

- 1981;11:175-83.
21. Hirokawa K, Iwafuchi M. Abnormal liver function during long-term intravenous hyperalimentation in children. *Jpn. J. Pediatr. Surg.* 1988;20:695-9.
  22. Matsumoto Y, Ohi R, Shimanuki M, et al. Hepatic dysfunction during TPN in pediatric surgical patients- with a special reference to quality quantity of amino acid solution. *Jpn. J. Surg. Nutr.* 1985;19:175-6.
  23. Ohta G, Pathogenesis and morphology of cholestasis. *Kan-tan-sui (Japan)*. 1986;13:841-7.
  24. Klaassen CD, Watkins, B. III. Mechanisms of bile formation, hepatic uptake and biliary excretion. *Pharmacol. Rev.* 1984;36:1-67.
  25. Scharschmidt BF, Van Dyke RW. Mechanisms of hepatic electrolyte transport. *Gastroenterology* 1983;85: 1199.
  26. Phillip MJ, Oda M, Mak E, et al. Microfilament dysfunction as a possible cause of intraphepatic cholestasis. *Gastroenterology* 1975;69:48-58.
  27. Tamura K, Kuroda H, Watanabe S, et al. Actin filaments of hepatocytes in experimental rat cholestasis. *Acta Pathologica et Cytopathologica* 1981;41:115-20.
  28. Roel JV, Ibrathim M, Yousef J. P corriveau and Beatriz Tuchweber. Phalloidin-induced morphological and functional changes of rat liver. *Liver* 1982;2:133-40.
  29. Dubin M, Maurice M, Feldmann G, et al. Influence of colchicines and phalloidin on bile secretion and hepatic ultrastructure in the rat. *Gastroenterology* 1980;79: 646-54.
  30. Fujiwara T. Brain-damaging potential of acidic amino acids in the parenteral nutrition for infants. *Jpn. J. Surg. Metab. Nutr.* 1982;16:123-34.
  31. Chen SC, Wu CH, Li YZ, Fujiwara T, Sunagawa M. Brain-damaging potential of infant rats treated intravenously with L-synthetic histidine. *Dokkyo Med. J.* 1995;10:325-30.
  32. Cohen MI, Litt IF, Schornberg SK, et al. Hepatic dysfunction associated with parenteral alimentation clinic and experimental studies. *Pediatr. Res.* 1973;7:334-8.
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- REFERENCES**
- The mitochondria, one of the organelles, became swollen in the hepatocytes of kinder-TPN rats (Fig. 1c). The swollen mitochondria can be seen in Fig. 2c (group II) and Fig. 3c (group III) with Fig. 1b (group I) for comparison. In kinder-TPN rats, the mitochondria were easily destroyed or degenerated, so our hepatocytes had more mitochondria than normal hepatocytes. Swelling of the mitochondria was probably caused by increased permeability of the mitochondrial membrane to L-form amino acids. The mechanism may be related to the increase in the amount of L-form amino acids in the serum. When the amount of L-form amino acids increases, the permeability of the mitochondrial membrane to L-form amino acids should become easily increased.