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Point of View

Human Pregnancy Nutrition: An Examination of Traditional Assumptions

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Summary: The traditional assumptions with reference to nutrition in human pregnancy which dominate clinical thinking and practice are critically reviewed.

The single most important environmental factor which determines the outcome of human pregnancy is maternal-fetal nutrition. The nutritional status of a woman at the time of implantation of the fertilized ovum directly determines whether this process will result in a functional, healthy placenta capable of carrying the fetus to term. The nutritional status of the pregnant woman during the early weeks of embryological development determines whether this process will take place in a normal fashion. And finally the nutritional status of both the pregnant woman and her fetus during the last half of pregnancy determines to a great extent the health of both and the birth length and weight of the infant.

Obvious as these facts now appear to be on the surface, they have not been recognized at this time by any significant number of academic obstetrical authorities in Western European medical culture. Confronted with a large number of common pregnancy complications among women and girls in poverty and ignorance, Western European obstetricians have contented themselves with idealistic speculations and assumptions which have long obscured their clear perceptions of the role of malnutrition in human reproduction. Edward Mellanby in 1933 presented a concrete definition of malnutition in human pregnancy:

> "I have attempted to show why nutrition is the most important of all environmental factors in child-bearing, whether the problem be considered from the point of view of the mother or that of the offspring. Both may suffer severely under conditions of malnutrition. *Malnutrition* in this sense does not mean an insufficiency of energy-bearing foods, but a deficiency of factors, usually only necessary in small amounts, which are nevertheless *essential* both to mother and offspring."

The purpose of this paper is to examine some of the common traditional assumptions and speculations regarding human pregnancy nutrition and to show how they continue to block understanding and scientific practice in this vital area of human health.

Human Fetus as a Parasite with Magical Placenta

In spite of Mellanby's call for application of scientific nutrition in human reproduction, it is still widely assumed by obstetricians that the human fetus is a parasite capable of taking all the essential nutrients it requires for optimal development and growth *at the expense of the mother regardless of maternal dietary intake and nutritional status.* The placenta has been considered to have special magical powers to extract nutrients from the maternal blood using esoteric "transport mechanisms" unrelated to basic physiologic processes operating in other body tissues. This speculation arose at a time when there was no clear understanding of the anatomy and physiology of the maternal-placental circulations. It has recently been restated by Page (1969):

> "For the most essential nutrients for growth, the placental membrane develops active transport systems which are capable

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of draining the host of vitamins, minerals, carbohydrates, and amino acids even to the detriment of a starved mother."

Now we know that the fetal capillary bed is simply immersed in a constantly moving stream of maternal blood, that the intervillous space is fed by jets of arterial blood arising from the uterine arterioles and drained by uterine veins and venules, and that from the point of view of maternal physiology this system functions as an arterio-venous shunt. There is no evidence to suggest that any other than known physiological processes regulating blood flow through vessels and transfer of water, gases, solutes, electrolytes, etc. across capillary walls are in operation within the placenta.

Since the uterine and ovarian arteries which supply the placenta with maternal blood arise from the maternal arterial circulation, it becomes obvious that the fetal capillary system must share nutrients with all maternal tissues supplied by means of other capillary systems, and in a sense the fetus must compete with all maternal tissues for essential nutrients. The situation would be different if the maternal placental blood supply were derived directly from the hepatic vein.

With these basic relationships in mind it is easier to understand how maternal malnutrition results in fetal malnutrition. If early in pregnancy placentation is impaired by maternal malnutrition of severe degree, spontaneous abortion and/or congenital fetal anomalies may ensue (Nelson, 1960; Thiersch, 1960; Hughes, 1961; Schull, 1961). With lesser degrees of malnutrition the blighted or stunted placenta may sustain fetal life but limit fetal growth. It is common to observe a low birth weight infant (less than 2,500 g.) delivered at or near term with a small, infarcted, poorly developed placenta. This situation can be observed with and without metabolic toxaemia of late pregnancy (Rumbolz, 1961). Regardless of the placentation, the maternal intake of essential nutrients and adequate calories during the last trimester is still an important factor in determining infant birth weight and length as Iyengar (1968) has so clearly demonstrated.

