## Propranolol and radioactive iodine in the treatment of thyrotoxicosis

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Radioactive iodine (RAI) has been used for the treatment of thyrotoxicosis for more than 25 years. As it does not affect thyroid function for about 3-4 weeks, carbimazole has often been used with RAI, but this masks the effects of RAI on thyroid function. Propranolol controls many of the features of thyrotoxicosis (Shanks, Lowe, Hadden, McDevitt & Montgomery, 1969) and makes many patients clinically euthyroid after 1-2 days of treatment (40-60 mg four times daily by mouth). As propranolol does not influence thyroid function (Hadden, Bell, McDevitt, Shanks, Montgomery & Weaver, 1969) it can be given in combination with RAI for the treatment of thyrotoxicosis without interfering with the normal indices for estimating thyroid function. In this way propranolol controls the clinical features of the disease while RAI is having its effect, and the precise effects of RAI on thyroid function can be followed.

One hundred and thirty-five patients with thyrotoxicosis have now been treated with propranolol and RAI. The first 60 were given 3.5-5.0 mCi (group 1) and the remainder 2.5 mCi (group 2) RAI. At the end of 6 months concentration of protein-bound iodine in the serum had returned to normal in 65% of patients in both groups. The incidence of hypothyroidism was similar in the two groups. Heart rate was reduced in all patients, but the increase in weight was significantly greater in those in which there was a reduction in concentration of serum protein-bound iodine. This suggests that patients with thyrotoxicosis may be divided into two groups, those who respond to radioactive iodine and those who do not. The response rate did not appear to depend on the dose used.

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## The effect of the acute intravenous administration of clonidine (St 155) on cerebral blood flow in man

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Cerebral blood flow was measured before and after the acute intravenous administration of clonidine in seven healthy, normotensive volunteers. This was effected by inhalation and extracranial monitoring of <sup>133</sup>Xe, according to the technique described by Veall & Mallett (1966), with correction for arterial recirculation and deconvolution of the decay curve into three components. This latter procedure was done with a small analogue computer, after the method of Crawley (1968). The first and second components were translated into flow, through grey and white matter respectively, in the conventional manner; and the last component was