ALTHOUGH IT IS widely believed that physiological experiments have proven the absence of any metabolic defect in the etiology of obesity, a clear-cut analysis of the manner in which they are presumed to have done so is difficult to find. Discussions of the subject often emphasize that the energy intake must exceed the expenditure when obesity is developing; but this elementary knowledge, deducible from the law of conservation of energy, needs no experiment. The question on which an experimental answer has been sought is whether the positive caloric balance associated with obesity arises from causes extrinsic to the metabolism or from some intrinsic metabolic defect.

Many experiments have failed to reveal any metabolic alteration from the normal in obesity. Basal metabolism tests have ruled out disease of the thyroid gland; and many explorations of the intermediary metabolism have given negative results. Since, however, these do not rule out the possibility of some still undiscovered defect, studies of the energy exchange have attempted to answer the essential question whether or not a metabolic aberration can, at all, account for obesity.

Studies of the energy exchange might well be expected to answer this crucial question by showing whether the excessive energy storage of the obese is causally dependent on the balance between energy intake and expenditure or if it maintains a significant degree of independence of that balance. In either case, the necessities of the energy equation,

energy intake = energy storage + energy expenditure,

must be fulfilled. In this equation, energy storage may be positive or negative in sign. Experimental investigation of the matter has been attempted by altering the energy intake and observing the effect of this on energy storage and expenditure. If the excessive energy stores of the obese depend passively on the balance between energy intake and expenditure, caloric restriction should be followed by utilization of stored energy in an amount equivalent to the calorie deficit of the diet, with no more decline in energy expenditure than would be anticipated from the reduction in body weight. If, on the other hand, the excessive energy stores arise from some intrinsic metabolic aberration, they would be expected to show a significant degree of independence of the balance between energy intake and energy expenditure; caloric restriction, in such a case, would not be followed by utilization of stored energy in an amount equivalent to the calorie deficit of the diet, and the energy expenditure would show a significant decline.

CONTROL STUDIES ON NORMAL INDIVIDUALS

Two types of energy balance studies on normal individuals serve as controls for studies on the obese. In one type, individuals of normal weight have been subjected to subcaloric diets; and in the other, normal-weight individuals have first increased their weight by purposeful overfeeding and then lowered their caloric intake. The effects of caloric restriction on metabolically normal individuals under these two circumstances form a background for evaluating the effects of caloric restriction on individuals in whom obesity has arisen, as it ordinarily does, spontaneously. Table 1 shows the effects of caloric restriction on the basal energy expenditure of individuals of normal weight subjected to undernutrition.

### Table I

<table>
<thead>
<tr>
<th>Source</th>
<th>Decline in basal calories</th>
<th>Decline in B.M.R.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benedit (Squad A) (1)</td>
<td>19 per cent</td>
<td>16.2 per cent</td>
</tr>
<tr>
<td>Benedit (Squad B) (1)</td>
<td>32 per cent</td>
<td>27 per cent</td>
</tr>
<tr>
<td>Taylor and Keys (2)</td>
<td>39 per cent</td>
<td>31.2 per cent</td>
</tr>
</tbody>
</table>

It is apparent from the table that a varying but significant decline in energy expenditure occurs when people of normal weight are subjected to subcaloric nutrition. This would indicate that the normal energy stores of the body maintain a considerable degree of independence of the energy balance. It would seem to coincide with evidence from other sources that the normal energy stores of the body are regulated by intrinsic metabolic mechanisms. They are not yielded for energy in an amount equivalent to the calorie deficit of a subcaloric diet and, as a result, there is seen the "specific reduction in metabolism coincident with undernutrition".

Table 2 shows the effects of reduction in the caloric intake on the basal energy expenditure of two normal individuals who first increased their weight by purposeful overfeeding.

