THE CANADIAN MEDICAL ASSOCIATION

LE JOURNAL DE

L'ASSOCIATION MÉDICALE CANADIENNE

OCTOBER 23, 1965 • VOL. 93, NO. 17

SYMPOSIUM ON NUTRITION

Overnutrition in Prenatal and Neonatal Life: A Problem?

W. A. COCHRANE, M.D., F.R.C.P.[C],* *Halifax, N.S.*

ABSTRACT

In most areas of the world nutritional problems are related to quantitative and qualitative deficiencies. However, in North America the possible harmful effects of overnutrition deserve careful consideration.

Little information is available concerning overnutrition during prenatal and neonatal life. The author draws attention to this possibility by outlining clinical and biochemical disorders occurring in the newborn which are related to excessive ingestion of calories, fat, protein, vitamins and minerals before and after birth. Particular reference is made to the ingestion of nutritional substances during pregnancy in amounts that are relatively innocuous to the mother but may be harmful to the infant *in utero*.

Further research in this interesting field is needed in an attempt to assess the effect on the fetus of qualitative overnutrition during pregnancy.

SOMMAIRE

Dans la plupart des régions du globe, les problèmes de l'alimentation se posent en termes d'insuffisances quantitatives et qualitatives. Tandis qu'en Amérique du Nord, on devrait plutôt considérer les effets pernicieux éventuels de la suralimentation.

On possède peu de documents sur la suralimentation pendant la vie pré-natale et la vie du nouveau-né. L'auteur attire l'attention sur cette possibilité en signalant les troubles cliniques et biochimiques qui surviennent chez le nouveau-né et qui sont en relation avec un apport excessif de calories, de lipides, de protéines, de vitamines et de minéraux avant et après la naissance. Il insiste surtout sur l'ingestion de substances nutritives pendant la grossesse, en quantités qui sont relativement inoffensives pour la mère, mais qui peuvent être nuisibles au nourrisson *in utero*.

Il faudra d'autres recherches dans ce domaine intéressant, en vue d'essayer d'évaluer l'effet sur le fœtus d'une suralimentation qualitative pendant la grossesse.

THE problem of qualitative and quantitative undernutrition has attracted a great deal of attention. Although this disorder may occur in some communities in Canada and the United States, it would seem more appropriate, in North America, to direct attention to the possible problems of overnutrition. Physicians and nutritionists are familiar with the problems associated with excessive caloric intake and obesity in the adult;[1] excessive intakes of certain fats, minerals and specific vitamins have also produced recognizable clinical entities. In children a great deal of attention has been directed towards infant feeding, but much less attention has been given to intrauterine nutrition and neonatal overnutrition.

The early studies by Ebbs,[2] Burke *et al.*[3, 4] and others emphasized the effects of inadequate diets during pregnancy on the course of pregnancy and the condition of the infant at birth. However, spe-

Presented before a joint Canadian-United States Conference on Nutrition, sponsored by the American Institute of Nutrition and the Nutrition Society of Canada, Toronto, September 14 and 15, 1964.

*Professor, Department of Pediatrics, Faculty of Medicine, Dalhousie University, and The Children's Hospital, Halifax, Nova Scotia.

Canad. Med. Ass. J.
Oct. 23, 1965, vol. 93

cific information on the relation of maternal nutrition to the intrauterine and subsequent growth and development of the baby has been somewhat slow to appear and, as yet, extensive studies have failed to clarify precisely the relationship of maternal nutrition to the outcome of pregnancy.[5] This failure is understandable when it is recognized that the process of intrauterine development involves not only a host-parasite relationship but an organ (the placenta) whose physiological function is not yet clearly defined. For some nutrients an active transport system exists to ensure an adequate supply to the fetus, while for others the nutrient may be preferentially retained in the maternal circulation.

TABLE I.—A COMPARISON OF THE LEVELS OF VARIOUS METABOLITES IN MATERNAL AND FETAL BLOOD*

Level higher in maternal blood	*Levels equal*	*Level higher in fetal blood*
Glucose (?)	Sodium	Total calcium
Copper	Potassium	Iron
Total protein,	Chloride	Inorganic phosphorus
albumin,	Zinc	Vitamin B_1
fibrinogen	Urea	Vitamin B_6
Organic		Vitamin B_{12}
phosphorus		Vitamin C
Choline esterases		Folic acid
Choline		
Vitamin A		
Vitamin E		

*From data reported by Morison, J. E.: Fœtal and neonatal pathology, 2nd ed., Butterworth & Co. (Publishers) Ltd., London, 1963, p. 5.

