Bullying in neonatal intensive care units: free for all

The increasing recognition of bullying (mobbing) at workplaces has resulted in the need for more evidence of this occupational hazard—immune to the professional status and difficult to define. Neonatal intensive care units probably represent the Antarctic, if the commonly repeated phrase in relation to bullying at workplaces is “What happened to NICUs just the tip of the iceberg”?. As places expected to function like a military base but run by civilians, such units probably provide the best training grounds for bullying under the disguise of legitimate management actions or “manifestation of work related stress”. Given the abundance of potential victims and the high prevalence of risk factors like technical/clinical inexperience, isolation, long shifts, and fatigue, it is not surprising that bullying of the new trainees is rampant in neonatal nurseries. Sadly, I have been a silent witness over 17 years to the devastating effects of bullying on the junior staff (especially the registrars) almost on a daily basis in neonatal nurseries. I have lost track of the number of trainees who resigned, lost self esteem, and developed a strong dislike towards neonatology due to constant bullying disguised as “constructive criticism” at work. Morning rounds provide the most common opportunity for professional humiliation of the registrars in neonatolgoy. However, there is no escape during the long, exhausting night shifts either. The choice is then between accepting sarcasm or public humiliation depending on whether seniors have to come in or not and the reactions of the onlookers. In a faculty where 27% of the consultants and 32% of nurses have been reported to have psychological abuse, it may be very difficult to sound the icebergs of workplace bullying in neonatology. The only ray of hope is a mentor who combines humanity with science during supervision of trainees.

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References

Positioning of long lines

We read the article by Reece et al and followed the subsequent correspondence with interest. In the light of the recent review commissioned by The Chief Medical Officer for England, physicians must be aware of potential complications of peripherally inserted central catheters (PICCs). Although the true incidence of such events will only be known with prospective data collection, retrospective studies suggest a complication rate (pleural/pericardial effusions) of 0.5% per line insertion. The Department of Health (DoH) paper in response to this review recommends placement of central venous lines outwith the cardiac chambers. However, complications related to central lines are not only confined to the thorax. We report three cases of delayed detection of peritoneal extravasation related to central venous catheters.

Case 1. An 8 week old malnourished infant was intubated and ventilated for acute onset severe respiratory distress after a brief flu-like illness. His endotracheal secretions were positive for respiratory syncytial virus, and a previously undetected myopathy was suspected and investigated. He required prolonged ventilation and received blood transfusions through a triple lumen right femoral venous catheter for anaemia. On the fifth day after admission, he developed abdominal distension, and a diagnostic tap under ultrasound guidance showed haemorrhagic ascites. A
contrast radiograph of the femoral catheter showed extravascular spillage of dye, in this case in the extraperitoneal space. In retrospect, the infant showed no rise in haemoglobin after two packed cell transfusions. Extravascular migration of the catheter tip was diagnosed and the catheter was promptly removed.

Case 2. A 24 week preterm female infant was admitted to the neonatal intensive care unit where she was ventilated and treated with surfactant. Severe respiratory distress of prematurity, patent ductus arteriosus, and sepsis complicated the initial course. The patency ductus arteriosus did not respond to two courses of indomethacin, and surgical ligation was scheduled on day 13 of life. A PICC was removed because of suspected catheter sepsis, and she was treated with intravenous antibiotics. She had a right femoral venous central single lumen catheter through which she received parenteral nutrition and blood transfusions on the unit. Her anaemia failed to respond to the three packed cell transfusions, and, when she developed abdominal distension with bluish discolouration in the groin, the femoral line was radiographed (fig 1). The PICC was removed and the infant made a full recovery.

In each of these case reports, femoral catheter tip migration was detected after extravascular extravasation of blood or parenteral nutrition fluid. Haemoperitoneum has been reported in the past as a complication of central catheters but may not be widely recognised. In comparison with PICCs, these catheters are shorter and more rigid, hence more likely to perforate the vessel wall. Femoral venous access is readily obtained in infants and is commonly used in intensive care units for parenteral nutrition, the view of many fluids, blood transfusions, and other parenteral treatment. Although malpositioning of a femoral catheter is readily detected in most instances during placement, a spontaneous extravascular migration of a previously well placed catheter tip is possible in some cases. As opposed to frank rupture of the blood vessel and haemorrhage into the retroperitoneum, slow extravascular infusion of blood in the low resistance extraperitoneal space may not be promptly detected in the absence of a high index of suspicion. In preterm newborns and malnourished infants, the integrity of the vessel wall may be compromised, and migration of a previously normally positioned catheter tip may be more likely. Femoral venous catheter tip positions must be reviewed in all cases of unexplained ascites and abdominal distension. Contrast radiography, digitalised image inversion, and ultrasonography have a role in determining catheter tip position and diagnosing malpositioned intravascular catheters.

We agree with the DOH recommendation that there should be a prospective national audit of such cases.

