

RELATION BETWEEN NEUROPSYCHIATRIC DISORDERS AND THYROID GLAND

THESIS

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الحمد لله
والى والدى والدى ...
اعتزافاً بفضل وعرفاناً بالجليل



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

سُبْحَانَكَ اللَّهُمَّ عَلَّمْنَا إِيَّاكَ مَا عَلَّمْنَا

إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

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INTRODUCTION

INTRODUCTION

Thyroid gland has been long known to maintain the normal level of metabolism in the tissue, that it stimulate the O₂ consumption of most of cells in the body and help to regulate the lipid and carbohydrate metabolism and it's necessary for normal growth and maturation. The absence of thyroid gland leads to poor resistance to cold, mental and physical slowing, and mental retardation and dwarfism (*Levey 1971, Green 1958, Ganong 1990*), but thyroid hormone dysfunction can produce some neuropsychiatric manifestations as shown in hyperthyroidism which reveals a high incidence of emotional and personality disturbance (*Martini 1971*) (*Johanson et al., 1928*) also to the attention, impulsivity and poor concentration (*Gardener 1971*), however the hypothyroidism may leads to minor behavioural and personality changes which confined to inertia, irritability, slow perception, thought, speech, and an impairment in intellectual function, (*Rorschach 1941*), also there is an association between major psychosis paranoid schizophrenic and other manifestations which have been called myxoedema madness. (*Chambers and Milne 1988*), also there is a relation between thyroid dysfunction and mood disorders (*Georgotas et al., 1988*). Lastly there is some relation between thyroid dysfunction and agorophobia (*Orenstein et al., 1988*) and

another some relations to cannabis intake (*Spadone 1990*) there is a tight relation of thyroid dysfunction and some neurological disease as in some muscle disease (*Brain and Their colleague 1986*)

Myasthenia Gravis and as well as to periodic paralysis (*Engle 1981*) and motor weakness (*Simpson 1981*).

Also, there is some cerebellar signs recorded with thyroid dysfunction as Tattering gait (*White 1984*).

Also, there is some neurophysiological changes reported with some neurological cases as in thyroid dysfunction (Hyperthyroidism) may increase deep tendon reflex and increase muscle contraction time (*Lawson and Weisben 1959*)

Also there is an electromyographic changes as paroxysmal spike discharges, slowing and seizure activity may appear in thyrotoxicosis (*Condon et al., 1954*)

Also Babinski signs and clonus may appear (*Ross and Schwab 1939*), moreover (*Vanasse 1989*) reported an abnormalities in the brain stem auditory and visual evoked potentials in adult hypothyroidism.

Also, there is a trial to use thyroid hormone in treatment of some cases of degenerative disease as in motor neurone disease (M.N.D) and dementia (*Osterweil 1992*).

REVIEW OF LITERATURE

AIM OF THE WORK

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The aim of this review is to high lights the up to date knowledge in the literature concerned with role of thyroid in:

1. Neuropsychiatric manifestation of thyroid disturbances and the point for differential diagnosis of other neuropsychiatric disorders.
2. Role of thyroid function in different neuropsychiatric disorders and the possible relations between them.
3. Searching for new drugs for treatment of some neuropsychiatric disorders which are related to thyroid dysfunctions.

ANATOMY

ANATOMY OF THYROID GLAND

The anatomy was described by *romanes*, 1893, and recently modified by *Last*, (1990).

The thyroid gland consists of two symmetrical lobes united in front of 2nd, 3rd, 4th Tracheal rings by Isthmus of gland tissue each lobe is pear shaped consisting of narrow upper lobe and broader lower lobe it lies under cover of sternothyroid and sternohyoid muscles, to the side of the larynx and trachea. The upper pole lies tucked away beneath the upper end of sternothyroid muscle, between it and ala of thyroid cartilage, the lower pole extends along the side of the trachea as low as sixth tracheal ring. The gland possesses its own delicate histological capsule or fascia propria, it lies free within an envelope of pretracheal fascia. The isthmus joins the anterior surfaces of lobes, towards their lower poles. The posterior surface of Isthmus is firmly adherent to the 2nd, 3rd and 4th rings of the trachea and pretracheal fascia is here fixed between them. This fixation and the investment of the whole gland by pretracheal fascia are responsible for the gland moving up and down with the larynx during swallowing. Fig. (1, 2, 3, and 4A).