### Table II

<table>
<thead>
<tr>
<th>Source</th>
<th>B.M.R. before overfeeding</th>
<th>B.M.R. after overfeeding</th>
<th>B.M.R. during caloric restriction</th>
<th>Decline from highest level</th>
<th>Decline from original level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gulick (5)</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Wiley &amp; 10%</td>
<td>-4.3%</td>
<td>-11.3%</td>
<td>7.2%</td>
<td>1.3%</td>
<td></td>
</tr>
<tr>
<td>Newburgh (6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In Gulick’s case, which showed a remarkable steadiness of the metabolic rate, there was a weight gain of...
26 pounds during the course of 11 months before caloric restriction. In the case of Wiley and Newburgh there was a weight gain of 9.7 pounds during 2 weeks of very intensive overfeeding before the reduction in caloric intake. The rise and fall in basal metabolism in this case may reasonably be attributed to the temporary increase in metabolism representing energy utilized in the metabolic work of adding flesh to the body; during the rapid fattening of steers the basal metabolism may rise as much as 36 per cent (7). The more prolonged experiment of Gulick would seem to have eliminated this temporary effect by allowing time for a levelling off of the weight at the higher level. The evidence from these experiments would seem to indicate that, in metabolically normal individuals who have increased their weight by purposeful overfeeding, the excessive energy stores are passively dependent on the balance between energy intake and expenditure; for these stores respond to caloric restriction by yielding their substance for energy in an amount equivalent to the calorie deficit of the diet, with the result that no significant decline in energy expenditure occurs. Although the normal energy stores of these subjects were presumably maintained by the same intrinsic mechanisms which operate in other normal individuals, their excess stores were obviously dependent on causes extrinsic to the metabolism. These individuals, when allowing the appetite to regulate the food intake, maintained constant weight at a normal level.

Studies of the Energy Exchange in Obesity

If the excessive energy stores in common obesity, which ordinarily arise spontaneously, show a passive dependence on the energy balance, as do the excessive stores of normal individuals in purposeful overfeeding experiments, their maintenance must be attributed to causes extrinsic to the metabolism, and the cause of obesity must be simple overfeeding. If, on the other hand, the excessive energy stores of the obese show a significant degree of independence of the energy balance, as do the normal energy stores of the body, their maintenance must be attributed to an intrinsic mechanism, and the cause of obesity must be a metabolic aberration involving the regulation of fat storage.

Experiments showing the effects of caloric restriction on the obese have been of two types. In one, efforts have been made to determine the amount of body substance oxidized; and in the other, efforts have been made to determine whether or not there has been a decline in the energy expenditure. Since oxidation of body substance offsets a decline in the energy expenditure, the two quantities are inversely related.

In Newburgh's studies (8), diets with a known caloric deficit were given to the obese; and, on the assumption that body fat contains 10 per cent water, the caloric value of body substance oxidized was calculated from the weight loss. It appeared from the calculations that the caloric deficit of the diet was completely compensated for by utilization of stored energy, and the conclusion was drawn that the energy expenditure remained constant. These results seemed to show that the excessive energy stores of the obese are passively dependent on changes in the energy balance and that "obesity is invariably the result of a disproportion between the inflow and the outflow of energy." Since, however, the experiments were not checked by actual determinations of the energy expenditure after caloric restriction, the results would appear to depend entirely on the value assumed for the water content of body fat. Lauter had determined this to be 10 per cent, but Bischoff had found it to be 29 per cent, Bozenraad 7 to 46 per cent, and Bozenraad had quoted others who found it to vary between 15 and 30 per cent (9). If the water content of body fat is higher than Newburgh supposed, a smaller quantity of stored energy must have been yielded by the fatty deposits than would be indicated by his calculations from the weight loss. In view of the uncertainty concerning the water content of body fat, it seems that no conclusions concerning the nature of obesity can be drawn from Newburgh's studies of the energy exchange.

Actual determinations of the energy expenditure of the obese after caloric restriction have shown that there is a significant decline. Table 3 gives the basic data from sources frequently referred to.

