at 8 d of age or older. Percentage of triglycerides in the total lipids of the liver and plasma increased progressively in the hypoxic newborn rats (Table 1) but beta-hydroxybutyrate and acetoacetate blood levels were normal for the age. Liver glycogen stores were depleted in the hypoxic suckling rats with fatty liver. When hypoxic rats were returned to sea level pressure for 8, 24, and 48 h before sacrifice, the level of total lipids in plasma and liver decreased, and the beta-hydroxybutyrate blood level increased significantly. Total liver lipids fell to normal values in the litter kept at sea level pressure for 24 h. Carbon monoxide produced similar results.

The fact that the rat is the only known species to show hypoxic impairment of the lipid metabolism with severe fatty liver degeneration in the perinatal period supports the existence of an unique type of lipid metabolism. Hypoxia could serve as a tool to unravel the characteristics of this apparently unique type of lipid metabolism.

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Letter to the Editor

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The excellent review by Brasel (1), "Endocrine Adaptation to Malnutrition" is a thorough attempt to look for generalities in a confusing field. It is possible that one of the points of confusion Brasel leaves unresolved contains within it information that may help to reduce the confusion, and suggests a need for greater liason between investigators.

Brasel points out that studies on growth hormone in malnutrition generally report high levels of this hormone, with exceptions, in cases where growth hormone levels were originally normal or diminished. The studies of Monckeberg *et al.* (2) and Robinson *et al.* (3) were cited. Both of these studies do more than this. Robinson *et al.* finds abnormally high growth hormone levels in malnourished children showing zero or negative weight gain. These levels are 2–5 times higher than during dietary treatment. Dietary treatment initiates the recovery or catch-up growth phase. Both Monckeberg *et al.* and Robinson *et al.* also report on growth hormone levels in children being treated for existing malnutrition.

Similar reports have been given by Beas *et al.* (4) and Godard (5). They point out that during treatment and/or recovery from protein-energy malnutrition, lower growth hormone values were found in children in whom growth rate was very slow [Robinson *et al.* (3) and Godard (5)] or in those in whom recovery was not yet initiated [Monckeberg *et al.* (2) Beas *et al.* (4)] and higher growth hormone levels were found in those showing satisfactory growth rates [Robinson *et al.* (3) and Godard (5)]. The administration of growth hormone to the children who were not growing significantly increased growth rates [Monckeberg *et al.* (2)]. At no time during the catch-up growth phase [Robinson *et al.* (3)] did the growth hormone levels approach the abnormally high values shown in the malnourished group.

Malnutrition is a state defined anthropometrically. As such it is a measure of an anthropometric state achieved and not the rate of change of nutritional status or growth. The results of Monckeberg *et al.* (2), Robinson *et al.* (3), Beas *et al.* (4) and Godard (5) indicate that this rate of change, shown during recovery, is significantly related to growth hormone levels.

It would help in the interpretation of all results on malnourished children if clinical status could be specified more precisely than is possible by currently accepted anthropometric classification schemes, or by traditional clinical divisions into named disease states. It would be illogical to ask that only growth rates should be given, although they are clearly important. It is also reasonable to assume that food intakes would be important determinants of hormonal profile, and also that patients can be not only recovering, but if studied early enough after the initiation of malnutrition, their hormonal profile may also reflect their rate of deterioration.

It is difficult to see how coherence can be obtained. Is it unreasonable to suggest a discussion among principal investigators is needed to agree on an ideal list of clinical data for any malnourished child studied, and to propose the requirement for a single archive where copies of such new data could be lodged by all investigators, and made accessible to all workers in the field, permitting more easily the detection of trends of the type reported by Monckeberg *et al.* (2), Beas *et al.* (4), Godard (5), and Robinson *et al.* (3)?

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Response

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I completely agree with Dr. Robinson that much of the difficulty in evaluating the data on growth hormone levels in malnutrition is related to the fact that the subjects are often in differing states of malnutrition and/or recovery. It would have been very helpful to me in my review if the types of additional information on the patients and their condition, which is mentioned in the letter, had been available. I can only support the proposal that investigators in the field develop a format for recording such data for the use of others with great enthusiasm. Good luck!

Letter to the Editor

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I have just read the paper in your journal by Sampson *et al.* "Manganese Balance Studies in Infants after Operations on the Heart" *17:* 263 (1983). There are one or two points that need correction. In Table 4 on page 264, it should be noted that both S26 and SMA are manufactured by Wyeth. The levels of manganese quoted for these two products are stated to be low, and if nmole/litre is converted to μg/litre the values would read: S26 - 14.8 ± 7.7 and range, 5.8–63; and SMA - 11.8 ± 7.7 and range 3.8–23. In fact our present label claim is 160 μg per reconstituted litre, a level significantly greater than the Sampson analysis.

As fortification of our nutritional products with manganese was introduced in September 1977, the levels quoted in the article must be associated with unfortified products manu-