In some instances where there is marginal supply of nutritive materials there may be competition between mother and fetus, which varies with each nutrient. Considerable discrepancy exists between the level of certain nutrient substances in human maternal blood as compared to that in the fetal blood (Table I). Proteins, fatty acids, vitamin A and vitamin E are present in higher concentration in the maternal blood than in the fetal blood, but the reverse is the case with iron, vitamin B_1, B_6, B_{12} and vitamin C. Much experimental evidence indicates that certain specific nutrient deficiencies can produce "congenital" anomalies in animals.[6] Thus, deficiencies in the diet of pregnant rats of riboflavin, vitamin A, folic acid and other substances at specific periods of gestation have resulted in various anomalies of the head and neck, cardiovascular system, and urinary tract, as well as certain neural defects. It is also well known that certain drugs administered during pregnancy have similar effects.

The concept of a "critical phase" during a developmental process at which environmental influences could, at a specific moment, disorganize fetal development and produce specific malformations may be somewhat restrictive. Certainly damage to an organ may occur not only when relevant embryological changes are occurring, but earlier and at any time before the stage of development is completed; the specific type of damage may depend on the time of the insult.

In contrast to the work on the effect of undernutrition on the neonate, very little has been done to examine the possible effects of overnutrition during pregnancy on the health of the baby. Some of the interesting aspects of overnutrition in intrauterine life will be examined in this communication, since they are directly related to physician-prescribed or physician-recommended nutrition habits during pregnancy.

OVERNUTRITION *in utero*

Excess Calories and Glucose

On the maternal side, excessive caloric intake during pregnancy may involve an additional risk to the infant.[7] The direct relationship between maternal obesity and toxemia of pregnancy is well recognized and presents a threat to the survival of the fetus.

However, a question that has as yet not been adequately answered concerns the relationship between large infants and maternal diet. In general the infant of the diabetic and prediabetic mother is larger than the infant of the non-diabetic mother. The role of carbohydrate metabolism and insulin is immediately considered as a factor of possible significance in the pathogenesis of the excessively large infant. Infants of diabetic mothers are generally grossly overweight because of surplus fat and carbohydrate stores in their tissues and organs. The water content of these tissues is low. The generally accepted hypothesis is that hyperglycemia in the mother results in fetal hyperglycemia and subsequent hypertrophy of the islets of Langerhans. A resultant increase in fetal insulin and a greater utilization of glucose received from the mother produces larger and fatter newborn infants who may be hypoglycemic at birth. Studies of insulin and glucose levels in these infants at birth have shown that high levels of insulin are associated with early onset of hypoglycemia.[8]

Similarly, excessively large infants are not uncommonly associated with prediabetic mothers. Baumgartner *et al.*[9] in 1950 observed an increased mortality in newborns with low birth weights, but also noted a greater mortality in large, heavy newborn infants. The possibility that excessive carbohydrate ingestion may account for large infants of prediabetic or even non-diabetic mothers may well be confirmed in the near future.

Vitamins

Warkany[6] has emphasized the effects on the fetus *in utero* of short-term deficiencies of certain substances. The possible harmful effects to the fetus of excessive amounts of vitamins ingested or administered to the mother during her pregnancy are as follows.

Vitamin K

Recent evidence has confirmed the toxic effects of excessive amounts of synthetic vitamin K administered to young newborn infants and prematures.[10] It has also been observed that administration of large amounts of vitamin K analogue to the mother shortly before delivery results in an increase in the serum bilirubin of the newborn infant.[11] This material, previously considered innocuous, is dangerous when given in large amounts to mothers shortly before birth; consequently, appreciably smaller dosages are now employed. The naturally occurring vitamin K does not have this effect.[12]

Vitamin B_6 (Pyridoxine)