It has recently become obvious that to ensure optimal fetal nutrition the dietary intake of the pregnant patient must be kept adequate until the onset of labour. We can now grasp the vital importance of vigorous treatment of nausea, vomiting, heartburn, indigestion, food fads, alcoholism and drug addiction, etc., which interfere with optimal maternal nutrition. We are now in a position to seriously challenge the almost universal practice among Western European obstetricians of maternal "weight control" during the last half of pregnancy.

Maternal "Weight Control" is a Scientific Goal in Human Prenatal Care

Apprehension about "excess weight gain" in human pregnancy stems primarily from certain misconceptions about the aetiology and pathogenesis of "toxaemia of late pregnancy" or "pre-eclampsia — eclampsia". Classical obstetrical thought has considered "excess weight gain" from over-eating as a possible *cause* of this dread disease which remains throughout this hungry world one of the major causes of both maternal and fetal deaths. "Obesity predisposes to toxaemia" is another way to state this speculation. What has been totally ignored here is the quality of the obese pregnant woman's diet. There is no longer any question that the obese woman who has adequate supplies of all the essential nutrients for her pregnancy will escape the disease entity I have termed metabolic toxaemia of late pregnancy (Brewer, 1969). It is the malnourished obese woman, the woman lacking essential nutrients, who develops this disorder. Tracy and Miller (1969) recently reported 48 "massively obese" pregnant women with weights reaching 250 pounds or more. Twenty-eight of these women showed no evidence of metabolic toxaemia; obviously obesity, per se, does not *cause* "toxaemia".

Operating independently of caloric intake is the factor of "weight gain" related to water and sodium retention. Here cause and effect have been totally confused. A sudden, rapid weight gain, as much as 15 to 20 pounds within a week, can occur in metabolic toxaemia of late pregnancy as the patient develops a generalized oedema related to malnutrition, hypoalbuminaemia, hypovolaemia, and haemoconcentration. This type of "weight gain" is *a result* of the underlying metabolic disease, *not a primary cause* of it. In the severely malnourished pregnant patient this type of weight gain

cannot be prevented by restriction of calories or sodium or by the use of saluretic diuretics, but rather by improving the maternal diet. Strauss pointed this out in 1935.

The irrationality and hazards of limiting "total weight gain" in human pregnancy can now be understood. If an arbitrary limit of 20 pounds is set, the healthy pregnant women with access to good foods often reaches the limit by the 30th week of gestation. She is then often told to starve herself (and her fetus) during the last 10 weeks during which the fetal demands for essential nutrients, particularly proteins, are rapidly increasing. There are also increased metabolic demands upon the maternal liver for conjugating and excreting placental steroids (Song and Kappas, 1968); this function is dependent upon adequate supplies of essential amino acids. There is no longer any rational basis for use of "weight control" and starvation diets in human prenatal care. Indeed, we have seen severe metabolic toxaemia and other nutritional complications of pregnancy in poorly nourished women and girls who failed to gain weight at all during pregnancy.

The Dangerous Sodium Ion

Margaret Robinson (1958) has offered us the most concrete clinical evidence that sodium chloride is not toxic, per se, to human pregnancy. Her classic work has largely been ignored because it challenges the ancient tradition that salt is in some way harmful to the pregnant woman. Here again cause has been confused with effect. In the malnourished patient with low serum colloid osmotic pressure, hypoalbuminaemia, hypovolaemia and haemoconcentration, and generalized oedema, an increased sodium intake will often increase the oedema. However, it must be recognized that the amount of sodium in the maternal diet is *not* the basic, primary cause of the hypoalbuminaemia and hypovolaemia. The scientific approach to prevent hypoproteinaemia in human pregnancy is to ensure adequate intake of high biological quality proteins and all essential nutrients involved in protein synthesis and metabolism. Indeed, a careful analysis of Robinson's work leads us to the conclusion that the observed superior reproductive performances of her patients on the "high salt diet" and the superior health of their infants were directly related to the fact that the "high salt diets" resulted in higher protein intakes. Pregnant patients put on "low salt diets" were advised to restrict their intakes of milk, salty cheeses, salty fish and meats; these same foods were encouraged for patients on the "high salt diets". Perhaps obstetricians have ignored Robinson's valuable contribution because she did not draw exactly the right conclusions from her own work. She emphasised that the pregnant woman and her fetus need "extra salt" to remain in good health; we know now that "extra salt" is not enough. The pregnant woman and her fetus must have all the essential nutrients and adequate calories, and sodium chloride is but one of the essential nutrients.

