Basal Metabolism in Nutritional Edema

By P. S. Venkatachalam, S. G. Srikantia and C. Gopalan

IT WAS first pointed out by Magnus-Levy[1] that there was a fall in energy metabolism in states of severe undernutrition. Benedict and his associates[2] confirmed the above observation in their studies on basal metabolism in induced semistarvation, where they found a mean fall of 15% to 20% in heat production. Studies on the effect of undernutrition on basal metabolism received particular attention during and immediately after World War II. The published work on this subject has been reviewed by Keys and co-workers.[3]

The fall in basal metabolism occurring in semistarvation could result from (1) a reduction in metabolizing tissue due to wasting and/or (2) a reduction in oxygen consumption per unit of metabolizing tissue. The object of the present investigation was to study the changes in basal metabolism in nutritional edema and to determine whether either or both of the above mentioned factors operated in determining its level in this condition.

Material and Methods

The patients studied in this investigation were male adults in advanced states of semistarvation. A detailed account of the clinical and biochemical features of the condition has been given elsewhere.[4]

The basal metabolism was determined in 10 cases immediately on admission and again after the disappearance of edema following on rehabilitation. The average time interval between the two determinations was 31 days. With the use of a Benedict-Roth metabolism apparatus, two runs of eight minutes each were recorded for each estimation. The mean of two readings on the same day, which did not in fact differ by more than 5%, was used for calculating the basal metabolism. Preliminary observations indicated that the training trend in these subjects was negligible and that the first determination could be taken as a reliable value. The body composition was also determined on the same day according to the method already described by us.[5] From the cellular solids thus estimated, the oxygen consumption per kilogram of cell solids was calculated. The body surface area was arrived at by the DuBois formula as expressed in nomographic form by Boothby and Sandiford.[6]

Results

Basal Metabolism Expressed in Terms of Surface Area

Expressed as calories per hour per square meter, the basal metabolic rate in 10 patients ranged from 23.3 to 34.4 calories at the time of admission, with an average of 27.96 calories. After rehabilitation the basal metabolic rate showed a

From the Nutrition Research Laboratories, Indian Council of Medical Research, Coonoor, South India.

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TABLE 1.—*Basal Metabolism in Cases of Nutritional Edema Before and After Rehabilitation*

Case no.	Basal metabolic rate: Calories per hour				Oxygen consumption per kg. of cell solids, cc. per minute	
	Absolute		Per sq. meter			
	Before	After	Before	After	Before	After
1	37.3	45.7	28.9	32.2	21.8	22.5
2	49.6	56.7	29.3	35.9	21.8	22.6
3	44.4	49.1	30.6	33.1	22.3	22.7
4	32.7	42.1	23.5	31.1	22.4	22.5
5	44.5	47.4	27.9	32.6	21.6	22.5
6	47.2	57.3	30.9	38.9	21.6	22.3
7	36.3	43.4	23.7	32.4	20.8	23.3
8	44.8	47.5	34.4	34.6	22.2	21.9
9	36.3	41.2	27.1	33.0	21.2	21.8
10	34.8	40.7	23.3	30.2	23.0	22.6

rise in every case, the values ranging from 30.2 to 38.9 calories, with an average of 33.4 calories. The basal metabolism estimated on admission was, therefore, found to be about 20% lower than the reported average of 35 calories per square meter per hour for normal Indian adults.[7] After treatment the basal metabolic rate was higher than at the time of admission but had not quite come up to the normal level except in one case. There was an absolute rise in basal metabolism in every case at the end of treatment. Since there was also a concomitant reduction in body surface area after the disappearance of edema, the basal metabolic rate showed the appreciable rise reported above.

Basal Metabolism Expressed in Terms of Cell Solids

The oxygen consumption per kilogram of cell solids ranged from 20.8 to 23.4 cc. per minute at the time of admission and from 21.8 to 23.3 cc. per minute after the disappearance of clinical edema (Table 1).

DISCUSSION

It will be seen from the above data that there is a rise of nearly 22% after treatment in the basal metabolic rate, as correlated to the surface area. On the other hand, the oxygen consumption per kilogram of cell solids was found to be raised by less than 3% on the average after treatment. This rise is within the limits of experimental variation. An assumption made to arrive at the body composition during the edematous stage was that depot fat was completely absent. This assumption may not have been entirely valid, and may have resulted in a slight overestimation of cell solids and consequent underestimation of oxygen consumption per kilogram of cell solids in the edematous stage. The absence of any appreciable rise in oxygen consumption per kilogram of cell solids in spite of this, is convincing evidence that there is no reduction in oxygen consumption per unit of metabolizing tissue in states of semistarvation.

A possible relationship between heat production and mass of "active tissue" was thought of by Rubner[8] long ago. Miller and Blyth[9] showed that the "lean body mass" was a reliable reference standard for the estimation of basal metabolic rate. Beattie and Herbert[10] pointed out that the expression of metabolic rate in terms of surface area in semistarvation led to results which were not a true index of the rate of cellular oxidation, since marked changes in the ratio of active to inert material take place in conditions of extreme weight loss.

Keys and co-workers[3] found that the mean oxygen consumption per kilogram of active tissue showed a decline of 15.5% after 24 weeks of semistarvation. By "active" tissue, however, these workers actually meant the entire cell mass, including both the cellular solids and intracellular water. The decline in oxygen consumption per kilogram of "active" tissue in semistarvation reported by these authors may well be due to increased cellular hydration. In cases of advanced semistarvation investigated here, it was concluded that there was excessive intracellular hydration.[5] Though the subjects studied by Keys and co-workers might not have been such advanced cases, it is possible that even in them, intracellular hydration might have sufficiently increased to cause an apparent decline in the oxygen consumption per kilogram of "active" tissue. Beattie and Herbert[10] using a method similar to that employed by Keys and co-workers for the determination of "active" tissue, observed in all but three of their subjects of semistarvation, no change in oxygen consumption per kilogram of "active" tissue. In the three subjects, however, the body weight loss exceeded 26% and in these there was an appreciable reduction in oxygen consumption per kilogram of "active" tissues. Presumably, in the latter there was excessive intracellular hydration.

Summary

1. At the height of edema, cases of semistarvation had subnormal basal metabolic rate.

2. There was an absolute rise in basal metabolism and a fall in body surface area after treatment resulting in an appreciable rise in basal metabolic rate per square meter per hour after treatment.

3. The reduction in basal metabolism at the height of edema was almost entirely due to a reduction in the quantity of metabolizing tissue and not to any reduction in the oxygen consumption per unit of metabolizing tissue.

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