International Conference on
Nuclear Medicine & Radiation Therapy
July 14-15, 2016 Cologne, Germany

Determination of beta radiation dose to thyroid from the ingestion of radioiodine (^{131}I) by patients for diagnostic and therapeutic purposes

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Abstract

Radioactive iodine (^{131}I) is successfully used for the treatment of hyperthyroidism and thyroid cancer. Thyroid is the critical organ for iodine. Iodine is taken up by the thyroid follicular cells. ^{131}I simultaneously emits two types of radiation: beta minus particles used for the treatment and gamma rays used for diagnosis. Due to the short range of beta minus particles in tissue, damaging effects of beta radiation is restricted to thyroid cells. Total activities from the ingestion of ^{131}I were evaluated in different compartments of the human body of patients by using the ICRP biokinetic model for iodine. A new dosimetric model was developed for evaluating committed equivalent doses due to ^{131}I intakes in the thyroid tissue of different age groups of patients by exploiting data obtained for specific beta-dose deposited by 1Bq of ^{131}I in the thyroid. Data obtained were compared with those obtained by using the ICRP ingestion dose coefficients for iodine.

I. Introduction

Radioiodine ^{131}I is successfully utilized in nuclear medicine for the treatment of hyperthyroidism and thyroid cancer. This radioisotope emits beta minus particles used for the treatment and gamma photons used for diagnosis. Due to the short range of beta minus particles in tissue, damaging effects of beta radiation is restricted to thyroid cells. Information from the emitted gamma rays are analyzed by a gamma camera coupled to a computer and are transduced in images.

^{131}I is produced in nuclear reactors by bombarding natural tellurium metal (^{127}Te) by neutrons; it disintegrates by emitting β^- particles with a half-life of 8.04 days to unstable xenon $^{131}Xe^*$ which decays to stable xenon (^{131}Xe) by emitting gamma photons of different energies and intensities. ^{131}I is widely used for diagnostic and therapeutic purposes. For many decades, radioactive iodine was orally administered to patients for the treatment of benign and malignant thyroid diseases. Indeed, a major part of the administered radioiodine will concentrate in the thyroid gland; the emitted β^- particles of short ranges will only damage thyroid cells without any harmful health effects to the neighboring organ tissues. However, the emitted gamma rays may cause radiation damage to other tissues of the patients and other individuals. There are two types of biological effects of ionizing radiation: deterministic and stochastic effects. Deterministic effects are caused by the decrease in or loss of organ function due to cell damage or cell death. In the case of treatment of thyroid cancer or metastases, hyperthyroidism and goiter, the cell killing effect in some or all cells of the thyroid gland or in the metastases is the desired effect. Other organs should not be affected in such a way that deterministic effects will occur. Therefore beta-emitting ^{131}I is often the radionuclide chosen for these treatments, although the associated gamma emission exposes also other organs of the patient and other individuals. Stochastic effects are those that result from radiation-induced changes in cells that retain their ability to divide. These modified cells sometimes initiate malignant transformation of a cell, leading to the development of a malignant clone and eventually to a clinically observable cancer.

II. Methods of study

2.1- Determination of activities due to ^{131}I in different compartments of the human body

In terms of the International Commission on Radiological Protection (ICRP) biokinetic model for iodine [1], one can divide the human body into the following compartments:
-The blood (B)
-The thyroid (Th)
-The rest of body (Rb)
-The urine (U)
-The faeces (F)

Total activities due to ^{131}I in the different compartments of the human body are obtained by solving the last differential equation system by using a Maple 8 code providing that at t=0 these activities are equal to zero except that in the blood which is equal to $A_c(0)$. Indeed, for an n^{th} compartment one has:

$$A_c^n\left(^{131}I\right)(t) = A_c(0)\sum_{l=1}^{3} a_l^n e^{-\gamma_l^n t} \qquad (1)$$

where $A_c(0)$=1Bq is the ^{131}I intake at time t=0, a_l^n is a constant and γ_l^n is a rate constant in d^{-1}.

2.2- A new dosimetric model for evaluating beta-committed equivalent dose due to ^{131}I in the thyroid gland of patients

Beta-equivalent dose rate ($Sv\ s^{-1}$) due to ^{131}I in the thyroid (Th) of a patient is given by:

$$\dot{H}_{Th}(^{131}I)(t) = A_C^{Th}(^{131}I)(t)\, D_{Sp}^{Th}(^{131}I)\, W_R \qquad (2)$$

Where: $A_C^{Th}(^{131}I)(t)$ (Bq) is the total activity due to ^{131}I at time t in the thyroid. $D_{Sp}^{Th}(^{131}I)$ is the specific beta-dose (Gy) deposited by β^- particles emitted by 1Bq of ^{131}I in the thyroid tissue. W_R is the radiation weighting factor which is equal to unity for beta- particles.