In 1954 Hunt *et al.*[13] described a newborn infant who had recurrent convulsive seizures which were controlled by the administration of large amounts of pyridoxine hydrochloride. The mother had received parenteral vitamin B_6 during her pregnancy, and Hunt suggested that the infant might be "pyridoxine dependent", since the seizures were only controlled by pyridoxine 2 mg. daily. This observation concerning the effect of pyridoxine administration during pregnancy raised the question of enzyme induction (the synthesis of a new enzyme in contrast to the activation of an existing enzyme) and its role in pyridoxine dependency. Similar cases subsequently reported were not associated with excessive maternal pyridoxine ingestion. Sufficient evidence from animal experimentation is not yet available to settle the matter of "pyridoxine dependency" resulting from excessive administration of pyridoxine during intrauterine life. If this theory is given any weight at all, it is reasonable to administer large doses of pyridoxine, at term or during labour, to the mother who has previously given birth to a pyridoxine-dependent infant; conversely, it would be harmful to give pyridoxine prior to term.

Ascorbic Acid

Forty-two cases of infantile scurvy were seen at the Children's Hospital in Halifax, Nova Scotia, from October 1959 to January 1961. Of particular interest was the fact that in seven of the patients a history of adequate administration of vitamin C was obtained. However, on careful follow-up by home visits it was found that five of these patients had not received sufficient vitamin C; that is, the mother's account of vitamin administration could not be substantiated. Two infants had classical scurvy, and after detailed study it was concluded that these children had both received adequate amounts of ascorbic acid. The parents of both infants were questioned, the pharmacy where the vitamin was purchased was visited, and the parents' stories were confirmed. Samples of the vitamin were obtained, were assayed for ascorbic acid and were found to have full potency, *viz.* 15.1 mg./c.c. It was estimated that the children received 60 mg. of ascorbic acid daily. The possibility of "conditioned" scurvy was raised when it was found on detailed questioning that both mothers had received large supplements of ascorbic acid during pregnancy. The ingestion of vitamin capsules and a large volume of fruit juices resulted in an approximate daily intake of 400 mg. of ascorbic acid.

In order to investigate the possible existence of "conditioned" scurvy, preliminary studies were carried out in association with Dr. W. W. Hawkins of the National Research Council in Halifax. Twelve female guinea pigs were divided into four groups of three each, and were allowed rabbit chow and water *ad libitum*. Each animal was given ascorbic acid, initially by mouth and subsequently by the subcutaneous injection of a neutralized solution. The amount of ascorbic acid administered was 3 mg. per day to one group; 20 mg. per day to the second group; 200 mg. per day to the third group; and 1 g. daily to the fourth group. When the first 10 young were born they were taken from the mothers and allowed rabbit chow and water *ad libitum*. Each of the young in the four groups was given 1.5 mg. of ascorbic acid daily by intraperitoneal injection. On the death of the young animals over the ensuing two to five weeks, radiographs were made and histological examination of long bones was carried out. In some cases blood was taken from the heart for determination of the ascorbic acid concentration, the injection of ascorbic acid for that day being omitted. In two of the 10 young guinea pigs marked bone hemorrhage and cellular changes were noted, in keeping with the histological changes seen in ascorbic acid deficiency. Both animals were the offspring of the mothers that received 1 g. of ascorbic acid daily. These findings suggest that an ascorbic acid dependency was induced in the young by exposure to the vitamin *in utero*. More extensive studies have been undertaken to confirm this initial observation. It is possible that the ingestion of excessive amounts of certain vitamins by the mother during pregnancy may "condition" the offspring to requirements of these substances that are greater than the present expected or recommended intakes. If the greater requirements were not met, deficiency symptoms might occur in such infants despite the addition of these substances to the infant's diet in amounts usually recommended.

Vitamin D

Cooke recently expressed the opinion that excessive intake of vitamin D by mothers who are perhaps sensitive to vitamin D may be a significant cause of mental retardation in the offspring.[16] There appear to be several such syndromes, which include cardiovascular anomalies, peculiar facies and mental retardation, related to hypercalcemia.

Canad. Med. Ass. J.
Oct. 23, 1965, vol. 93

The condition of idiopathic hypercalcemia in infants has been recognized for many years and has been considered by many to be related to excessive intake of or hypersensitivity to vitamin D[14]. Recently Garcia *et al.*[15] described a 9-month-old infant with supravalvular aortic stenosis, probable mental retardation, typical facies and hypercalcemia. Because of the early onset and extent of the lesions the condition was believed to have begun *in utero*. Cooke suggested that excessive intake of vitamin D during pregnancy by women with an abnormal sensitivity to the vitamin may account for this entity.[16] The hypothesis seems reasonable but further investigation is necessary.