The Dangerous Oedema

Frank Hytten (1969) has recently shown that the majority of otherwise healthy pregnant patients who produce healthy, term infants, will develop oedema in the last half of pregnancy. We must term this "physiological oedema", and it most commonly involves the lower extremities, but it also is often generalized to involve the face and hands. In the lower extremities this is related to the increased venous pressure, to a slight lowering of colloid osmotic pressure, and probably to changes in the connective tissue ground substance so that more water is stored. This latter effect Hytten attributes to effects of the placental steroids. Hytten agrees with my contention that it is unscientific "to treat" this physiological oedema with saluretic diuretics.

How then is it possible to distinguish physiological oedema from pathological oedema associated with metabolic toxaemia, renal diseases, hepatic diseases, heart failure, etc.? The answer lies in a careful medical history and appropriate laboratory studies. The answer lies in a correct analysis of the pregnant woman's nutritional status and application of modern methods of differential medical diagnosis. The widespread use of saluretic diuretics for "the oedema of late pregnancy" has done far more harm to maternal-fetal and infant health than good. Saluretic diuretics often mask the onset of metabolic toxaemia in a poorly nourished patient; these drugs cause many undesirable side effects in both mother and fetus (Gray, 1968). They are absolutely contra-indicated in the severely toxaemic patient with hypoalbum-

inaemia, hypovolaemia and haemoconcentration because their use results in further haemoconcentration, hypovolaemic shock and, too often, maternal and fetal deaths (Brewer, 1962). The agressive promotion of saluretic diuretics by the private drug industry in the United States has blocked out understanding of the role of malnutrition in human reproduction by keeping obstetricians' minds focused on sodium, oedema and drugs. It is imperative that primary attention now be directed to essential nutrients, adequate calories — to the holistic view of human maternal-fetal nutrition (Brewer, 1967).

Pregnancy Stresses the Kidneys

Over 20 years ago Thomas Addis (1948) after many years of clinical observations of pregnant patients with chronic glomerular renal diseases recognized that this assumption is wrong. He pointed out that because of the utilization of amino acids by the growing fetus, placenta, and uterus during pregnancy, the kidneys actually have less work to do. The kidneys of the rat actually get slightly smaller during pregnancy while the liver gets larger. His observations of many patients with chronic renal diseases of varying degrees of severity led him to the conclusion that chronic renal disease has no adverse effects on pregnancy and on the other hand, pregnancy seems to have no adverse effect upon the course of chronic renal diseases. His observations were made on patients who were receiving good medical care and adequate nutrition. The widespread use of low sodium diets and saluretic diuretics has generally been harmful to patients with renal diseases, and in many instances iatrogenic metabolic toxaemia of late pregnancy has been produced in such patients. Addis correctly inferred that if any organ is "stressed" by pregnancy it is the liver, not the kidneys.

Toxaemia of Late Pregnancy is a Syndrome

When a pregnant patient develops in the last half of gestation symptoms such as headache, swelling, nausea, vomiting, spots before the eyes, stomach pain, loss of appetite, weakness, and clinical signs such as generalized oedema, proteinuria, arterial hypertension, coma, convulsions, congestive heart failure, pulmonary oedema, ascites, hypovolaemic shock, then the obstetrician begins to think in terms of "toxaemia of pregnancy" or "pre-eclampsia — eclampsia".

The recognition of the role of malnutrition in the pathogenesis of a specific disease entity, metabolic toxaemia of late pregnancy, now makes possible a more rational approach to the patient presenting clinically with the signs and symptoms listed above:

1. Disorders which only occur in pregnant patients
   - A. Metabolic toxaemia of late pregnancy: mild, severe
   - B. Physiological oedema of pregnancy
   - C. Molar pregnancy with the toxaemia syndrome
2. Disorders which occur in pregnant and in non-pregnant patients
   - A. "Essential" hypertension
   - B. Other medical causes of hypertension (including renal and adrenal diseases)
   - C. Urinary tract diseases
   - D. Central nervous system diseases
   - E. Hepatic diseases
   - F. Congestive heart failure from other causes
   - G. Diabetes mellitus
   - H. Malnutrition with hypoalbuminaemia and oedema as the only clinical signs
3. Combinations of disorders 1 and 2 occurring in the same patient (Rippman, 1969).

