

CONCLUSION

The final conclusions reached as a whole were as follows : Myxedema is a well-defined disease which affects women more often than men. The victims are for the most part middle-aged. This condition is due (in all cases) to destructive changes in the thyroid gland, the most common change being substitution of delicate fibrous tissue for the glandular structure.

In reviewing the early history of myxedema, one cannot fail to be impressed by the remarkable acumen of the clinicians of the late 19th century. The careful, detailed and pictureque descriptions of this disease in the reports of William Gull, William Ord, and the Myxoedema Committee of the Clinical Society of London remain even today classic clinical observations unsurpassed by any subsequent studies. William Ord's remarks, as he named the entity myxoedema, were prophetic : "..... my suggestion is that the whole collection of symptoms are related as effects of the jelly like swelling of the connective tissue.....".

It must be apparent that much remains to be learned about myxedema.

Serious non-thyroid illness and caloric deprivation, which so often accompany systemic illness, have diverse and still incompletely understood effects on thyroid hormone economy. We observed the following alterations in routine

thyroid function tests : a decreased serum T_3 concentration; normal or, in critically ill-patients, a low total serum T_4 level; in patients suffering from liver and renal disease.

With the recent advent of sensitive assays for TSH and better methods for serum T_4 , it is now possible to define more quickly and accurately the thyroid-metabolic status of most of the sick patients; the vast majority are euthyroid.

Therefore, knowledge of all of the ways in which systemic illness may influence thyroid function tests is crucial in assessing the thyroid status of patients with serious non-thyroid disease.

There is an inverse relationship between thyroid function and the concentration of various lipids in the serum. This relationship is especially apparent when one follows the fluctuations during the management of individual cases of either hyper or hypothyroidism. With treatment, lipid values returned to normal in almost all cases.

The thyroid hormones act directly on mitochondria and thereby control the transformation of the energy derived from oxidations into a form utilizable by the cell. Through their direct actions on mitochondria, the hormones also control indirectly the rate of protein synthesis and thereby the amount of oxidative apparatus in the cell. A rationale for the effects of thyroid hormone

excess or deficiency is based upon studies of the mechanism of thyroid hormone action. In hypothyroidism, slow fuel consumption leads to a low output of utilizable energy. In hyperthyroidism, rapid fuel consumption leads to a high energy output, but as efficiency decreases, the utilizable energy produced decreases. Many of the chemical and physical features of these diseases can be reduced to changes in available energy.

In conclusion, human hyperthyroidism has been shown to be associated with a reduction in the number of erythrocyte $\text{Na}^+ + \text{K}^+$ pump units. The direction of the change suggests that the effects of thyroid hormone on $\text{Na}^+ - \text{K}^+ - \text{ATPase}$ are probably more complex than previously anticipated. In any event, such changes in erythrocyte $\text{Na}^+ + \text{K}^+$ - pump units may provide a measure of thyroid hormone action at a cellular level in the human and this could well prove to be of use clinically.