« INRODUCTION AND AIM OF THE WORK »

The thyroid gland maintains the level of metabolism in the tissues that is optimal for their normal function, thyroid hormone stimulates the o_2 consumption of most of the cells in the body, regulates lipid and carbohydrate metabolism, and is also necessary for normal growth and maturation (Ekholm, 1989). The main function of the gland is to synthesize and secrete the thyroid hormones, namely, thyroxine (T_4), triiodothyronine(T_3)(Ingbar and woeber, 1981). The thyroid gland plays an important role in the normal metabolic rate and that is why a precise control system is operating to provide the right amounts of thyroid hormones at different conditions, both suprathyroid and autoregulatory mechanisms are involved in this control system (Morley, 1981). The secretion of thyrotropin (TSH), which is the major modulator of thyroid function, is regulated at the level of the pituitary thyrotroph by the antagonistic effects of thyroid hormones and the TSH releasing hormone (TRH), the former inhibits and the latter stimulates the synthesis and secretion of (TSH), Therefore, excess thyroid hormone leads to decreased secretion of (TSH), and thyroid hormone insufficiency is associated with (TSH) hypersecretion. (Morley,1981).TSH stimulates all steps of thyroid hormone synthesis and secretion (Dumont and vassart, 1979).

Ischemic heart disease is defined by world health organization

(1979) as : myocardial impairment due to imbalance between coronary blood flow and myocardial requirement, caused by changes in the coronary circulation (Ardehali and ports 1990).In the last 20 years, epidemiolgic and experimental studies have provided considerable evidence linking certain risk factors for coronary atherosclerotic heart disease (CAHD), among many factors that have been shown to be important as risk factors of ischemic heart disease are hyperlipidemia, hypertension, cigarette smoking, sex , obesity , psychosocial tension, and diabetes mellitus(Martinez et al., 1998). When risk factors coexist, they multiply the risk of chronic atherosclerotic heart disease (CAHD) (Streeten et al., 1998). The most frequently recognized cause of myocardial ischemia is occlusive coronary artery atherosclerosis, which either causes direct arterial narrowing or thrombous formation (factor et al., 1998).

The cardiovascular system is one of the most important targets on which thyroid hormones act (klein and Ojamaa, 2001). More than 80% of the biologically active hormone T_3 derives from peripheral conversion of T_4 secreted by the thyroid gland (Pilo et al., 1990) .Clinical and experimental evidence has

shown that T_3 play a major role in modulating heart rate and cardiac contractility as well as arterial peripheral resistance,(polikar et al., 1993). T3 actions are carried out by binding with specific nuclear receptors that regulate responsive genes encoding for structural and functional cardiac proteins, direct , extranuclear, nontranscriptional effects, have also been descrided (klein and Ojamaa, 2001).

In severe illnesses of non-thyrodial origin (Gomberg et al., 1998),including myocardial infarction (Eber et al., 1995) and chronic heart failure (Hamilton et al., 1990), down regulation of thyroid hormone system and changes in thyroid homeostasis may occur. This condition which has been called “euthyroid sick syndrome” or “ Low T3 syndrome (Docter et al., 1993). However , as the severity of illness increase there is a drop in both serum T3 and T4(Mclver and Gorman, 1997).

The aim of the present work is to study the thyroid functions in patients with ischemic heart disease in different clinical presentations.