Weighing breast fed babies

Harding et al.4 state that the problem of hypernatraemic dehydration in breast fed babies is attributable to “the reluctance of midwives to weigh breast fed infants”.5 They imply that weighing between 72 and 96 hours would be an effective intervention, but provide no evidence to support this claim. It does not surprise me that the views of midwives and consultant neonatologists differ on this point. Whereas midwives in this country receive specific training in the management of breast feeding, most paediatricians do not. Indeed most are (to coin a political aphorism) “one club golfers” where the assessment of breast feeding is concerned: weighing might be one indicator of a baby’s fluid balance but there are other ways of assessing the adequacy of breast feeding, including feed observation. Given these differences in expertise, one might reasonably ask which group of professionals give the correct answer!

As Harding et al. point out “normal weight loss is at its maximum” between 72 and 96 hours after birth. In the majority of many midwives (and mine) this constitutes the strongest argument against weighing a baby who has been observed to feed well. Demonstrating this weight loss frequently underestimates breast feeding need no matter how carefully the physiology of the phenomenon is explained. Additionally (particularly in primiparous mothers), lactogenesis is only just becoming established between 48 and 72 hours. Thus the volume of milk transferred to the infant is still rising sharply between 72 and 96 hours of age.

Before early weighing can be recommended as a screening test for hypernatraemic dehydration more information is required. Firstly the precision of weighing in practice and the accuracy of measured differences need clarification. In relation to this the predictive value of a weight change on day 3–4 needs to be ascertained. Secondly the comparative merits of other methods of assessing breast feeding need more emphasis (the American Academy of Pediatrics consensus statement cited by the authors views weighing as only one aspect of the assessment). Thirdly the nature, effectiveness, and cost of resulting interventions need to be considered (more than “encouragement to continue feeding” may be needed). Finally the potential adverse consequences of weighing (alluded to above) need to be set against any diagnostic benefit.

In summary, I fully agree that the prevention of hypernatraemic dehydration is important but am not persuaded that early weighing is the answer. In my view better training of health professionals, including paediatricians, in the management of breast feeding would go further.

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Chasing spurious hyponatraemia

The need for a conservative approach to hyponatraemia in preterm neonates was emphasised by Manzer.6 Chasing hyponatraemia (serum sodium: 124 mmol/l) during hyperglycaemia requiring insulin infusion in a 1060 g neonate was associated with fluid retention and patent ductus arteriosus (PDA). The maximum serum sodium levels achieved were 136 mmol/l (maximum sodium supplements: 15 mmol/day). However, spurious hyponatraemia is well known during hyperglycaemia and hypertriglyceridaemia.7

Elevation of serum glucose levels induces a translocation of water from the intracellular fluid to the extracellular compartment sufficient to reduce serum sodium.8 Correction factor of Katz states that the serum sodium level decreases by 1.6 mmol/l for each 5.6 mmol/l increase in glucose level.9 Hyponatraemia during hypertriglyceridaemia is a method dependent artificial reduction of serum sodium concentration resulting
from displacement of a portion of a water phase of the plasma by lipid. This problem (which may be observed with flame emission spectrometry) can be avoided by using a sodium selective electrode without dilution. Hyperglycaemia and hypertriglyceridaemia are common in extremely low birth weight (ELBW) neonates—the group at highest risk for chronic lung disease (CLD) and symptomatic PDA. The incidence of hyperglycaemia in very premature neonates ranges from 20% to 86% and is at least 18 times greater in ELBW neonates. The consequences of chasing such spurious hyponatraemia in neonates at risk for CLD and PDA cannot be over emphasised.

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References

BOOK REVIEW

Monographs in Clinical Paediatrics: Neonatal Jaundice.

This monograph is a useful summary of the problem of significant hyperbilirubinaemia in the neonate with full description of the biochemical and pathophysiological aspects of the condition. Each of the subjects is also dealt with from a historical perspective, which provides considerable interest. I was particularly interested to note that the first ever exchange transfusion performed on a jaundiced child (five previous children had died of bilirubin toxicity) is described as “transfusion and exanguination” via the saphenous vein and sagittal sinus respectively; this was carried out as a continuous exanguination/transfusion. Since that time, many techniques have been used, although the approach to the sagittal sinus via the anterior fontanel is no longer practiced. Interestingly, although other continuous techniques have been developed they are now rarely used; there is a common preference for a “in/out” technique.

In the chapter on The Clinical Approach to the Jaundiced Newborn, the algorithm of the American Academy of Paediatrics is reproduced. I was particularly interested to look at the approach to persistent jaundiced and was disappointed to find that the only advice given for jaundice persisting more than three weeks was to “perform appropriate physical and laboratory assessment of infant including possibility of cholestatic jaundice” when it had already been established that the infant did not have abnormal physical exam results, dark urine, or light stools. In my view, the demonstration of normal levels of conjugated bilirubin at this age is sufficient to rule out significant liver disease, which requires further management.

There is an interesting discussion of whether total or unconjugated bilirubin should be measured in assessing risk for kernicterus. The surprising conclusion (supported by anecdotal case reports) is that conjugated bilirubin should not be subtracted from the total unless it exceeds 50% of the total, when there is some evidence that even conjugated hyperbilirubinaemia may contribute towards kernicterus in the presence of a high total bilirubin.

I conclude that this is a useful reference text and an excellent source of definitive information, but it is unlikely to find its way to the benchbook section of the neonatal library.

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