<table>
<thead>
<tr>
<th>Source</th>
<th>Decline in Basal Calories</th>
<th>Decline in B.M.R.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strong and Evans (10)</td>
<td>14 per cent</td>
<td></td>
</tr>
<tr>
<td>Brown and Olshon (11)</td>
<td>17 per cent</td>
<td></td>
</tr>
<tr>
<td>Lyon and Dunlop (9)</td>
<td></td>
<td>13 per cent</td>
</tr>
<tr>
<td>Keeton and Bone (12)</td>
<td>12.2 per cent (calculated from graph)</td>
<td></td>
</tr>
<tr>
<td>Moller (13)</td>
<td>12.5 per cent (calculated from graph)</td>
<td></td>
</tr>
</tbody>
</table>

In children, according to tables furnished by Mulier and Topper (14), the decline in B.M.R. is less, but the authors point out that the pelidisi of Pirquet is a better measure of energy expenditure in children than is the usual surface area rule. The evidence from the basic data of all of the investigators would seem to indicate that, in the adult obese individual, caloric restriction is followed by a significant decline in energy expenditure, for the decline is greater than the decline in either the weight or the surface area (9) (15).

Evaluation of the Results

The decline in energy expenditure of the obese during caloric restriction, though less than that of people of normal weight during subcaloric nutrition, stands in striking contrast to the lack of decline in the metabolism of normal individuals who have first increased their weight by purposeful overfeeding and then lowered the caloric intake. It would seem from these results that, although the excessive energy stores of the obese are yielded for energy in response to a negative energy balance more readily than are the normal energy stores of the body, they, nevertheless, show a significant degree of independence of the energy balance. They do not yield their substance for energy in an amount equivalent to the calorie deficit of the diet. The excessive energy stores of the obese, there-
fore, appear to resemble the normal stores in being maintained by intrinsic metabolic mechanisms. The evidence would appear to indicate that energy storage in the obese is regulated very much as it is in people of normal weight, but at an abnormally high level. This would seem to necessitate an explanation of obesity as due to some metabolic aberration involving the regulation of fat storage.

These generalizations concerning obesity are based on the averages found in the various studies. While there appears to be a considerable degree of uniformity in the averages derived from the basic data of the various investigators, a considerable range is seen among individuals within a particular study. Strang and Evans (10) for example, found declines ranging from 4 to 23 per cent in the basal caloric expenditure of the obese on low calorie diets. In Benedict's Squad A of individuals of normal weight, the decline in basal caloric expenditure during subcaloric nutrition was not far different, ranging from 9.5 to 26.6 per cent (16). There appear to be wide variations in the response of both normal-weight and obese individuals to subcaloric nutrition, some of each group showing a greater resistance to a negative caloric balance than others.

The variations among the obese might be explained in the following ways. In some of the subjects who display a considerable lability of the energy stores, the metabolism may be relatively normal, and the excess weight may be due largely to purposeful overfeeding, chiefly of carbohydrates, as it is in metabolically normal individuals who have subjected themselves to overnutrition experiments. On the other hand, the relative lability of the energy stores might be accounted for by an incipient failure of the fat storage mechanism, which would come to full realization in the diabetic state. In those obese subjects whose energy stores show a considerable resistance or stability, some metabolic aberration involving fat storage would be the most reasonable inference. The evidence, in any case, does not seem to support the extreme view that obesity is invariably due to simple overfeeding.

**Objections to the Evidence**

Although it would seem that energy balance studies have given valid evidence for some explanation other than simple overfeeding in a considerable proportion of obese subjects, there appears to have been some hesitancy in accepting this evidence, even by some of the investigators themselves. This body of evidence and its significance have been assailed in many various ways.

Strang and Evans (10) introduced a novel method of calculating metabolic rates of the obese, based on the ideal, rather than the actual weight. This caused the metabolism of the obese to appear hypernormal, and tended to minimize the significance of the actual decline in energy expenditure. The method was based on the assumption that adipose tissue is metabolically inactive and that its weight, therefore, should not be taken into account when metabolic calculations are made. The discovery that adipose tissue is metabolically active (3), however, would appear to rule out the validity of metabolic calculations based on ideal weights.