OVERNUTRITION IN THE NEONATE

It is now becoming evident that during the neonatal period—the first 28 days of life—the young infant, adjusting to new surroundings, has certain physiological limitations. It is during this period that excessive administration of ordinarily innocuous substances may result in cellular damage and even death of the infant.

Excessive Solute in Infant Feeding

The feeding of large amounts of protein and electrolyte to young infants exposed to relatively high environmental temperatures for long periods of time, or to infants with temporary renal insufficiency, may result in severe illness and possible death. The limited concentrating ability of the premature and newborn infant kidney requires the expenditure of a certain volume of water per kg. of body weight for adequate removal of the solute resulting from a normal diet. If the intake of protein is increased above the average level (i.e. the point at which 15% of total calories are provided by protein), harmful effects may occur. The clinical syndrome of hypernatremic dehydration follows the administration of excessive protein to sick or premature infants who are not ingesting sufficient water to enable their immature kidneys to excrete the solute load thus produced. There is still a tendency in many nurseries to give premature infants increased amounts of protein, which may be added by supplementation to a skim-milk formula. This "strengthening" of the formula may be resorted to when the infant is not gaining satisfactorily. This problem is illustrated in the following case report.

An 8-week-old infant was born after 28 weeks' gestation and weighed 2 lb. 6 oz. From the third to sixteenth days of life she received a formula of two-thirds breast milk and one-third powdered 2%-fat milk which was gradually increased in amount. On the sixteenth day she was given a formula of powdered 2% milk diluted in the recommended ratio of one tablespoon to 2 oz. of water. No additional carbohydrate was added. The child's weight increased from 2 lb. 9 oz. to 4 lb. 14 oz. by the forty-eighth day of life. The formula was then "strengthened" further to one tablespoonful of powdered 2%-fat milk to 1 oz. of water. Eight days later the child developed a low-grade fever with rapid respirations and took her feeding poorly. On physical examination she was a fairly active and well-developed infant weighing 5 lb. 8 oz. There was some pitting edema of the feet and ankles, puffy eyelids and a rather doughy skin. The hemoglobin was 8.5 g./100 ml. and the urine revealed a trace of albumin with white cells and granular casts. The serum electrolytes were: sodium, 179 mEq./l.; chloride, 147 mEq./l.; potassium, 5.5 mEq./l. and blood urea nitrogen 62.5 mg./100 ml. The CO_2 content was 5 mEq./l. The child was treated with intravenous 10% glucose and water and sodium bicarbonate, and showed remarkable and rapid improvement. Subsequent investigation of the electrolytes revealed marked improvement within 24 hours and by the fifth hospital day they were completely normal. Radiological examination of the kidneys revealed no abnormality. The child did well and was discharged from the hospital nine days after admission, weighing 6 lb. 3 oz.

This case illustrates the dangers that may be associated with the administration of a formula high in solute. Pratt and Snyderman[17] have shown that premature infants are particularly prone to the development of hypernatremia as a consequence of high solute feeding. In infants the insensible water loss through the skin is proportionately greater than in the older child because of the relatively greater body size. Elevated environmental temperature, overbreathing, or diarrhea increase the water loss and thus decrease the amount available for solute excretion. The obligatory loss in the urine due to a high solute load is thus more likely to produce dehydration and/or hypernatremia. Under normal circumstances the kidneys of full-term infants can handle the extra load of solute provided by certain unmodified milk formulas; this does not seem to be the case with the immature kidneys of the premature infant.

It has been stressed by the Committee on Nutrition of the American Academy of Paediatrics that concentrated feeding mixtures consisting of cow's milk, if not supplemented by additional water, provide a smaller margin of safety against heat stress than do more dilute feedings.

It is therefore evident that the enthusiastic administration of certain special feeding preparations, involving increased protein intake, to young infants may be extremely hazardous.

