SESSION II

Chairman: Professor Sir W. Melville Arnott

Birmingham Medical Research Expeditionary Society 1977 Expedition:
Effect of a Himalayan trek on whole body composition, nitrogen and potassium

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Summary
The body composition of the 17 members of the BMRES was studied using body weight and fat fold calipers, with measurements of whole body potassium and nitrogen. The full assessment was made just before departure from Birmingham and immediately on return. Daily observations of body weight and fat folds were made during the trek to high altitude.

During the ascent all subjects lost body fat but there was little change in lean body mass. After return it was found that there had been further loss of body fat and some loss of lean tissue also. One subject who took methandienone retained potassium but had no increase in body nitrogen. There was no correlation between changes in body composition and acute mountain sickness.

Introduction
Prominent among the many changes that occur in the human body on exposure to high altitude are symptoms of headache, nausea and anorexia. To some extent these symptoms may improve with acclimatization to altitude although survival at heights over 5000 m is only at the cost of a steady deterioration in physical health. Weight loss is a common feature in climbers on high altitude expeditions and one has only to look at the well known photographs of, say, the Everest or Ogre expeditions to see the severe degree of emaciation in even these super-athletes under such conditions. Michael Ward (1975) in his classic text on Mountain Medicine has said that the ability to maintain body weight appears to be the best index to successful acclimatization and, conversely, poor acclimatization is almost invariably accompanied by severe weight loss.

The BMRES trek was a rather more modest sort of expedition than the ones mentioned above but the authors thought that it would be of interest to study in detail the body composition of their subjects before, during and after the expedition and relate the findings to the incidence of acute mountain sickness (AMS).

Methods
Body weight was measured on a seat scale in Birmingham and a simple platform scale during the trek. Fat fold thickness was measured with Harpenden calipers and total body fat (TBF) derived by the method of Fletcher (1962). Lean body mass (LBM) was taken as the difference between TBF and body weight. During the trek daily fat fold measurements were made but only in duplicate at the 2 sites. In order to calculate TBF during the trek a conversion factor was derived for each subject by comparing the fat folds measured before departure and those obtained on the first day of the trek.

Whole body potassium (WBK) was estimated in the University of Birmingham Whole Body Counter by measuring the radioactivity from the $^{40}$K present naturally in the human body. The counter had been calibrated by the established method using $^{42}$K.
Whole body nitrogen (WBN) was measured by neutron activation analysis in the Department of Physics of the University of Birmingham. The subject is irradiated with a beam of fast neutrons generated in a cyclotron. Some of the nuclei of $^{14}\text{N}$ capture neutrons to form an excited state of the isotope, $^{15}\text{N}$, which decays almost instantaneously to its ground state with the emission of prompt gamma rays which may be identified and quantified. Body nitrogen can be measured with an accuracy of $\pm 4\%$ (Dabek et al., 1977). Whole body protein was calculated from WBN and all the body composition measurements were combined with standard values for the nitrogen, protein, intracellular water and potassium concentrations in various tissues to derive the sizes of intracellular water, protein, fat, extracellular water and other compartments. The results were compared with those of 'reference man' (International Commission on Radiological Protection, 1975).

The entire set of detailed body composition measurements were carried out in Birmingham during the week before the trek and repeated within 3 days of the return to minimize the effects of rapid recovery of any changes. The trek in the Himalayas lasted 23 days (27 Oct.-18 Nov. 1977). Weight and 2 sites TBF were recorded daily during the first 12 days (the period of ascent) and again back at Kathmandu on the 23rd day.

Because of the different circumstances, the subjects have been divided into 3 groups.

Group A. Most of the party, 14 of the 17, were grouped together as their experience in terms of exertion, timing, distance, etc., were virtually identical.

Group B. This comprised 2 of the fittest subjects who made the return part of the trek at high speed with the blood samples. Because of this they had 2 more rest days in Nepal.

Subject C. This was an individual who took an anabolic steroid, methandienone (Dianabol, Ciba) in a dose of 20 mg/day throughout the trek.

Results

The mean body composition of the whole group before departure was similar to 'reference man' but

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<th>Table 1. Body composition of the BMRES subjects and 'reference man'</th>
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<tr>
<td><strong>BMRES</strong></td>
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<tr>
<td>Body weight (kg)</td>
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<tr>
<td>Total body fat (kg)</td>
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<td>Lean body mass (kg)</td>
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<tr>
<td>Height (m)</td>
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<tr>
<td>Whole body potassium (g)</td>
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<td>Whole body nitrogen (kg)</td>
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The BMRES were taller and slightly less fat than the standard (Table 1).

During the 12 days of the ascent all subjects lost weight. There was a marked tendency to lose TBF mean loss 2.7 kg; range 0-5 kg; Fig. 1) but at this stage there was no consistent change in LBM (range +2-6 kg to −3-2 kg, Fig. 2). There was no correlation between these changes and the incidence of AMS. On the return to Kathmandu the overall mean loss of body weight was 4.1 kg; this was made up of a 2.8 kg loss of TBF and a 1.3 kg loss of LBM.

When the detailed studies made before departure from Birmingham and after return were compared, similar changes were found (Fig. 3). For Group A the mean weight loss was 3.3 kg (range, 0.7-7.6 kg). Only 3 subjects had no weight loss: Group B, who were fitter than average and who had a longer period of recovery in Kathmandu, and Subject C who took an anabolic steroid. Loss of TBF accounted for most of the weight loss in Group A, but there were mean losses of 0.7 kg protein and 0.3 kg water. In Group A the changes in WBK were smaller and less variable than the changes in WBN (mean, −2.8% compared with −5.7%; largest changes...
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- 9% compared with -16%). Two of the 3 individuals who had suffered significant loss of potassium had AMS but no other relationships were noted.

A large and highly significant increase in WBK of 20 g was experienced by Subject C, who took the anabolic steroid but his WBN did not alter. Subject C did not notice any clinical benefit from taking methandienone and there were no apparently related changes in his plasma electrolytes, pattern of electrolyte excretion or fluid balance. On the other hand, his daily aldosterone excretion was very low compared with all the other members of the party, compatible with water and salt retention.

Discussion

Some of the subjects had moderately severe AMS but bronchitis and gastrointestinal upsets with loss of appetite were more troublesome than AMS to some others. The mean loss of fat and protein indicated a negative energy balance of about 4000 kJ/person/day throughout the 23-day trek. There may be several reasons for this, e.g. high energy output and hard physical work under stressful conditions, and an unbalanced diet which was unattractive to some. The significant losses of WBN and WBK in Group A suggested loss of cells and extracellular connective tissue. This was not reflected by loss of LBM but the situation could have been complicated by increase of body water. It might have been expected that the prolonged exercise would have led to some increase of muscle bulk which would have resulted in an increase of the ratio WBK/WBN (Burkinshaw, Hill and Morgan, 1978). In Group A there might have been an increase in this ratio (P=0.1 to 0.05, one-tailed t-test) that is, there might have been a sparing of the muscle cells at the expense of a greater loss of extracellular protein. An increase of muscle mass at the expense of extracellular protein is a possible explanation of the large increase of WBK in the subject who took the anabolic steroid. Perhaps an overall increase of lean tissue bulk was prevented by negative calorie balance.

References


Hughes, D. & Williams, R.E. (1967) The calibration of a whole-body radioactivity counter for the measurement of whole body potassium content in clinical studies. Clinical Science, 32, 495.