Keeton and Bone (12), in interpreting their results, assumed that a decline in energy expenditure is not significant as long as the metabolic rate does not fall below the limit of minus 10 per cent, which is widely accepted for clinical diagnostic purposes as the arbitrary low limit of normal. In physiological experiments, however, the significance of the results depends on measurements of the actual changes in variable quantities. Moller interpreted his one case in the same manner as Keeton and Bone.

Rynearson (17) in discussing the decline in energy expenditure, which he concludes is of some importance, points out that different investigators have found different results. Examination of the basic data of the various investigators, however, shows a remarkable uniformity of results and a difference only in some of their conclusions. In Moller's one case, there was a temporary though significant rise in the metabolism at the 19th week, coincident with a levelling off of the weight reduction curve which, according to the author's explanation, would indicate a departure from the diet at this time. It would seem that a decline in metabolism persists when dietary restrictions are adhered to.

**Adjustments in the Energy Balance Secondary to Primary Alteration in Energy Storage**

The basic data of those who have investigated the energy exchange in obesity would seem to indicate that, in a considerable number of cases, this condition arises from an intrinsic aberration in the regulation of energy storage. Certain results must, of necessity, follow from this: for it is evident from the energy equation, energy intake = energy storage + energy expenditure, that a primary increase in energy storage must be compensated for either by an increase in the energy intake or by a decrease in the energy expenditure. The forces influencing an increased energy intake would be felt subjectively as an increase in appetite but, if food is restricted, the adjustment in the energy equation must take place in the only way possible: through a decrease in the energy expenditure.

A primary increase in fat storage, with these secondary effects, has been found by Brooks in animal experiments. Some of his animals with obesity-producing lesions in the hypothalamus gained more weight than the normal controls, though the food intake was kept the same (18). As long as the food intake was kept at normal the basal caloric expenditure remained below normal (19). The animals readily ingested more food when it was provided, and their basal caloric expenditure then rose. They rapidly became obese; however, for the increase in energy expenditure did not keep pace with the increase in energy intake. The weight of such animals can be brought to normal by severe caloric restriction, but it invariably rises to a high level again when food restrictions are removed.

Similar circumstances in common obesity in humans are indicated by the studies of Brown and Öhlsön (11). Their obese subjects, after reducing, maintained their weight at a normal level by means of caloric restriction, but at the expense of a subnormal basal caloric expenditure. They maintained normal weight on a food
intake lower than that of other people of the same age and dimensions; and they characteristically experienced hunger. It would appear that when obese people reduce their weight by caloric restriction, the intrinsic metabolic aberration promoting excessive fat storage is still operative, producing an urge to increase the food intake and, if this is restricted, a decline in the basal metabolism.

**Significance of a Lowered Basal Metabolism**

In view of the hazards of the obese state, it might seem that the formerly obese individual might well afford to endure hunger and to accept the lowered basal metabolism as an inconsequential matter. It seems, however, that the significance of a lowered basal metabolism has been obscured by the wide normal range allowed for clinical diagnostic purposes. Du Bois states that the basal metabolism test has been useful chiefly in the diagnosis of disease of the thyroid gland (20). As long as the basal metabolism stays within the limits which usually exclude a diagnosis of thyroid disease, further thought of it is usually abandoned, perhaps on the ground that enough heat is being produced to keep the body warm. A more widely applicable significance of the basal metabolism was indicated by Benedict, who considered it to be a "very good index of the general state or level of vital activities" (21). The basal calorific expenditure represents the amount of metabolic work being accomplished by the body: and heat and to spare is ordinarily given off as an end product, very much as water over a dam runs downstream after turning the machinery of a mill. A decline in the basal energy expenditure following a decline in the food intake must be attributable to a decline in the amount of nutrient which the organism is able to convert into types of energy necessary for its vital activities. A great part of the basal energy expenditure represents energy utilized by the liver; and it seems that the basal energy expenditure must reflect, in large measure, the level of caloric nutrition in this organ. In view of the caloric needs of the liver for repair and for prevention of damage (22), a prolonged caloric shortage here, in many patients, can not be regarded lightly. Actually, there is hardly any evidence that caloric restriction produces, in the long run, the benefits which the obese are often led to expect (23).