The sepcific beta-dose is given by:

$$D_{Sp}^{Th}(^{131}I) = k\sum_j \frac{K_j R_j S_j}{M_{Th}} \qquad (3)$$

Where: K_j is the emission percentage of a beta minus particle of index j and average energy E_β emitted by ^{131}I [2]. R_j (cm) is the range of a beta minus particle of index j and average energy E_β emitted by ^{131}I in the thyroid tissue. $k=1.6\times10^{-13}$ J MeV $^{-1}$ is a conversion factor. S_j ($MeVcm^{-1}$) is the stopping power of the thyroid tissue for a beta minus particle of index j and average energy E_β emitted by ^{131}I. R_j and S_j were determined by using an ESTAR code and the chemical composition of thyroid given by in the ICRP Publication 89 [1]. M_{Th} is the mass (g) of the thyroid.

The beta- committed equivalent dose (Sv) due to ^{131}I in the thyroid gland is given by:

$$H_{Th}(^{131}I) = \int_0^{\tau} \dot{H}_{Th}(^{131}I)(t)\,dt \qquad (4)$$

where τ is the exposure time which is equal to 50 y for adults and to 70 y for children.

References

[1]: The Inetrnational commission on radiological protection. Age-dependent doses to members of the public from intakes of radionuclides. Part1, I CRP publication 56, 1989.

[2]: The Inetrnational commission on radiological protection, Radiation dose to patients from radiopharmaceuticals. ICRP publication 53, 1987.

III. Results and discussion

3.1- Total activities determined in different compartments of the human body of patients from the ingestion of ^{131}I

Activities due to ^{131}I were determined in different compartments of the human body of patients from the ingestion of ^{131}I by using. Variation of the ^{131}I activity in blood, thyroid, rest of body and urine as functions of time are shown in Fig. 1 for adults for ^{131}I uptakes=5%, respectively. It is to be noted that the residence time of ^{131}I in the thyroid and rest of body is higher than that in blood . This is because the transfer rates of ^{131}I from thyroid to the rest of body and from the rest of body to faeces are lower than that from blood to urine. The retention function for ^{131}I in thyroid is higher for adult than for 1y children patients, respectively. This is due to the fact that the transfer rate of ^{131}I from the thyroid to the rest of body is higher for 1y children than for adults . One can note that when the ^{131}I uptake increases (from 5% to 55%, for instance) for adults and 1y-children the ^{131}I activity in urine decreases. This is because the transfer rate of ^{131}I from blood to the urine decreases when the ^{131}I uptake increases. The presence of ^{131}I in urine of patients after lower ^{131}I uptake treatment could represent a source of radiation for their relatives when using common family toilets.

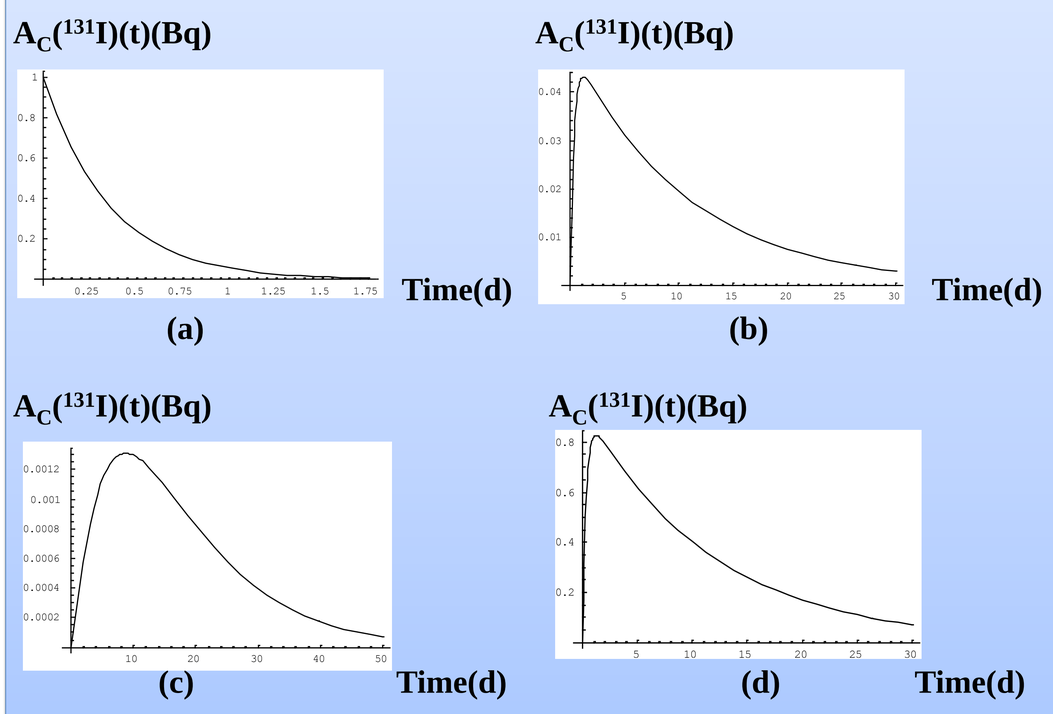

Fig. 1. Variation of the ^{131}I activity as a function of time in blood (a), thyroid (b), rest of body (c), and urine (d) for adult patients. The ^{131}I uptake is of 5 %.

