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THE CLINICAL CARDIOVASCULAR FEATURES OF HYPERTHYROIDISM

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The fully developed clinical picture of toxic goiter or hyperthyroidism is familiar to all practitioners of medicine, consisting generally of weight loss, hyperthemia, nervousness, palpitation, tachycardia, struma, fine tremor of the extended fingers, prominence of the eyes and other eye signs, sweating, increased pulse pressure.

There are, however, a number of instances of toxic goiter where the presenting symptoms or complaints are such to be predominantly cardiac. This may lead to an impression, on the part of the patient, that his trouble is heart disease, and may indeed deceive the clinician as well, unless the latter carries a keen awareness of some of the vagaries of hyperthyroidism. These cases have thus come to be spoken of as “masked hyperthyroidism.”

Among the cardiovascular features of hyperthyroidism, tachycardia is the most striking phenomenon encountered. The averaged pulse rate, in one hundred cases of hyperthyroidism, ranged around 120 per minute, although one individual in the series had a pulse rate of 44. The systolic blood pressure of patients with hyperthyroidism, in both the nodular and diffuse hyperplastic types, is often slightly elevated, and the diastolic pressure somewhat lowered; thus giving a pulse pressure equal to 50 percent or more of the systolic level. The pulse is dynamic in character, as a rule, and a collapsing or Corrigan pulse is frequently noted. Over the femoral artery, in the groin, a systolic tone is generally heard, occasionally of sufficient intensity to be characterized as a “pistol shot” sound. Inspection of the large arteries usually reveals well marked peripheral throbbing, especially notable in the abdominal aorta. The cardiac apex impulse is often diffuse, covering two or more interspaces, and in some cases abrupt, causing a distinct systolic shock. In others, there may be present a questionable thrill, difficult to time.

The character of the heart sounds may best be described as thudding or loud. The first sound at the apex is often abrupt and roughened, so that it has occasionally led examiners to speculate as to the possible presence of mitral stenosis.
HISTORICAL ISSUE — SMITH

Systolic murmurs are relatively common, usually present at the cardiac apex and louder in the pulmonic area. At times, the murmur in the pulmonic area may have a suggestively rubbing quality, and be increased in intensity by pressure with the bell of the stethoscope. This type of murmur generally disappears after relief of thyroid toxicity, while in a few who have subsequent relapses into hyperthyroidism, its presence may again be noted. It is interesting to speculate on the cause of this murmur. The strong probability exists that it may be related to the frequently observed dilatation of the pulmonary conus on x-ray in these individuals.

A slight degree of cardiac hypertrophy is present in about 40 percent of cases. In some individuals, on account of the rather violent apical pulsation communicated to the chest wall, an impression of cardiac hypertrophy, which may be present on physical examination, fails confirmation on radiographic study.

Congestive cardiac failure is very rarely found in patients with hyperthyroidism who have regular cardiac rhythm, unless there is an associated valvular lesion or some degree of degenerative myocardial change from other cause than hyperthyroidism.

No single sign or manifestation should be thought pathognomonic of hyperthyroidism, but a combination of suggestive cardiovascular findings may be of considerable value in arriving at a correct diagnosis. The immediate effect of this condition on the heart is to produce a marked degree of cardiac overactivity, of which the various findings noted above are signals. This overactivity is generally commensurate in degree with the intensity of the thyroid toxicity and the increased cardiac output. Together with the other evidences of overactivity mentioned, we would like to stress as of suggestive value when present, the rubbing sound which we have described over the pulmonic area during systole, especially since it seems to have been largely overlooked in descriptions by other authors. This sound is of assistance to the diagnostician only in adding to the total sum of objective data suggesting hyperthyroidism. It may no doubt very occasionally be present in the absence of thyroid toxicity, but its intensity during the height of the disease, disappearance following relief of toxicity and reappearance in the few instances of recurrent hyperthyroidism has been striking.

The possibility of erroneously diagnosing rheumatic heart disease in cases of pure hyperthyroidism seldom offers real difficulty. The patients having mitral stenosis and hyperthyroidism generally have very frank signs of the former, with a pronounced presystolic rumbling murmur — a murmur which because of cardiac overactivity, is exaggerated during the period of thyroid intoxication. The examiner should not be too easily misled by less definite suggestions, such as a systolic shock or any ill-defined thrill palpated at the cardiac apex. Again, the peripheral arterial phenomena, which are common in aortic incompetence and occur in the majority of instances of hyperthyroidism (increased pulse pressure, Corrigan pulse, throbbing arteries and pistol shot sounds over the femoral artery) should merely serve to stimulate the examiner to consider the diagnosis of hyperthyroidism, especially if tachycardia be present and the diastolic murmur of aortic incompetence absent.
HYPERTHYROIDISM

Electrocardiographic findings in hyperthyroid patients with sinus rhythm are not diagnostic of this condition. However, it is frequently true that these patients show large P waves in lead II, above 1.7 mms., R waves above 16.5 mms., and tachycardia, and about 75 percent of them will show fine vibrations in the string shadow, reflecting the fine tremor of the extremities. The T waves are flat, diphasic or inverted in 25 percent of such patients. In electrocardiograms taken after relief of thyroid toxicity, there is a noticeable tendency for the P and R waves to decrease in voltage, the tremor to lessen and formerly flat or negative T waves to become again positive.

