

# Disturbance of $^{99m}\text{TcO}_4$ : Uptake of the Thyroid Gland

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## Abstract

Radionuclide thyroid imaging is crucial in establishing an accurate diagnosis to differentiate between Plummer's disease and Graves' disease as well as to discover ectopic thyroid tissues, it is used in the aftercare of the well-differentiated thyroid carcinoma, and provides global and regional functional information of the parenchyme. The article gives an overview of possible disturbing mechanisms in the uptake of  $^{99m}\text{TcO}_4$  of the thyroid gland.

## 1. INTRODUCTION

Radionuclide thyroid imaging is crucial in establishing an accurate diagnosis when single or multiple thyroid nodules are palpated, as it is not otherwise possible to differentiate between Plummer's disease and Graves' disease developing in a multinodular gland, or to discover ectopic thyroid tissues along the path of embryologic migration from its origin to the porta hepatis. It is also a diagnostic tool in the aftercare of the well-differentiated thyroid carcinoma. In addition to ultrasound investigations radionuclide imaging provide global and regional functional information of the parenchyme.

With the application of  $^{99m}\text{TcO}_4$  the substance is captured by functional active cells of the thyroid gland. At this stage uptake takes place as an active process at the basal cell membrane against a concentration gradient of the blood circulation.  $^{99m}\text{TcO}_4$  as a generator radionuclide can be provided at any time. Its advantage lies in a better energetic level for the registration in the gamma camera (140 KeV), in a low local radiation exposure of the thyroid gland because of the failing beta radiation component, and in a short physical half-life of 6 hours, so that  $^{99m}\text{TcO}_4$  can be applied in a 20 as much higher radioactivity amount als  $^{131}\text{J}$ . The nuclear load of the thyroid gland exceeds to 3,4 mGy and is about 300 as much lower as in  $^{131}\text{J}$  and 10 as much lower als in  $^{123}\text{J}$ . Although of similar physicochemical features,  $^{123}\text{J}$  has to be manufactured in a cyclotron, and so is of less availability and quite expensive.

Enrichment of  $^{99m}\text{TcO}_4$  occurs via trapping, and it is discharged without being changed. After intravenous injection maximum concentration is achieved within 15 to

20 minutes. In numerous studies it could be proved that 15 to 20 minutes after application the exposure data correlate well with the data of radio iodine, so that the early measuring of the percentual uptake of  $^{99m}\text{TcO}_4$  ( $^{99m}\text{TcO}_4$ -Uptake - TcTU) is a standard for iodavidity of the thyroid gland [Mahlstedt 1976; Shimmins 1971]. Especially in combination with specific laboratory parameters as  $\text{TT}_4$ ,  $\text{TT}_3$ ,  $\text{FT}_4$  and TSH TcTU allows the quantitative determination of the status of the thyroid gland.

The best diagnostic measures for detection of autonomia of the euthyreotic goitre are the quantitative radionuclide imaging under suppression conditions. As regulatory mechanisms are – besides of the organic metabolism – the same as in the uptake of iodine by the thyroid gland itself, TcTU depends on the endogen stimulation of TSH and the intrathyroidal iodine content of the thyroid gland. Because of the close correlation between the thyroid iodine uptake and TcTU the individual intake of iodine of a patient may cause an essential effect on TcTU.

## 2. DISCUSSION

In a study concerned with 236 euthyroid individuals [Baehre et al. 1988] living in an area of iodine deficiency 227 of whom had endemic goitres. In these subjects autonomy could be suspected owing to an inhomogeneous activity distribution on the thyroid scintigram or a subnormal TSH response to TRH. Previously, in 190 separated controls without evidence of autonomy, the reference ranges for the thyroid  $^{99m}\text{Tc}$  pertechnetate uptake under suppression (TcUs), a measure for the non-suppressible thyroid iodide clearance, and for suppressibility of circumscribed thyroid regions, had been determined. These two parameters

obtained by high-resolution quantified scintigraphy were used for an accurate detection of thyroid autonomy among the 236 individuals. In the total investigated collective, the prevalence of autonomy was 77 % in patients with a goitre weight above 50 g. The individuals with abnormal suppression were grouped into four classes of TcUs. In these classes, free thyroxine index and total triiodothyronine increased with increasing TcUs, whereas delta TSH decreased. This finding indicates a continuum of different extents of autonomous thyroid function, whereas in the individual patient, the extent can be determined using the pertechnetate uptake under suppression. In addition, FT4I, TT3 and delta TSH in each of the TcUs classes depended on the individual iodine supply.

There are two ways for taking influence on TcTU. First a high concentration of plasma iodine can influence  $^{99m}\text{TcO}_4$  uptake into the cell. High concentrations of plasma iodine e.g. after application of contrast medium may lead to a complete blockade of  $^{99m}\text{TcO}_4$  uptake, as well as others, e.g. iodized sodium, iodized mineral water, and iodiferous pharmaceuticals. Selective inhibition can be caused by thiocyanate as a decomposition product of vegetable food so as cabbage, manioc or peanuts. Higher concentrations of thiocyanate can also be found in smokers, which may explicate a higher prevalence for goitre.

There may also be indirect influence through the hypothalamic-hypophyseal feedback mechanisms, e.g. caused by intake of thyroid hormones. Substances as carbimazol cause suppression of the iodization, so that after some weeks of therapy show an increased TcTU because of increased TSH concentration.

Changes of free hormonal blood concentration can be caused by changes of the TGB concentration, e.g. in pregnancy, where TGB and  $\text{TT}_4$  concentration can increase.

Several diseases can also cause changes in protein binding of the thyroid hormones, so as nephrotic syndrome and decompensated liver cirrhosis.

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