**Treatment of Obesity**

There is no doubt that obesity constitutes a clinical problem of the highest importance; but, in many cases, treatment by caloric restriction does not appear to be a very satisfactory solution. It would seem that a rational treatment of obesity should be based on measures which take into account the pathologic physiology of this condition and which avoid a decline in the basal energy expenditure.

During the past half-century many investigators have opposed the concept of obesity as due to simple overnutrition; and a number of studies of the intermediary metabolism have, in fact, revealed variations from the normal in the obese (24, 25, 26). Work in this field has advanced rapidly since the introduction of isotopes in biochemical experiments (27); and it appears that the question of the specific nature of the intrinsic metabolic fault leading to excessive fat storage will soon be, if it is not already, clarified. There appears reason to believe that it is an enzyme block in the breakdown of carbohydrate at the pyruvic acid level. As a result of this block, it seems, much of the carbohydrate ordinarily ingested is converted to fat, while the presence of excess pyruvic acid in the tissues hinders the oxidation of fat (28).

If obesity can be caused in this way, restriction of carbohydrate, specifically, in the diet should make ineffective the metabolic aberration which promotes and maintains excessive fat storage, for there would be less pyruvic acid formed. The excessive energy stores should then yield their substance for energy, causing energy storage to become negative in sign in the energy equation,

\[ \text{energy intake} = \text{energy storage} + \text{energy expenditure} \]

The energy equation would then, of necessity, come into balance through an increase in the energy expenditure or a decrease in the energy intake, or both; and the normal relation of the food intake to energy needs (29) would be expected to allow for effective weight loss, with the intake of the non-carbohydrate elements of the diet, protein and fat, regulated entirely by the appetite.

An experiment which appears to have demonstrated this was performed at the Russell Sage Institute in 1928 (30, 31). Three subjects lost some weight on an ad libitum intake of lean and fat meat and they all showed an increase in the calorific expenditure during the period of weight loss. Table 4 shows these results.

**Table IV**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Loss of weight (kilograms)</th>
<th>Increase in basal calories</th>
<th>Increase in B. M. R.</th>
</tr>
</thead>
<tbody>
<tr>
<td>V. S.</td>
<td>4.2</td>
<td>8.9 per cent</td>
<td>7 per cent</td>
</tr>
<tr>
<td>K. A.</td>
<td>2.9</td>
<td>16 per cent</td>
<td>5 per cent</td>
</tr>
<tr>
<td>E. F. D. B.</td>
<td>2.8</td>
<td>16 per cent</td>
<td>6 per cent</td>
</tr>
</tbody>
</table>

None of these subjects was very much overweight. The weight loss occurred during the first month on the diet; and two of the subjects, who then continued on the diet for a full year without any ill effects from it, maintained a relatively constant weight level. It appeared that, on this diet, in which the subjects derived, by choice, about 80 per cent of the calories from fat, 1 to 2 per cent from carbohydrate, and the rest from protein, the excess fatty deposits of the body were utilized for energy but the normal energy stores were maintained.

The proportions of protein and fat in the diet would be represented by three parts of lean meat to one part of fat, by weight. A small amount of carbohydrate is present as the glycogen of the meat. The subjects of the experiment ingested food to the amount of 2000 to 3100 calories a day, the precise amounts depending upon their appetites. In round figures this would be represented by 2 to 3 ounces of fat and 6 to 9 ounces of lean meat at each of the three meals of the day.
This diet, with some modifications, has formed the basis of a treatment of obesity which has been used in clinical practice for more than two decades (32). During the past few years it has been used in a group of industrially employed individuals (33, 34); and it appears to have many advantages over treatment by caloric restriction. It seems that it would be useful, especially, in those cases of obesity in which the application of low calorie diets results in hunger or in a decline in the energy expenditure.