A key to understanding this "enigmatic" problem lies in recognition of the unscientific assumption that the patient with a chronic hypertension present in the non-pregnant state is prone to develop a superimposed "toxaemia of pregnancy". The chronic hypertensive patient develops a superimposed metabolic toxaemia *only when she is malnourished.* The chronic hypertensive patient may also develop physiological oedema of pregnancy or proteinuria from urinary tract infection. It is now apparent that accurate differential diagnosis is not an easy matter in many cases, especially when we encounter the patients for the first time late in pregnancy after these signs and symptoms are well established.

There are a large number of speculative assumptions regarding the pathogenesis of "toxaemia of late pregnancy". Many of these I have discussed in detail in my book (Brewer,

1966). All of these ideas function to block out of the obstetrician's mind the role of malnutrition in human reproduction:

1. A reduction in utero-placental blood flow of the maternal circulation is a *primary event* in the pathogenesis of the disorder.
2. Maternal arteriolar vasospasm causes "hypoxia" and tissue damage to both fetus and maternal tissues.
3. The disorder is in some vague manner related to primaparity, twin gestation, polyhydramnios, obesity, diabetes mellitus, chronic hypertension, chronic renal diseases, the seasons and atmospheric conditions.
4. Glomerular lesions observed in patients with the severe disease are the cause of the clinically impaired renal function.
5. Liver function is not significantly impaired in patients with severe disease.
6. Lowered serum proteins, especially hypoalbuminaemia, play no role in producing the generalized oedema in the severely toxaemic patient with hypovolaemia and haemoconcentration.
7. "Toxaemia of late pregnancy" can be prevented by saluretic diuretics, restriction of dietary sodium and "weight control" beginning early in gestation.
8. Reduction in urinary excretion of placental steroid metabolites is always related to "placental insufficiency", a decrease in placental production of oestrogens and progesterone.

There is at present a growing body of evidence to support the contention that not a single one of these speculations is valid in the sense of contributing real knowledge about how to prevent this disorder or how to treat it rationally.

There Are No Nutritional Complications of Human Pregnancy

It has been assumed for years by obstetrical authorities in the "industrially advanced" nations that malnutrition severe enough to cause diseases among pregnant women, their fetuses and infants, does not exist. This is the most rigid kind of class bias which has overtones of racism in many areas of the world. We have many workers to thank for basic clinical facts concerning the *protective role* of scientific maternal-fetal nutrition: (Ross, 1935; Strauss, 1935; Ebbs et al., 1942; Burke, 1943; Cameron and Graham, 1944; Ross, 1947; Ferguson, 1951; Hamlin, 1952; Robinson, 1958; Hibbard, 1964; Iyengar, 1968) and many others. Mellanby was concerned with the role of malnutrition in lowering the pregnant woman's resistance to common clinical infections such as puerperal sepsis; his theoretical conceptions have been amply confirmed by hundreds of workers throughout the world and summarized recently by Scrimshaw et al. (1968).

For over 6 years I have been carrying on a prenatal nutrition education project among medically indigent women and girls in the Richmond Health Center, Richmond, California. It has become obvious to me that an aggressive educational programme which teaches the prenatal patient with the authority of the physician what is a good, adequate diet for herself and for her fetus and how important this diet is for her own good health and the health and development of her infant, will eliminate the following complications: metabolic toxaemia of late pregnancy, abruptio placentae (atraumatic), severe nutritional anaemias, severe infections of kidneys, lungs, liver and uterus. With aggressive management of nausea, vomiting, anorexia, food fads, heartburn, failure to gain weight, and by abandoning the classical methods of "weight control" and low sodium diets and saluretic diuretics, it is possible to lower the incidence of low birth weight infants to less than 3%.

CONCLUSIONS

In those nations where food supplies are adequate, the application of modern nutrition science in human prenatal care can in a short time improve maternal and infant health dramatically. The recognition of the vital, protective role of scientific nutrition in human reproduction must motivate physicians, public health workers, and politicians in all nations to take steps to improve maternal-fetal nutrition among women in poverty and ignorance. This is a grave responsibility of medical science to the future generations of mankind; *it is a special responsibility of obstetrical science.*

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