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Reply

We must agree that our results differ slightly from those of Prof. Jonckheer. It is our experience (*1*,*2*) that if patients show no antithyroid antibodies, and remain euthyroid under amidarone, only half of them will show thyroid iodine contents that are above normal. On the other hand, 80% of the patients who become hyperthyroid under amiodarone therapy have higher than normal thyroid iodine, the mean content being 2.8 times normal (*2*). We look forward to reviewing Prof. Jonckheer's data upon publication and comparing it with ours.

In Graves' disease, the thyroid iodine content was normal in 58% of our cases, high in 20%, and low in only 22%, which is at variance with Jonckheer's data (*3*). However, we agree that our statement should have been more precise. Furthermore the total iodine content in most iodine-induced thyrotoxicosis is high, whereas most patients with Graves' disease have a normal *or low* thyroid iodine content (*1*). In fact, what we wished to emphasize is the lack of elevation of the thyroid iodine content among patients with Graves' disease. This stresses the role of x-ray fluorescence in discussions of the hypothesized mechanism of iodine-induced hyperthyroidism.

We agree that patients with antithyroid antibodies, when treated with amiodarone, should be followed up very closely. We have shown recently (*4*) that in one third of these patients with thyroid antibodies, iodine supplementation (500 μg/day) progressively increased the thyroid iodine content, up to very high levels in some cases; nevertheless, none of these patients became hyperthyroid. In patients with high cardiovascular risk, measurements of the thyroid iodine content during amiodarone therapy could prove to be useful in the prediction of amiodarone-induced hyperthyroidism.

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Re: Thyroid Iodine Content Measured by X-Ray Fluorescence in Amiodarone-Induced Thyrotoxicosis: Concise Communication

The report of Léger et al. (*1*) in the July issue of the *Journal* is interesting in that the syndrome described differs markedly from iodine-induced thyrotoxicosis occurring in other parts of the world. I would make the following points:

1. The incidence in this series appears to be much greater than the 80 per 100,000 population that is usual in the Iodbasedow syndrome (*2*).
2. Decreased radioiodine uptake was certainly not a feature in our series.
3. In the great majority of cases from other centers, once thyrotoxicosis has developed it continues despite iodine withdrawal.
4. Response to antithyroid drugs was a feature in our patients.

It is noteworthy that the amount of iodine consumed by the French patients was much higher than that required in others, and that the "hardness" of their glands was so obvious. When describing the Iodbasedow phenomenon in Tasmania, we found that clinical assessment was a mandatory part of our investigation, and this is curiously lacking in the article under discussion. Johns et al. (*3*) describe several patients who were clinically and biochemically euthyroid before amiodarone, but who developed biochemical but no clinical evidence of hyperthyroidism when taking this drug. Clinical thyrotoxicosis did not develop in these patients when the drug was continued.

I suspect that Léger et al. are describing the effect of excess iodine plus the peripheral and central effects of amiodarone, namely the combined effect of reduced T_4 to T_3 production with increased T_3 and increased TSH response to TRH, producing increased T_3 and T_4 output (*4*).

This is not Iodbasedow or iodine-induced thyrotoxicosis as described by Kocher (*5*), others, and ourselves.

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Reply

We were surprised at Dr. Connolly's suspicion that we have confused amiodarone-induced thyrotoxicosis with the known other