have been noted in turkeys [3] and other animals.[4] A toxic crop of a grain similar to "bhajra"—namely, *P. scrobiculatum* L.—contains a tranquillising agent.[5 6] *Aspergillus flavus* has been associated with toxic-feed diseases,[7] and from it an endotoxin [8] and aflatoxins [9] have been isolated. Besides these, and *Penicillium puberulum* which also produces a toxin,[10] we now have a phycomycete, a toxic product of which causes a clinical syndrome in man. It is of especial interest because of the epidemic nature of the disease, in which respect it resembles ergotism.

Forty years ago Narasimhan [11] described a condition of severe nausea, vomiting, and death due to cardiac arrest in nearly 200 people, caused by consumption of *Eleusine coracana* Gaertn., the staple grain in Mysore, which had been contaminated by *Heterosporium* sp.

The main lesion in aflatoxicoses is hepatic periportal necrosis [12] with anorexia and features similar to those of biliary cirrhosis, ending fatally in two to three weeks.[13] By contrast, in our series only 3 patients died (before investigation into the syndrome started) and none of the rats have died.

Two wholly breast-fed infants, of mothers who had the disease, also had symptoms. This would seem to be due to passage of the toxin in the milk, similar to the excretion of a toxic metabolite in the milk of cattle fed with aflatoxins.[14] Nephrotoxins from *Aspergillus* [15] are active only when administered parenterally,[16] while polyuria from *R. nigricans* could be produced, in rats, by a single oral administration.

The syndrome differs from hysterical or psychogenic polydipsia [17–19] in that even on forced withdrawal of fluids, polyuria continued and patients became dehydrated. Besides diabetes insipidus after posterior encephalitis [20] and epidemic albuminuria in United States troops in 1926,[21] this is the only other known "urological" epidemic. Further experiments on the isolation of the mycotoxin and its relation with the metabolism of the antidiuretic hormone are in progress.

We suggest that this epidemic polyuria and polydipsia caused by a fungus be termed the "Sassoon Hospital syndrome".

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FASTING SERUM-TRIGLYCERIDES CONCENTRATION AND DISTRIBUTION OF SUBCUTANEOUS FAT

Summary Among a group of twenty-eight men aged 35–54 a highly significant correlation was found between fasting serum-triglyceride and the ratio of subscapular skinfold thickness to triceps skinfold. This relation was not attributable to a common correlation with obesity or glucose intolerance. In a similarly selected group of twenty-one men aged 55 and over there was no significant correlation between serum-triglycerides and the skinfold ratio. The hypothesis is advanced that in the younger men the correlation is due to a hormonal factor.

INTRODUCTION AND METHODS

WE examined the hypothesis that in middle-aged men, distribution of subcutaneous fat may be more significantly associated with blood-lipids than is amount of fat. In particular, on the basis of findings by Albrink and Meigs,[1] we postulated that a preponderantly central distribution of subcutaneous fat would be associated with higher fasting serum-triglyceride levels.

The data used related to fifty men who had been selected from the population of Tecumseh, Michigan, for the study by Ostrander et al.[2] on the relations between serum-triglycerides, carbohydrate intolerance, and coronary heart-disease. These men had all been examined previously in the course of the Tecumseh Community Health Study [3] between 1962 and 1965. From the population studied at that time, four groups were recalled for the special study carried out in 1966. The groups were matched for sex and age but differed in the presence or absence of coronary heart-disease and hyperglycæmia. Thus there were a hyperglycæmic and a normoglycæmic group with coronary heart-disease and a hyperglycæmic and a normoglycæmic group without coronary heart-disease. Hyperglycæmia was defined in terms of the blood-glucose 1 hour after the ingestion of 100 g. of glucose. For men aged under 45 years the critical value taken was 170 mg. per 100 ml. and for men aged over 45 it was 195 mg. per 100 ml. Skinfold measurements had been obtained from all men in 1962–65 but were not repeated at the time of the biochemical determinations. The methods and criteria used are described elsewhere.[4] We calculated the ratio of subscapular skinfold to triceps skinfold as an index of the centrality of fat distribution. The records of one man were unobtainable and he is excluded from this report.

TABLE I—CORRELATION COEFFICIENTS (r) IN 28 MEN AGED 35–54

Variable	Skinfold ratio	Serum-triglyceride	Serum-cholesterol
Skinfold ratio	..	0·699*	−0·074
Relative weight	0·253	0·060	0·341
Sum of fat folds	0·119	−0·100	0·146
Glucose index†	0·099	0·158	0·271
Serum-cholesterol	−0·074	−0·126	..
Age	−0·107	−0·090	0·023

* $P < 0{\cdot}001$.
† The sum of blood-glucose values at 0, 30, 60, 90, and 120 minutes after ingestion of 100 g. of glucose.

