Summary

* Full clinical examination.

* Evaluation of obesity by anthropometric measurements which included : Body weight, body length and height, upper and lower measurements, mid arm circumference, chest circumference, abdominal circumference, and skin-fold thickness.

* Biochemical estimation of serum hormones by radio-immunoassay technique for : Fasting Serum Insulin, Growth hormone, Thyroid hormones (T3 and T4), Cortisol, and Cyclic AMP.

The present study has shown that :

There was a highly significant increase in body weight of obese infants and children in all age groups of both sexes as compared to the control. Body weight of obese boys and girls throughout all age groups was above the 90th percentile of Tanner and Whitehouse (1976).

We found no significant difference in body length and height between obese and control infants and children in all age groups. The height of obese boys was found to be between the 25th and 97th percentiles

of Tanner and Whitehouse (1976). That of obese girls was found to lie between the 10th and 97th percentiles of the same standards.

There was a highly significant increase in triceps and subscapular skinfold thickness of our obese infants and children of both sexes in all age groups. Values of triceps skinfold thickness of obese boys and girls were found to be above the 90th percentile of the standards of Tanner and Whitehouse (1975). That of subscapular skinfold thickness were found to be above the 85th percentile of the same standards.

Certain body indices were calculated : Ponderal index, Body built index, Weight/Height ratio, and Fat index. We found that the last three indices were useful in confirming the diagnosis of obesity, while the 1st one was not useful.

We found a highly significant increase in mid arm circumference, abdominal circumference and chest circumference of obese infants and children of both sexes as compared to the control. However, all these

measurements are not a sensitive as an enthropometric indicators for the diagnosis of obesity. Also we found no significant difference in neither the upper segment nor in lower segment of the body.

The blood pressure in our study revealed a significant increase in systolic and diastolic blood pressure of obese infnats and children of both sexes and in all age groups as compared to the control. These findings are of great importance as it has been suggested that many of the factors responsible for lry hypertension and atherosclerosis in adults begins in childhood.

The presence of positive family history of obesity in our study in 26 patients (66 - 66%) of cases points to the importance of genetic factor in the predisposition of obestiy in childhood.

We noticed that the earlier the order of birth of the child in his family, the more prone to develop obesity; not only in obese children born to obese families, but also to those born to families without history of obesity.

As regards to the hormonal profile in our children we found that :

Serum insulin level (uU/ml) was significantly higher in the obese groups (24 ± 3.577), (36.64 ± 3.997), and (39.98 ± 3.771) as compared to the control (12.7 ± 2.2), (23.83 ± 3.658) and (26.92 ± 0.616) in age groups (0 - 2), (>2 - <6), and (6 - 12) years respectively. It was suggested that such high serum insulin values are probably due to the insulin resistance in obese patients.

Serum growth hormone level (ng/ml) was normal in age groups (0 - 2 years) and (>2 - <6 years), the mean values for obese infants and children were (3.51 ± 0.85), and (1.943 ± 0.385) as compared to the control (3.54 ± 0.997) and (2.51 ± 0.589) respectively. But in age group (6 - 12 years) we found a significant decrease in GH concentration (1.286 ± 0.158) compared to the control (2.11 ± 0.329). It was suggested that increased growth takes place in obese children despite low GH presumbly because of normal somatomedin levels propably as a result of hyperinsulinaemia.

Thyroid functions in our study revealed that the mean fasting levels of T3 (ng/dl) were (152.7 ± 12.26), (154.07 ± 9.341), and (154.5 ± 12.703) as compared to the control (112.4 ± 11.12), (144.83 ± 15.762) and (160.85 ± 9.3), and that for T4 (µg/dl) in obese patients were (10.37 ± 0.612), (9.771 ± 0.9), and (10.328 ± 0.641) as compared to the control (7.64 ± 1.324), (9.716 ± 0.763), and (8.142 ± 0.334), in age groups (0 - 2), (>2 - <6), and (6 - 12) years respectively. There was a significant increase in T3 in age group (0 - 2) years, and a significant increase in T4 in age groups (0 - 2), and (6 - 12) years in obese infants and children in comparison to the control. As regarding T3/T4 ratio there was non-significant change in the three age groups. In conclusion, it seems that thyroid function in obesity is normal, however, there is increase in T3 in age group (0 -2), and in T4 in age groups (0 - 2), and (6 - 12) years, although the individual values fall within the normal range. The underlying mechanisms for these changes may be an error in the conversion of T4 to T3, due to increase in reverse T3 on the expense of T3, or attributed to increased tissue resistance to T3.

Serum cortisol level (µg/dl) in age group (0 - 2) years was significantly increased in obese infants (12.318 ± 1.262) as compared to the control (7.46 ± 1.42). But in age groups (>2- <6) and (6 - 12) years, the mean values of fasting serum cortisol in obese patients were insignificantly changed (5.518 ± 0.835) and (5.426 ± 0.78) as compared to the control (5.66 ± 1.48) and (3.45 ± 1) respectively. It was suggested that these changes in serum cortisol concentrations are proportional to body mass, and 2ry to obesity and not the cause.

Our results revealed that plasma cyclic AMP (nm/L) was significantly increased in age group (0 - 2) years in obese patients (3.414 ± 0.479) as compared to the control (2.07 ± 0.494), but no significant change was obtained in age groups (>2 - <6) and (6 - 12) years in the obese patients (2.356 ± 0.386) and (2.532 ± 0.445) as compared to the control (3.408 ± 0.868) and (1.889 ± 0.705) respectively. The increase in the level of cyclic AMP in age group (0 - 2) years may be attributed to the associated increase in serum levels of T3, and

T4, or in serum cortisol due to stimulation of ACTH; in the same age group.

There has been an erroneous tendency to associate malfunction of the endocrine organs with obesity. It is more likely that disturbances of endocrine origin may appear as secondary manifestations than as the initiating cause as it was found that these hormonal changes reverts to normal after weight reduction.