**Summary**

Analysis of the results of studies of the energy exchange in obesity, in regard to their evidence for or against a passive dependence of the excessive energy stores on the balance between the inflow and the outflow of energy, indicates that these stores have a significant degree of independence of the energy balance. This appears to necessitate an explanation of obesity on the basis of some intrinsic metabolic defect. The decline in energy expenditure which occurs when the obese go on low calorie diets appears to have the same significance as it has when people of normal weight are subjected to undernutrition. A treatment of obesity, alternate to that of caloric restriction, takes into account the metabolic defect in obesity, aims at a primary decrease in the excessive energy stores, and allows for weight reduction without any decline in the energy expenditure and without any enforcement of caloric restriction.

**References**


32. Personal communication from Dr. Blake F. Donaldson; New York.


AppenDIX

References and Notes, in connection with paper, OBESITY: OVERNUTRITION OR METABOLIC DISEASE? by A. W. Pennington.


In this article, Newburgh summarizes his work on the energy exchange, most of which was done around 1930. The matters he takes up do not follow one another in a logical sequence but, rather, tend to carry the reader away from points on which concrete conclusions are expected.

It is clear, however, that he obtained his results from measuring the weight loss and calculating the caloric equivalent of tissue oxidized, rather than making direct determinations of the energy expenditure. For example:

Page 697: "Under these circumstances, change in body weight could be attributed to deposition or loss of adipose tissue, provided the observations of any subject were continued for several weeks. When the record was completed the average daily metabolic mixture would correspond with the diet except that any change in body weight over the whole period would have to be reduced to a daily average and then 90 per cent of it either added to or subtracted from the dietary fat. (Since adipose tissue contains 10 per cent body water and dietary fat is expressed in anhydrous terms, 90 per cent of the weight is used.) The calories of the diet, thus corrected, were taken to be the daily heat production of the subject."

The reader becomes impressed with the correctness of the predictions of weight loss; but these could be made on the basis of empirical observations during weight reduction, regardless of the water content of the body fat and of the caloric equivalent of tissue oxidized for energy. Thus, without a check by actual determinations of the caloric expenditure during weight reduction, the results obtained depend entirely on the assumption of a 10 percent water content in body fat. Their effect on loss in weight and on the metabolic rate in obese patients, how- ever, we find the following:

Note the wide range of 4 to 23 percent in the decline in basal calories.


Page 720: "...the simple principle will remain that obesity is invariably the result of a disproportion between the inflow and the outflow of energy. The former must always be greater than the latter, either because the intake has increased or the outgo has diminished." Thus, in summarizing, Newburgh gives data deducible from the law of conservation of energy and requiring no experiment; but to it he adds the implication that the entire pathogenesis of obesity can be summed up in this way. His statement would seem to indicate that the excessive stores of the obese are passively dependent on the disproportion between the inflow and the outflow of energy. His experiments did not prove this, however, for they were based on the assumption concerning the water content of body fat.

It is only after a very painstaking study of this article and of Newburgh's earlier papers that it becomes clear as to exactly what he was trying to find out in his experiments, how he proceeded to find out and what his results were. Many of my colleagues tell me, "Newburgh has done a lot of work on the matter," but no one seems to know exactly what he did, or did not do.


Table 14, page 545, gives the observed basal calories before and after weight reduction, in cases 1 to 5. These are listed in the first two columns below. In the third column are my calculations of the percentage decline.

Note: There was a decline in metabolism in all but two of the cases.


The author reports on the metabolism in only one

September, 1953
OBESITY: OVERNUTRITION OR DISEASE OF METABOLISM?

It is noted that there was a steady decline in B. M. R. during the first three weeks of dieting. Then there were variations in the metabolism curve. It will be seen that these variations coincide, quite accurately, with temporary levelings of the weight reduction curve. In the text, the author attributes such levelings to departures from the diet, some of which were known and others inferred from the effects on the weight curve when departures from the diet were definitely known, such as at Christmas time.