Lactic Acid Milk Formulas

Lactic acid milk has been used as a special feeding for sick infants for many years. Recently this type of feeding has been shown to produce marked and severe metabolic acidosis when administered to prematures. Goldman *et al.*[18] in 1961 compared lactic acid evaporated milk and evaporated milk feedings in premature infants. Sixteen premature infants fed a proprietary lactic acid milk

preparation for seven to 10 days were compared to 16 controls fed non-acidified half-skimmed milk. There was less weight gain in the experimental group (6.4 g./kg./day) than in the controls (14.0 g./kg./day). The poor weight gain in the experimental group was associated with a metabolic acidosis. In some cases there was a fall in CO_2 from 20.0 to 12.0 mEq./l. and in others from 22.1 to 13.6 mEq./l. The physiological explanation, according to these authors, was that there had been an increased excretion in the urine of some of the ingested lactic acid as lactate, leaving an excess of hydrogen ions.

It seems, therefore, that the feeding of lactic acid milk formulas to young premature infants is associated with a risk which increases in proportion to the excess of lactic acid.

Excessive Sodium Chloride Intake

Recently publicity was given in the daily press and national magazines to the tragic story of the death of newborn infants following the accidental administration, in the feeding, of large amounts of sodium chloride. Dahl, Heine and Tassinari[19] have studied the possible role of high sodium chloride intake in the diet of infants in the Western hemisphere and its relationship to hypertension in the adult. Experiments were carried out in which rats were chronically fed a diet high in sodium chloride. If high salt intakes were initiated at the time of weaning (approximately three weeks of age) the rats were more prone to develop hypertension than were older rats maintained on the same high sodium diet. Dahl, Heine and Tassinari[19] suggested that the modern diet of young infants may contain concentrations of sodium chloride so high as to result in average daily intakes comparable, on a body-weight basis, to the highest reported intake in man (27 g./day), as may occur among the northern Japanese. In this latter group hypertension is common and severe. Infants in the United States between the ages of 1 and 3 months may receive supplementary proprietary foods in which the sodium content is high. The same authors[19] have demonstrated that in rats the consumption of a diet high in sodium chloride during early life can induce hypertension which becomes self-sustaining after the extra sodium chloride is removed from the diet. Although some of the figures cited in their article may be questioned, their work should move many physicians to consider the possible latent effects of prolonged excessive amounts of salt in the diet, particularly during the neonatal period.

Protein

The amount of protein required by young infants has not yet been accurately determined; indeed, the amount of protein recommended for these infants per kg. body weight per day has decreased over the years. Generally speaking, harmful consequences have not been observed with increased protein in infant feedings except when water is lacking for the excretion of accumulated nitrogenous wastes *(vide supra)*.

Omans *et al.*[20] reported a long-term study of protein intake and growth in premature infants. The babies grew as well on formulas that contained 3.0 g./kg./day as on formulas containing 8.0 g. protein/kg./day. Kennedy,[33] in experiments on young rats, found that pathological lesions ordinarily seen in the kidneys of aged rats appeared at a much earlier age when as little as a twofold increase in protein load was instituted. Since there appears to be no advantage in feeding large amounts of protein to young infants, consideration should perhaps be given to the harmful effects of excess intake and to the advisability of prescribing the minimal amount of protein consistent with normal growth.

Amino Acid

The addition of amino acid supplements to certain foods has been suggested for some time on the grounds that a balanced intake of essential amino acids must be provided. If the ingestion of certain amino acids is excessive the body can adapt within certain limits but the margin of safety is very much less than is the case with vitamins.

In 1955, Albanese *et al.*[21] suggested that the addition of lysine to cow's milk formulas given to malnourished infants resulted in better weight gain and a higher concentration of serum protein and hemoglobin. The use of lysine was advocated by many pediatricians without clear evidence of its benefits. A subsequent review emphasized the possible hazards of such supplementation in producing imbalances in the body's store of other amino acids by altering the requirements for these other amino acids.[22]

A number of examples of amino acid imbalances and toxicities have recently been reviewed by Harper.[23] The question of amino acid supplementation has not been resolved, but there appears to be sufficient evidence to suggest that supplementary administration of specific amino acids may be of questionable value and probably should be avoided because of the amino acid imbalance that may result.

