | | | |
| T + 5 min | 151 | 79 | 48 | 31 | 61 | 0.75 |
| T +20 min | 131 | 73 | 45 | 28 | 57 | 1.0 |
| T +30 min | 130 | 68 | 42 | 26 | 53 | 1.0 |
| T +60 min | 153 | 51 | 34 | 17 | 41 | 0.21 |

HR: heart rate; SAP: systolic arterial pressure; DAP: diastolic arterial pressure; MAP: mean arterial pressure; PP: pulse pressure; FiO₂: fraction of inspired oxygen; T: time.

Nowlen et al. [1] reported 61 cases of central venous catheter-related pericardial effusions in infants. Sudden cardiac collapse requiring cardiopulmonary resuscitation was reported in 37 cases (61%). The use of point-of-care ultrasound (POCUS) to monitor peripherally inserted central catheter (PICC) tip position in preterm neonates [2] and detect complications such as pericardial effusion is well recognized. In this particular case, access to skilled personnel who can perform bedside targeted neonatal echocardiography (TnECHO) was potentially life saving [3]. In

addition, this case highlights the importance of comprehensive multimodal monitoring in critically ill neonates to determine the magnitude and breadth of the physiologic disturbance. Cerebral neurophysiology monitoring, such as aEEG, may assist in the timely recognition of impending acute cardiovascular compromise [4]. Near-infrared spectroscopy (NIRS) is also a useful tool for monitoring brain health, through longitudinal measurement of cerebral oxygenation, and may provide additional insights regarding high-risk patients or clinical situations where there are

downstream neurophysiological consequences of acute hemodynamic instability [5]. A prior study has shown the usefulness of integrated hemodynamic monitoring with TnECHO and NIRS in infants with compromised systemic circulation [6]. In summary, this case highlights the value of brain monitoring in acute cardiovascular emergencies; specifically, enhancing the clinical appraisal of the magnitude of the event, prioritizing its urgency and demonstrating response to intervention. The value of aEEG in monitoring the neurological impact of hemodynamic instability and appraising response to cardiovascular therapy requires prospective evaluation.

Established facts and novel insights of this case are presented in **Tab. 2**.

Table 2. Established facts and novel insights.

| | |
|--------------------------|--|
| Established facts | Peripherally inserted catheters are at risk of malposition, extravasation and may cause pericardial effusion in infants. |
| | Pericardial effusion may have high mortality and causes acute cardiovascular collapse. |
| Novel insights | During cardiovascular collapse, neurological monitoring using continuous aEEG may enhance clinical appraisal of the magnitude of the illness and demonstrate response to intervention. |
| | Access to bedside neonatologist performed TnECHO enables early diagnosis and prompt therapeutic intervention. |

aEEG: amplitude integrated electroencephalogram; TnECHO: targeted neonatal echocardiography.

Statement of Ethics

The parents of the subject have given written and verbal consent to publish this case.

Declaration of interest

The Authors have no conflicts of interest. There are no funding sources to report.

References

1. Nowlen TT, Rosenthal GL, Johnson DJ, Vargo TA. Pericardial effusion and tamponade in infants with central catheters. *Pediatrics*. 2002;110:137-42.
2. Motz P, Von Saint Andre Von Arnim A, Iyer RS, Chabra S, Likes M, Dighe M. Point-of-care ultrasound for peripherally inserted central catheter monitoring: a pilot study. *J Perinat Med*. 2019;47(9):991-6.
3. El-Khuffash A, McNamara PJ. Hemodynamic Assessment and Monitoring of Premature Infants. *Clin Perinatol*. 2017;44(2):377-93.
4. El-Naggar WI, Keyzers M, McNamara PJ. Role of amplitude-integrated electroencephalography in neonates with cardiovascular compromise. *J Crit Care*. 2010;25(2):317-21.
5. Sood BG, McLaughlin K, Cortez J. Near-infrared spectroscopy: applications in neonates. *Semin Fetal Neonatal Med*. 2015;20(3):164-72.
6. Elsayed YN, Louis D, Ali YH, Amer R, Seshia MM, McNamara PJ. Integrated evaluation of hemodynamics: a novel approach for the assessment and management of preterm infants with compromised systemic circulation. *J Perinatol*. 2018;38(10):1337-43.