

sion, samples were obtained at the bag, distal to the in-line 0.22 micron filter, and after mixing with fat emulsion. Recovery of carnitine was 88.1 to 109% of theoretical across all sampling sites and time points.

Based on this data, we conclude that carnitine is highly stable in TPN and TNA formulations and that no clinically significant loss of carnitine occurs through bag, tubing, or filter absorption.

## 卡尼汀(肉毒碱)在儿科肠外营养和全营养混合液中的稳定性

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长链脂肪酸进入线粒体进行氧化供能时需要卡尼汀参与,他是儿科肠外营养病人的条件必需营养素。根据我们以前的临床研究结果,推荐卡尼汀的剂量为  $20\text{mg}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$ 。然而,肠外营养和全营养混合液中卡尼汀的稳定性尚未得以正确评估。

在儿童肠外营养和全营养混合液中以  $130\sim 200\text{mg/L}$  的标准加以卡尼汀,所配混合液中卡尼汀稳定性的测定时间分别为:配制后的当时和配制后的第1天、第7天、第15天和第30天(此间在  $4\sim 5^\circ\text{C}$  中保存)。测定后的制剂则置室温保存,并用荧光灯持续照射,分别在0、6、12、18和24小时采取标本。用碳14放射分析技术重复测定同一标本的卡尼汀浓度,这是稳定性的标志。同时检测混合液的pH值变化、颜色变化和可见沉淀物的发生。

结果表明:保存30天的混合液,没有pH值和颜色的变化,也没有可见沉淀的发生。在配制后当时,6个不同标本的卡尼汀回收率为  $93.9\% \pm 5.31\%$  (理论值的均数  $\pm$  标准差,区间为  $87.8\% \sim 101\%$ )。  $4\sim 5^\circ\text{C}$  存放的标本,在不同时间点所测定的结果相近。存放30天后,再室温放置24小时,卡尼汀的理论回收率为  $93.3\% \pm 7.30\%$  (区间为  $81.0\% \sim 102\%$ )。半胱氨酸和雷尼替丁都不影响卡尼汀的稳定性。我们进行包括经Y型管输注20%脂肪乳的模拟输液试验,经24小时的输注之后,采集袋中、末梢  $0.22\mu\text{m}$  滤器中及与脂肪乳混合后的标本。不同时间点和不同部位标本的卡尼汀回收率是理论值的  $88.1\% \sim 109\%$ 。

上述资料提示:卡尼汀在肠外营养和全营养混合液中是高度稳定的,袋子、管子和滤器对卡尼汀的吸收不具有临床意义。

(牛玉坚译)

## 8. Synthetic dipeptides—a new dimension in clinical nutrition

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Recent investigations clearly indicate that certain amino acids previously considered as non-essential are conditionally indispensable substrates in various diseased states: Hypermetabolic and hypercatabolic situations are accompanied by a marked depression of the intracellular glutamine pool. This depletion of glutamine stores leads to severe complications, such as infection, poor wound healing, impaired immunity, increased intestinal permeability, and finally multiple organ failure. In the liver tissue of fetuses and preterm and term infants the endogenous synthesis of the sulfur containing amino acid cysteine is impaired due to low or undetectable activity of the key enzyme cystathionase. Low intracellular concentrations of taurine are a typical feature in chronic renal failure and presumably associated with muscle fatigue. Under certain pathological conditions like phenylketonuria; liver and kidney diseases, and in premature infants the aromatic amino acid tyrosine is considered conditionally indispensable due to impaired synthesis from phenylalanine. These data strongly support the notion that glutamine, cysteine/cystine, taurine, and tyrosine should be essential parts of artificial nutrition in various diseased states. However, unfavourable physical/chemical properties as well as metabolic limitations hamper their use as parenteral substrates in routine clinical setting. Glutamine quantitatively decomposes in aqueous solutions during heat sterilization and long-term storage to yield pyroglutamic acid and ammonia. In addition, provision of free glutamine in adequate amounts to patients is always associated with a high water load due to its limited solubility. Tyrosine and cystine are poorly soluble (only 0.4 and 0.1g/L  $\text{H}_2\text{O}$ , respectively); cysteine rapidly oxidizes to yield cystine. Parenteral free taurine might not be available for the depleted intracellular compartment because of a low transport rate due to the very high intracellular/extracellular transmembrane gradient.