

**PROPRANOLOL AND <sup>131</sup>I IN THE TREATMENT OF****DIFFUSE THYROID HYPERPLASIA WITH HYPERTHYROIDISM**

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Under ordinary circumstances there is a definite delay between the establishment of the diagnosis of hyperthyroidism and the symptomatic improvement of the patient. This is true whether the patient is put on antithyroid medication, is prepared for surgery or is treated with radioactive iodine. In 1966 Krikler (1) reported symptomatic control of thyrotoxicosis by the use of an adrenergic beta receptor blocker, propranolol. Confirmatory reports of symptomatic improvement of the peripheral manifestations of thyrotoxicosis have been gradually accumulating over the past few years. Hadden's and Shanks' reports (2,3) showed that patients with severe thyrotoxicosis could be made comfortable and free from symptoms of hyperthyroidism by this management. Because of the preceding encouraging reports, we decided to study the value of propranolol in the symptomatic treatment of hyperthyroidism treated with <sup>131</sup>I.

**PATIENTS AND METHODS**

This study includes 18 consecutive patients, age 35-62, on whom a diagnosis of diffuse thyroid hyperplasia with hyperthyroidism was made by clinical and laboratory evaluation. On physical examination the gland was enlarged but not nodular and there was no suggestion of cold nodules on the scan. Our baseline laboratory evaluation included a 24-hr radioiodine uptake, a T<sub>3</sub> (Triosorb) and serum T<sub>4</sub> levels by column or radioactive T<sub>4</sub> by saturation analysis. Clinical and laboratory followup evaluations were scheduled at 2, 5 and 11 months after treatment and from then on an annual basis to detect the onset of hypothyroidism or relapse. The treatment consisted of the administration of 5 mCi of <sup>131</sup>I in capsule form preceded by or followed by the administration of 10-40 mg q.i.d. propranolol. Propranolol administration was monitored by the patient's physician and was discontinued when it was felt that there was no additional clinical need for symptomatic treatment. All were ambulatory patients who were not in cardiac decompensation; none were diabetic and only one had a history of allergic bronchitis.

**RESULTS**

There was prompt symptomatic improvement in all of the patients. This took place sometimes within a week or 10 days of the initiation of propranolol treatment and was manifested by a drop in pulse rate and moderate to striking relief of agitation and nervousness. Duration of propranolol therapy was quite variable, lasting up to 4 weeks, depending on the evaluation of the clinical need for its administration by the patient's physician. There were no side effects reported. There have been no clinical or laboratory relapses to date. The followup periods have ranged from 2 to 16 months. The response pattern of the thyroid-function tests was predictable. There was uniform depression of uptake in all cases at the 2 month followup, while normalization of T<sub>3</sub> and T<sub>4</sub> levels lagged behind in four of the patients. By the fifth-month followup none of the thyroid-function tests were at the hyperthyroid level. The 24-hr uptake values had returned to normal in 13 of the patients. Five patients had developed clinical and/or laboratory signs of hypothyroidism and were put on replacement therapy. No additional instances of hypothyroidism have been detected as of this time.

**DISCUSSION**

As stated by Hadden *et al* (2) patients with thyrotoxicosis treated by this method have been rendered comfortable and free from symptoms of hyperthyroidism. Their management has been simplified, and no side effects were noted. It is generally accepted that the symptomatic effect of propranolol therapy in patients with thyrotoxicosis is due to its beta adrenergic blocking effect. However, a recent communication by Burke (4) suggests that there may be a direct effect on the basal and stimulated glucose consumption by the thyroid cells.

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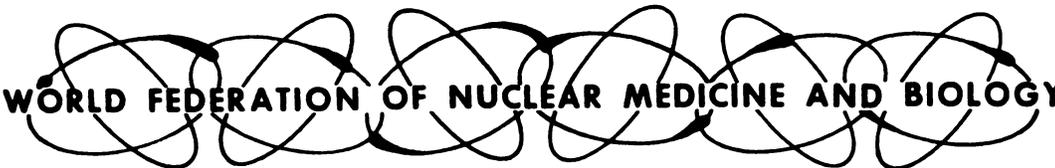
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## CONCLUSIONS AND SUMMARY

Eighteen consecutive patients with diffuse thyroid hyperplasia and thyrotoxicosis have been treated with a single dose of 5 mCi of  $^{131}\text{I}$  in capsule form and 10–40 mg q.i.d. of propranolol (a beta adrenergic blocking agent). Symptomatic improvement was immediate. There have been no laboratory or clinical relapses during followup periods of up to 16 months. The uniformly good symptomatic response and absence of side effects recommend additional evaluation of this combined therapeutic approach.

## REFERENCES

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