3.2. Beta committed equivalent dose to thyroid due to the ingestion of ^{131}I by patients

In order to test the validity of our dosimetric model committed equivalent doses per unit intake $h_{Th}(^{131}I)$ were determined in the thyroid of various age groups of patients from the ingestion of ^{131}I. Data obtained are shown in Table 1. Considering uncertainties on ranges and stopping powers , the relative uncertainty of the committed equivalent dose is estimated to be about 10%. Data obtained are in good agreement with those obtained by using the ICRP model [2]. $h_{Th}(^{131}I)$ is influenced by the thyroid mass and activity integral (Eq. (4)). It is to be noted from results shown in Table 1 that committed equivalent dose per unit intake to thyroid is clearly higher for children than for adults, for a given ^{131}I uptake. This is because the thyroid mass is predominant (Eq. (4)). Indeed, the ratio of the corresponding highest committed equivalent doses [h_{Th}(1y)/h_{Th}(Adult)] is equal to 10.29 and the ratio of the corresponding thyroid masses [M_{Th}^{-1}(1y)/M_{Th}^{-1}(Adult)] (Eq. (4)) is equal to 11.11, whereas the ratio of the corresponding ^{131}I activity integral is equal to 0.93 for an ^{131}I uptake of 5% to thyroid increases with the ^{131}I uptake.

(a)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	0.66±0.07	0.68
5 years	0.36±0.04	0.37
10 years	0.16±0.02	0.17
15 years	0.11±0.01	0.11
Adult female	0.08±0.01	-
Adult male	0.06±0.01	0.07

(b)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	2.0±0.2	2.00
5 years	1.1±0.1	1.10
10 years	0.50±0.05	0.51
15 years	0.34±0.03	0.34
Adult female	0.24±0.02	-
Adult male	0.20±0.02	0.21

(c)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	3.4±0.3	3.40
5 years	1.8±0.1	1.90
10 years	0.83±0.08	0.84
15 years	0.56±0.06	0.56
Adult female	0.40±0.04	-
Adult male	0.34±0.03	0.36

(d)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	4.8±0.4	4.70
5 years	2.6±0.2	2.60
10 years	1.2±0.1	1.20
15 years	0.79±0.08	0.79
Adult female	0.57±0.06	-
Adult male	0.48±0.05	0.50

(e)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	6.3±0.6	6.10
5 years	3.4±0.3	3.30
10 years	1.5±0.1	1.50
15 years	1.02±0.1	1.00
Adult female	0.73±0.07	-
Adult male	0.62±0.06	0.64

(f)

Patient age group	Dose coefficient in thyroid $h_{Th}(^{131}I)$(μGy/Bq)	
	This method	ICRP dose coeffcient (53)
1 year	7.8±0.8	7.40
5 years	4.3±0.4	4.10
10 years	1.8±0.2	1.90
15 years	1.2±0.1	1.20
Adult female	0.89±0.09	-
Adult male	0.76±0.08	0.79

Table 1. Data obtained for the ingestion dose coefficient for ^{131}I in thyroid ($h_{Th}(^{131}I)$) for different age groups of patients and different ^{131}I uptakes: 5% (a), 15% (b), 25% (c), 35% (d), 45% (e) and 55% (f), by using this method and the ICRP ingestion dose coefficient [2]

IV. Conclusion

In this study, ^{131}I activity was calculated in different compartments of the human body of different age groups of patients. It has been shown that the ^{131}I activity is influenced by the ^{131}I uptake and transfer rate of ^{131}I between the different compartments. A new dosimetric model based on the formalism of specific beta dose deposited by 1 Bq of ^{131}I in the thyroid tissue was developed and validated and beta radiation doses to the thyroid from the ingestion of radioiodine (^{131}I) by patients for were evaluated. It is concluded that committed equivalent doses to the thyroid gland are influenced by the ^{131}I uptake, transfer rate of ^{131}I, mass of thyroid, and energy of the emitted beta minus particles. ^{131}I uptake was measured for female patients presenting different hyperthyroidism pathologies by using a gamma camera and the resulting committed equivalent doses were determined.