

In preterm infants, does the supplementation of carnitine to parenteral nutrition improve the following clinical outcomes: Growth, lipid metabolism and apneic spells?

Part B: Clinical commentary

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Carnitine is found in human milk (1) and is absent in total parenteral nutrition (TPN) (2). Preterm infants have lower tissue carnitine stores than term infants (3,4), and levels drop quickly within the first two weeks of life if fed diets lacking carnitine (5,6). Because of the role of carnitine in fatty acid oxidation (which contributes to energy metabolism and growth), there has been considerable interest in carnitine during the neonatal period (2). There are at least two theoretical reasons for adding carnitine to neonatal diets: nutritional biochemistry and simulation of human milk composition. It is for these two reasons, and not for clinical reasons, that carnitine is added to infant formula in concentrations similar to those found in human milk (7). As with enterally fed infants, there is no clinical advantage of adding carnitine to TPN of intravenously fed infants, as noted in the previous data analysis (Part A, pages 571-572). However, all studies were small and mostly short-term. There are no studies on carnitine supplementation in infants with short bowel syndrome or infants receiving home TPN who may need intravenous feeding for years and who may have the highest risk of developing functional carnitine deficiency. Interestingly, while there is insufficient clinical data to recommend supplementation of TPN with carnitine for preterm infants, carnitine is being added to infant formulas without the support of evidence-based clinical data or

without firm recommendations by the Canadian Paediatric Society or Health Canada (7,8). Considering the evidence available, there is no clinical advantage in adding carnitine to short-term regimens of TPN of newborn infants. Whether or not carnitine supplementation offers an advantage to infants receiving long-term parenteral nutrition remains to be researched.

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