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The Various Types of Hyperthyroidism

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With 57 illustrations and 96 tables

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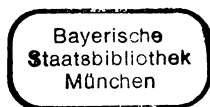
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III.1.1 Hyperthyroid heart disease: Diagnostic and therapeutic aspects

P. C. Scriba

The secondary cardiopathy following an abnormality of the thyroid gland function may be accessible to causal treatment. This is one of the reasons for the cardiologists interest in endocrinology. The cardiovascular changes to be observed in thyrotoxicosis may be clearer in perspective when dealt with in comparison to hypothyroidism. For this reason, a case of myxedematous heart disease is demonstrated first.

The condition of myxedematous heart disease was first described and named by Hermann Zondek. Zondek had to leave our country in the early 30s and died 1979 in Jerusalem [1]. Prof. Zondek was one of the outstanding endocrinologists who as a victim of German policy and crime disappeared from Berlin and left the country so much poorer. In these days, we shall commemorate in this country the 50th anniversary of a day of terror and shame, the Reichskristallnacht. Remembering H. Zondek may add to our awareness of the past and help in aiming at a better world.

A brief description of the heart in hypothyroidism should start with the fact, that myocardial function is itself altered in the sence of a diminished function. Vora et al. [2] were able to demonstrate a slowed diastolic thinning rate of the posterior ventricular wall. – Further, the prolonged systolic time interval may be used as peripheral sign of hypothyroidism [3]. – The reader is referred to recent reviews for further details [3, 4]. It is well known, that hypothyroid patients tolerate less digoxin than euthyroid patients. The reverse is true for hyperthyroid patients. Giving a constant dose of 0.25 mg digoxin or 0.1 mg digitoxin, respectively, resulted in lower serum levels of digoxin in thyrotoxic patients as compared to euthyroid and hypothyroid patients, respectively [5]. The levels were negatively correlated to creatinine clearance, the latter obviously contributing to what is known as refractoriness to digitalis in hyperthyroidism. Notably, the levels for digitoxin were not different under these conditions.

A systematic comparison of the changes of the heart in thyrotoxicosis and hypothyroidism [3] has to list among others the two most prominent aspects of 1. cardiac failure and 2. arrhythmia in thyrotoxicosis.

Congestive heart failure and cardiac hypertrophy are sometimes though certainly not regularly observed in thyrotoxic patients as demonstrated in 2 such cases. Dr. Dillmann has dealt with the direct effect of thyroxine on the

myocardium. Forfar et al. [6] were able to show, that the left ventricular ejection fraction was decreased in thyrotoxic patients by exercise, whereas it increases in controls. This and the ratio of the ejection time to LVET were not normalized by propranolol. The latter observation was used as an argument for the direct effect of thyroxine.

In addition, there is evidence in the literatur [7] that a prolaps of the mitral valve may be observed in approximately half of the patients with immunogenic hyperthyroidism but only in 2 of 20 patients with thyrotoxicosis due to thyroidal autonomy. Further, the prolaps of the mitral valve was correlated to endocrine orbitopathy rather than to actual thyrotoxicosis. A "myxedematous" state of the mitral valve specific for immunogenic hyperthyroidism was therefore claimed [7].

Absolute arrhythmia due to atrial flutter or fibrillation may be observed in 10–15% of all thyrotoxic patients [3, 4]. When euthyroidism is achieved, some 60–90% of these patients return to normal sinus rhythm spontaneously. If medical or electrical cardioversion has to be applied at all, this should be done only after the patient has been euthyroid for a few months.

Regarding this problem the other way round, some 6–10% of all patients with absolute arrhythmia are reported to have thyrotoxicosis. Interestingly, Forfar et al. [8] have studied 4 patients who actually had only a negative TRH test but no T_4 or T_3 elevation and could only be converted after antithyroid treatment. – The explanation of arrhythmia in thyrotoxic patients is complex. Direct thyroxine effects and an increased sensitivity towards catecholamines play their role. Some of the patients probably have preexistent and contributing coronary heart disease. This may be concluded from occasional ECG changes of the ischemic type which disappeared after antithyroid treatment as observed in some of our patients. An increase in supraventricular extrasystoles may trigger atrial fibrillation.

Finally, the well known effectiveness of blocking beta-receptors in thyrotoxic patients has to be mentioned. A word of caution may be added. Beta-receptor blocking may be unnecessary and probably even dangerous if given too long and bradycardia should be carefully avoided in the thyrotoxic patient.

In summary, this short overview was meant as a clinical introduction to the review by W. H. Dillmann.

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