RESULTS

The men ranged in age from 35 to over 70 and were divided for the analysis into a younger group aged 35–54 and an older aged 55 and over. Table I lists linear

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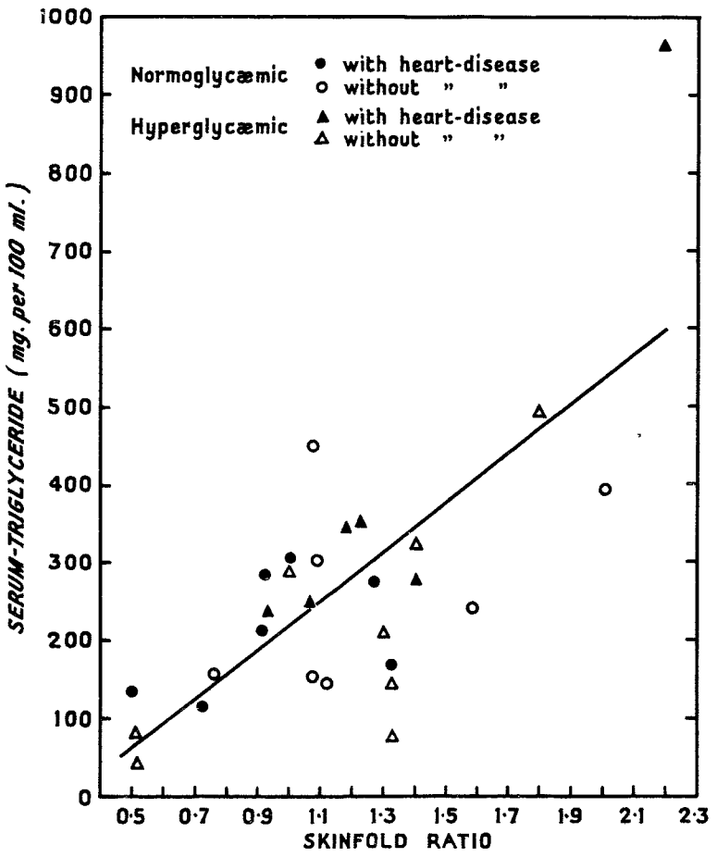

Relation between skinfold ratio and fasting serum-triglyceride concentration in twenty-eight men aged 35 to 54.

Serum-triglyceride (mg. per 100 ml.)=305·24 (skinfold ratio) −86·92. $r=0{\cdot}699$; $P<0{\cdot}001$.

correlation coefficients between variables for the twenty-eight men aged 35–54. There was no significant relation between serum-cholesterol and skinfold ratio, but there was a highly significant correlation between skinfold ratio and serum-triglyceride levels. The figure shows the distribution of the twenty-eight observations about the regression line. The four groups of values are scattered along the line, although there is a tendency, noted by Ostrander et al.,[2] for the hyperglycæmic men with coronary heart-disease to have high triglyceride levels.

We considered the possibility that the correlation was due to some common relation to a third variable. Table I shows that both skinfold ratio and triglycerides are positively but insignificantly correlated with relative weight. In table II the data are divided by relative weight and in each grouping partial correlation coefficients are calculated between skinfold ratio, triglycerides, and sum of the two fat folds. It is clear from this that the relation between skinfold ratio and triglycerides is seen in all ranges of relative weight (although falling short of statistical significance in the highest range) and is not due to a common relationship with total amount of subcutaneous fat. From table I it will be seen that glucose intolerance as represented by the glucose index (measured at the time of serum-triglyceride determination) does not account for the relation.

A series of two independent variable regression equations were calculated for the prediction of serum-triglyceride. None of the other variables listed in table I made a significant contribution to the regression when skinfold ratio was included in the equation.

For men over the age of 54 there was no significant correlation between skinfold ratio and serum-triglycerides (table III). It seems that in this age-group skinfold ratio is more closely related to overall obesity as measured by the sum of the fat folds. The two age-groups did not differ in mean relative weight.

TABLE II—PARTIAL CORRELATION COEFFICIENTS*: SKINFOLD RATIO (1), SERUM-TRIGLYCERIDES (2), AND SUM OF FAT FOLDS (3) BY RELATIVE WEIGHT GROUPING FOR MEN AGED 35–54

Relative weight	No.	$r_{12\cdot3}$	$r_{23\cdot1}$	$r_{13\cdot2}$
<100	9	0·875†	−0·264	0·225
100–109	12	−0·706‡	−0·189	0·191
⩾110	7	0·404	0·059	0·384

* $r_{xy\cdot z}$ = correlation coefficient between x and y holding z constant.
† $P<0{\cdot}01$. ‡ $P<0{\cdot}05$.

TABLE III—CORRELATION COEFFICIENTS, 21 MEN AGED 55 AND OVER

Variable	Skinfold ratio	Serum-triglyceride	Serum-cholesterol
Skinfold ratio	..	0·071	0·007
Relative weight	0·285	0·180	−0·026
Sum of fat folds	0·493*	0·258	−0·093
Glucose index†	−0·115	−0·002	0·071
Serum-cholesterol	0·007	0·385	..
Age	−0·196	−0·121	0·068

* $P<0{\cdot}05$. † Data available on only 20 men.

DISCUSSION

Although firm deductions cannot be made from a group of this size and constitution, these results suggest that for men in early middle age the fasting serum-triglyceride is correlated with centripetal subcutaneous fat distribution. The magnitude of the correlation in these data is surprising in view of the crudity of the measure of fat distribution used and the time lapse between the body measurements and the biochemical determinations. If the relation is a true one it is not necessarily compatible with the hypothesis of innate and acquired forms of obesity as postulated by Albrink and Meigs.[1] In the first place, the correlation is seen clearly among the thinner men (table II) and secondly, the work of Garn [5] shows that as an individual gains or loses weight he tends to preserve a constant proportional distribution of subcutaneous fat. On this evidence, it seems that the relationship between serum-triglycerides and body-fat distribution is due to a factor which is constant for an individual, independent of obesity, and which is not operative, or is obscured, in men aged over 54 and in obese women.[6] A hormonal factor seems likely. Although the status of serum-triglycerides as a risk factor in coronary heart-disease is still uncertain [7] the relationships between obesity, blood-lipids, and the disease might be clarified by an examination of the relevance of subcutaneous fat distribution.

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