Although the authors state that there was no reduction in basal metabolic rate, calculations from their tables show an average decline of 2.8 per cent in boys and 4.8 per cent in girls.


Page 513, Table 128. Basal calories per 24 hours are given before and after dieting. From these, I have calculated, in the third column, the decline in basal calories and, in the fourth column, the percentage decline:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Basal calories on normal diet</th>
<th>Basal calories at end of diet</th>
<th>Decline in basal cal.</th>
<th>Per cent decline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bro</td>
<td>1481</td>
<td>1271</td>
<td>210</td>
<td>14.2</td>
</tr>
<tr>
<td>Can</td>
<td>1758</td>
<td>1590</td>
<td>168</td>
<td>9.6</td>
</tr>
<tr>
<td>Kon</td>
<td>1818</td>
<td>1429</td>
<td>389</td>
<td>21.4</td>
</tr>
<tr>
<td>Gar</td>
<td>1815</td>
<td>1450</td>
<td>365</td>
<td>20.1</td>
</tr>
<tr>
<td>Gul</td>
<td>1698</td>
<td>1427</td>
<td>271</td>
<td>16.0</td>
</tr>
<tr>
<td>Mon</td>
<td>1858</td>
<td>1544</td>
<td>314</td>
<td>16.9</td>
</tr>
<tr>
<td>Moy</td>
<td>1683</td>
<td>1331</td>
<td>357</td>
<td>18.7</td>
</tr>
<tr>
<td>Pen</td>
<td>1766</td>
<td>1295</td>
<td>471</td>
<td>26.6</td>
</tr>
<tr>
<td>Pee</td>
<td>1589</td>
<td>1217</td>
<td>372</td>
<td>23</td>
</tr>
<tr>
<td>Tom</td>
<td>1526</td>
<td>1217</td>
<td>309</td>
<td>20.2</td>
</tr>
<tr>
<td>Vea</td>
<td>1604</td>
<td>1264</td>
<td>340</td>
<td>21.2</td>
</tr>
<tr>
<td>Averages</td>
<td>1886</td>
<td>1587</td>
<td>319</td>
<td>19</td>
</tr>
</tbody>
</table>

Note the variation in per cent decline: from 9.6 to 26.6. Note that only one of the cases showed a decline greater than 23 per cent, which was shown by Strang and Evans in their obese subject No. 1.


Page 18. Here he discusses the decline in metabolism of the obese on low calorie diets. In my paper I have given the basic data from all of the papers he has referred to. He referred to Rony's book, Obesity and Leanness, but Rony used the material from Strang and Evans and from Keeton and Bone.


Effects of the diet on the weight:

<table>
<thead>
<tr>
<th>Subject</th>
<th>Start of diet (Kg)</th>
<th>End of one week</th>
<th>End of one month</th>
<th>End of year</th>
</tr>
</thead>
<tbody>
<tr>
<td>V. S.</td>
<td>72.2</td>
<td>70.2</td>
<td>68</td>
<td>69.4</td>
</tr>
<tr>
<td>K. A.</td>
<td>59.4</td>
<td>58.3</td>
<td>58.5</td>
<td>58</td>
</tr>
<tr>
<td>E. F. D. B.</td>
<td>76.0</td>
<td>73.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Effects of the diet on caloric expenditure (per hour):

<table>
<thead>
<tr>
<th>Basal metabolism of V. S.</th>
<th>Basal metabolism of K. A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/2/28 mixed diet 60.96</td>
<td>1/10/28 mixed diet 52.35</td>
</tr>
<tr>
<td>4/12/28 meat 66.38</td>
<td>1/31/4/13/28 meat 60.71</td>
</tr>
</tbody>
</table>

"After meat was taken for 6 weeks the B.M.R. of V. S. was 7 per cent above that found in the period of mixed diet and similarly the rate of K. A. rose 5%.” It is also stated that the rate of E. F. D. B. rose 6%. He stayed on the diet only 2 weeks. It did not disagree with him but he felt unhappy about it or had qualms about it.