Fat

Atherosclerosis has been observed in the young as well as in the old. Moon[24] described early signs of arteriosclerosis as constant findings in the coronary arteries of young infants. VanBelle, Hartroft and Donahue[25] have reported evidence which confirms these findings. Lesions of the coronaries and other arteries of infants, suggestive of the early stages of atheroma, were seen as early

Canad. Med. Ass. J.
Oct. 23, 1965, vol. 93

as 3 days of age. The relationship of the ingestion of saturated fatty acids and cholesterol to atherosclerosis is still unclear, but sufficient evidence exists to warrant further consideration of the relationship between dietary factors and atherosclerosis in infants. The high intake of animal fat by infants, in the form of milk and eggs, and the findings of apparent early arterial lesions suggest that degenerative vascular disease may well be best treated in the future by adjustment of prenatal and neonatal diets. A reduction of certain foods in the mother's diet during neonatal and even fetal life may provide at least partial prophylaxis against atherogenesis.

Early and Excessive Feeding of Young Infants

During the past decade an increasing tendency has been displayed by many physicians, and particularly pediatricians, to introduce strained foods into the diet of infants within the first few weeks or even days of life. It is not unusual for a 4-week-old infant to receive a full diet of milk, infant's meats, fruits, vegetables and cereal supplements and vitamins. The possible hazards of this type of program include an increasing incidence of food allergy and harmful effects of excessive growth and weight gain. Food intake alone can profoundly affect the size and the rate of growth.

Widdowson and McCance[26] studied the effect of the introduction of early feedings on growth. Newborn rats were separated into litters of three and litters of 16 and 20. Rats suckled in litters of three weighed two to three times as much at weaning and their bodies contained four times as much fat as did the rats suckled in litters of 16 and 20. The overfed animals grew fatter and there was a more rapid increase in length and an earlier skeletal maturation than in the underfed rats. Hence it would appear that in rats early and excessive feeding results in accelerated growth which may in turn have deleterious effects.

Aside from the possible increased risk in terms of food allergy and excessive growth, the practical aspect of the added budgetary expense of canned foods should be considered. In contrast to these potentially harmful effects, the early feeding of a "full" diet would seem to have no benefit over the judicious use of foods that will ensure an adequate supply of vitamins C and D, and, at a slightly older age, of iron. There appears to be no justification for the introduction and excessive administration of strained foods to infants under 3 months of age.

Minerals

(a) *Calcium*

In 1961 Beal[27] reported that healthy infants and children in the United States ingested between 1000 and 1800 mg. of calcium a day. It has also been suggested that children can grow normally on 300 mg. of calcium a day.[28] The calcium intake for young infants presently recommended in the Canadian Dietary Standard is 500 mg. a day.[29] In the past, large intakes of calcium have been recommended even though little accurate information was available to support such recommendations. For this reason, consideration should be given to the possible harmful consequences of excess calcium. Idiopathic hypercalcemia is a condition described primarily in England and Switzerland. It is characterized by lethargy, anorexia, interference with growth, hypotonia, polyuria, and elevated calcium levels in the blood occurring in 3- to 12-month-old infants. It is believed to be related in part to excess vitamin D administration and possibly to excess calcium ingestion.[30]

This condition may be "triggered" during the first one or two months of life and in this respect a preventive dietary program may be important. In the light of available evidence it is unwise for the physician to encourage the ingestion of excessive amounts of calcium or vitamin D.

(b) *Phosphorus*

Excess phosphorus intake during early life is now recognized as a hazard to small infants. Tetany of the newborn, resulting in seizures during the first two weeks of life, and associated with low calcium and elevated phosphorus levels in the blood, is well recognized. The cause of this disorder is believed to be the presentation of an excessive phosphate load to the immature kidneys as a result of the use of a cow's milk formula, associated with a transient hypoparathyroidism in the neonate.

TABLE II.—COMPARATIVE ELECTROLYTE CONTENT OF HUMAN AND COW'S MILK*

	Cow's milk	*Human milk*
Total minerals (g./100 ml. whole milk)	0.72	0.21
Electrolytes		
Calcium (mEq./l.)	62.4	16.5
Potassium (mEq./l.)	35.3	14.1
Sodium (mEq./l.)	25.2	6.5
Magnesium (mEq./l.)	9.9	3.3
Chloride (mEq./l.)	29.0	12.1
Phosphorus (mg./100 ml.)	96.0	15.0

*Calculated from data reported by Macy, I. G., Kelley, H. and Sloan, R.: A compilation of the comparative composition and properties of human, cow and goat milk, colostrum and transitional milk, National Research Council, National Academy of Sciences, Washington, 1950, p. 63.