The following is a case report of an individual who presented himself to the examiner for what he believed to be a cardiac condition, where the diagnosis was proved to be hyperthyroidism. R. G., age 24, was a newspaper reporter. Two weeks previously, a life insurance examiner had noted the presence of a systolic murmur and questioned his insurability on this account. There had been no previous rheumatic history. The patient himself had no other complaints. He felt entirely well. On examination, there was a slight smooth enlargement of the thyroid. His blood pressure was 140/52, pulse 80/minute and regular. There was no evidence of cardiac hypertrophy. The rhythm was regular, but a systolic murmur, grade III, was heard in the pulmonic area. The intensity of this murmur, almost of a rubbing quality, increased with pressure of the bell of the stethoscope. It was somewhat less marked with the patient sitting. No diastolic murmur could be heard in the lateral position or after exercise. The pulse had a bounding quality. There was a slight tremor of the extended fingers and the eyes showed a slight lid-lag. The impression of a mild hyperthyroidism was confirmed by a basal metabolism determination of plus 32 percent. The patient was placed on Methimazole, and in three months the basal metabolism dropped to plus 5 percent. The systolic murmur could scarcely be heard, and it had lost its rubbing quality. The patient had gained eight pounds, the pulse rate had dropped to 68, blood pressure 128/70. He was presumed again to be euthyroid, and incidentally, to be insurable from a cardiac standpoint.

A second patient illustrative of “masked hyperthyroidism,” was C. S., a female aged 69, seen in February, 1954. This patient had been treated elsewhere and the true nature of her illness had not been recognized. When first seen at the office, the predominant signs and symptoms were of a cardiac nature. She had had a thyroidectomy in 1910 for “inward goiter.” A bilateral salpingoophorectomy had been done 4 years previously for a granulosa tumor of the right ovary. Her height was 5 feet, 3 inches, weight 239 pounds. Previously, in 1940, the weight had been 300 pounds. Her chief complaint was nausea and vomiting. She had diabetes and a blood sugar of 220. The blood pressure was 180/80. There was a pulse of 120. Paroxysms of auricular fibrillation were present. There was slight exophthalmos and lid-lag. Moist rales were noted at both bases, plus engorgement of the liver and ankle edema. The thyroid, beneath the scar, was increased in mass about two and a half times normal. Electrocardiogram showed sinus tachycardia, large P waves in lead II, diphasic T waves in leads I and II, flat T in AVL. A fine tremor was noted in the limb leads. X-rays showed moderate cardiac hypertrophy and some widening of the superior mediastinum. Tracer studies with I^{131} showed a high contact count in the mid line, an inch below
the thyroid scar and a 24 hour retention of 70 percent. Methimazole, 10 mgms. every 6 hours, was given initially. Two therapeutic doses of $^{131}I$ orally, 5-29-54 and 9-27-54, resulted in complete clearing of the congestive failure and remission of the evidence of hyperthyroidism as well as clearing of the diabetic problem. The weight dropped to 214 pounds. The pulse was 80, blood pressure 170/90. However, the exophthalmos had progressively increased in the face of adequate control of the hyperthyroid state.

Patients with thyrotoxicosis seldom develop congestive failure unless their hearts are afflicted with an added handicap, such as rheumatic valvular disease or a degree of myocardial degenerative change which may occur with arteriosclerosis or hypertension. The majority of these patients are beyond 50. Usually the gland is of the nodular or adenomatous type and frequently auricular fibrillation develops, either in a transient manner or as an established arrhythmia. Characteristically, the ventricular rate may not slow on digitalization to the usual degree brought about by digitalis administration in euthyroid patients. In some of these patients, because of a low position of the thyroid, recognition of a small nodular goiter may be missed if the thyroid is only felt for in the recumbent position. In fact, the gland may be placed retrosternally, and so escape physical detection altogether. Such patients with congestive failure, where the thyroid element in the sum of clinical manifestations is overshadowed by the cardiac features, again constitute a large percentage of the group of "masked hyperthyroidism." After relief of toxicity from hyperthyroidism, any cardiac insufficiency that remains must be thought to be due to other factors and treated accordingly.

In summary, it should be emphasized that a really careful use of the physician’s own powers of observation, coupled with an awareness of the clinical cardiac manifestations encountered in hyperthyroidism, continues to be of inestimable value in the early detection of this condition.