Table II illustrates the large amount of phosphorus in cow's milk as compared to human milk. Gittleman and Pincus[31] have studied the incidence of hypocalcemia in newborns and have described the relationship between hypocalcemia and various milk formulas including whole cow's milk, evaporated milk and human milk. In a study of 27

to 41 infants receiving one or other of these formulas the percentage of infants developing hypocalcemia was 13.3, 24.4 and 0.0, respectively. It is evident that hypocalcemia in infants receiving human milk is extremely uncommon in comparison to those fed whole cow's milk or evaporated milk. Recently low-solute-milk feeding preparations have been advocated for young infants, partly because of the lower concentration of phosphorus in such feedings, which simulates that of human milk.

Whether permanent damage or other sequelae may follow in such cases is not known, but the dangers of phosphorus excess should be considered, and if possible prevented, particularly in the premature or small full-term infant.

SUMMARY

The hazards of excessive administration of certain nutrients during the neonatal period are briefly discussed. Only a limited amount of information is available concerning the effect on the fetus of maternal nutritional excesses. The physician should be interested not only in certain anatomical abnormalities, but also in the possibility that altered nutrient requirements in the neonate may result from exposure *in utero* to high nutrient concentrations which may produce certain "conditioned" deficiencies. In the light of the recognized hazards of certain nutritional excesses and the potential hazards of others, levels for infant feedings and for prenatal and infant vitamin and mineral supplements should not exceed levels that fulfill the recommendations of the national dietary standards.[29, 32] There is little or no evidence that intakes above these levels are beneficial and they may in fact cause harm.

REFERENCES

1. YOUNG, C. M.: *Canad. Med. Ass. J.*, **93**: 900, 1965.
2. EBBS, J. H.: *Ibid.*, **46**: 1, 1942.
3. BURKE, B. S. *et al.*: *Amer. J. Obstet. Gynec.*, **46**: 38, 1943.
4. BURKE, B. S. *et al.*: *J. Nutr.*, **38**: 453, 1949.
5. MCGANITY, W. J. *et al.*: *Amer. J. Obstet. Gynec.*, **67**: 501, 1954.
6. WARKANY, J.: *J. A. M. A.*, **168**: 2020, 1958.
7. TOMPKINS, W. T. AND WIEHL, D. G.: *Amer. J. Obstet. Gynec.*, **62**: 898, 1951.
8. STIMMLER, L., BRAZIE, J. V. AND O'BRIEN, D.: *Lancet*, **1**: 137, 1964.
9. BAUMGARTNER, L. *et al.*: *Pediatrics*, **6**: 329, 1950.
10. BOUND, J. P. AND TELFER, T. P.: *Lancet*, **1**: 720, 1956.
11. LUCEY, J. F.: *Pediat. Clin. N. Amer.*, **8**: 413, 1961.
12. DYGGVE, *Acta Paediat. (Stockholm)*, **49**: 230, 1960.
13. HUNT, A. D., JR. *et al.*: *Pediatrics*, **13**: 140, 1954.
14. American Academy of Pediatrics. Committee on Nutrition: *Ibid.*, **31**: 512, 1963.
15. GARCIA, R. E. *et al.*: *New Eng. J. Med.*, **271**: 117, 1964.
16. Nutrition. Too much of a good thing: *Time*, **84**: 59, December 11, 1964.
17. PRATT, E. L. AND SNYDERMAN, S. E.: *Pediatrics*, **11**: 65, 1953.
18. GOLDMAN, H. I. *et al.*: *Ibid.*, **27**: 921, 1961.
19. DAHL, L. K., HEINE, M. AND TASSINARI, L.: *Nature (London)*, **198**: 204, 1963.
20. OMANS, W. B. *et al.*: *J. Pediat.*, **59**: 951, 1961.
21. ALBANESE, A. A. *et al.*: *Amer. J. Clin. Nutr.*, **3**: 121, 1955.
22. Editorial: *Pediatrics*, **17**: 965, 1956.
23. HARPER, A. E.: Amino acid toxicities and imbalances. *In*: Mammalian protein metabolism, Vol. 2, edited by H. N. Munro and J. B. Allison, Academic Press Inc., New York, 1964, p. 87.
24. MOON, H. D.: *Circulation*, **16**: 263, 1957.
25. VANBELLE, J., HARTROFT, W. S. AND DONAHUE, W. L.: Early atheromatous lesions in aortas and coronary arteries of infants and children, paper presented at the Canadian-United States Conference on Nutrition, Toronto, September 14 and 15, 1964.
26. WIDDOWSON, E. M. AND MCCANCE, R. A.: *Proc. Roy. Soc. [Biol.]*, **152**: 188, 1960.
27. BEAL, V. A.: *Amer. J. Pub. Health*, **51**: 1107, 1961.
28. World Health Organization. F.A.O./W.H.O. Expert Group: *W.H.O. Techn. Rep. Ser.*, **230**: 1, 1962.
29. Canada. Department of National Health and Welfare. Information Services Division: Dietary standards for Canada recommended by Canadian Council on Nutrition, May, 1963 (Canadian Bulletin on Nutrition, Vol. 6, No. 1), Queen's Printer, Ottawa, 1964.
30. HAWORTH, J. C.: *Canad. Med. Ass. J.*, **80**: 452, 1959.
31. GITTLEMAN, I. F. AND PINCUS, J .B.: *Pediatrics*, **8**: 778, 1951.
32. National Academy of Sciences—National Research Council, Food and Nutrition Board. Recommended dietary allowances, 6th rev. ed., 1964; Publication No. 1146, National Academy of Sciences, National Research Council, Washington, D.C., 1964.
33. KENNEDY, G. C.: *Brit. Med. Bull.*, **13**: 67, 1957.

PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

SOME INSTRUCTION MIGHT BE GIVEN

It is very unsatisfactory to be told by the philosophers that these things are appearances only; that mind not only does not but *cannot* act on body, nor can the body act on mind, when all the time we see, apparently, both these interactions proceeding continually before us. And if we, the orthodox practitioners, are in this outer intellectual darkness, what must be the state philosophically of those persons known as "faith healers" and "Christian scientists", whose whole position is that the mind can profoundly affect the body even to the extent of dismissing the existence of bodily pain: these persons must be lost indeed.

While the gulf gets wider between the medical interactionists and the philosophical parallelists, a school of medical psychologists has arisen in Vienna under the leadership of Dr. Sigmund Freud which takes into consideration not only conscious but unconscious states as causal. By what is called psycho-analysis, Freud has discovered that many dreams and many cases of hysteria are produced by the emergence of some subconscious state into the conscious level—a subconscious state usually of a disagreeable kind that had on that account been suppressed.

According to Freud all our mental states have emotional colourings; those that are pleasant are fostered or retained, those that are unpleasant are suppressed or relegated to the subconscious realm. In conditions of relaxed attention, sleep, illness, fatigue, etc., the subconscious emerges into the conscious and there, as a real cause, is responsible for the imagery of dreams, the characteristics of hysteria, as well as all sort of inadvertencies, mistakes, slips of the pen and tongue. A great deal of hysteria, according to Freud, is the result of some one or more suppressed because disagreeable mental experiences working away in the subconscious realm, with the results that are disastrous to the health of the central nervous system. One can readily see that here we are on ground common to psychology, ethics, religion and philosophy—certainly the region of the non-material in medicine; this is one of the latest findings of medical science; it is distinctly non-materialistic, and it recognizes the psychic as causal.

The physician should be more than ever versed in psychology, not only to comprehend the phenomena of undoubted insanity, but in order intelligently and effectively to apply the methods of psychotherapeutics which, although caricatured by "cranks", are based on vital and important truths. In exactly the same category stands hypnotism, a valuable therapeutic condition if rightly employed in suitable cases. "Personal magnetism", the influence of the physician's character, has no meaning if the mental is not causal.

I have no desire to add a single subject to the already large number in the medical curriculum, but I do think that between the physiologist, the professor of medicine and the lecturer on insanity, some instruction might be given on the influence of mind on body and body on mind. —D. F. Harris, *Canad. Med. Ass. J.*, 5: 870, 1915.