continued medication is required, is therefore to be welcomed. Several procedures have been proposed in the past and tested experimentally. Stein and Nicoloff (1971) substituted triiodothyronine (T₃) for previous therapy for four weeks and measured protein-bound iodine and radioactive iodine uptake, and again after a further 10 days off all therapy. Removal of hormonal suppression allowed the thyroid/pituitary axis to recover function in euthyroid patients. Krugman and colleagues (1975) simply withdrew thyroid medication for 35 days, and found that serum thyroxine (T₄) and thyrotropin (TSH) levels reliably differentiated between euthyroid and hypothyroid (low T₄, high TSH) patients.

Now Rizzolo and Fischer (1982), writing from family practice in the USA, have used T₄ and TSH assays to investigate the 24 patients diagnosed as having hypothyroidism in their practice of some 6,000 enrolled patients (a prevalence of 4/1,000). Of these 24, only four had a diagnosis meeting modern criteria, that is a documented low T₄ and elevated TSH level before starting therapy. Only 10 of the remainder could be located and included in the study: four were on thyroxine after subtotal thyroidectomy, two after radioactive iodine therapy, and four had idiopathic hypothyroidism. The method of testing was simple: after basal blood tests, all selected patients were asked to discontinue thyroid hormone therapy for three weeks, when their T₄ and TSH were retested. The six patients with normal results were followed up three months later, and on retesting all were found to be euthyroid. Four with low T₄ and raised TSH were restarted on thyroxine for life. Clinical symptoms and signs did not correlate with biochemical findings.

In their similar study of 46 patients, Krugman and colleagues found that even those who became biochemically hypothyroid were not clinically hypothyroid until the fifth week. Since a low T₄ could occur up to 25 days after stopping therapy, a careful 35 days was advised between tests. At that time the hypothyroid could be reliably differentiated from the euthyroid by high TSH levels. Other workers have indicated that care is necessary with three groups of patients. In clinically euthyroid patients with Hashimoto's disease, raised TSH levels may be found with a normal T₄. This situation may also be found after radioactive iodine therapy for thyrotoxicosis. Presumably the pituitary is driving the thyroid harder to produce normal T₄ output from its remaining functioning tissue in both cases. Both instances would lead to medication being unnecessarily continued, and thus would err on the side of safety. Patients with pituitary disorders may respond variably and need special care.

Both these studies are small in number, and a larger one is obviously needed to substantiate their findings; ideally it should be done in the UK, where clinical practice may differ. Nonetheless, the implications are considerable. We may shortly have another strategy for eliminating unnecessary medication to join the slowly growing library of methods so far developed for digoxin (Manning and Brown, 1977), night sedation (Wells, 1973) and now tranquillizers (pages 745-752 in this issue of the Journal). The fine details, especially whether withdrawal should be for 21 or 35 days for maximum reliability, await further elucidation. At the moment, a follow-up examination at three months would be mandatory for those patients taken off treatment; because of the forgetfulness and apathy of hypothyroid patients, it is up to the general practitioner to ensure that this is done.

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**References**


**Epidemiology and research in a general practice**

GEORGE Ian Watson was well known throughout general practice, but particularly in the College as Honorary Director of the Epidemic Observation Unit, as a major figure on the Research Committee, and as a distinguished President from 1970 to 1972. His sudden death in 1979 came before he had finished writing a book that was eagerly awaited by his friends and colleagues, since it described his research in his own
general practice in Peaslake with special reference to the respiratory tract.

Only two other general practitioners in Europe have produced similar work: Pickles, who was a model for Ian Watson, reported his observations from his Yorkshire practice in Epidemiology in Country Practice in 1939, and in 1978 Huygen from Nijmegen in the Netherlands described a remarkable series of illnesses among the families in his care in The Medical Life Histories of Families. Epidemiology and Research in a General Practice completes the trio: it comprises 16 chapters of the unfinished book and nine previously published articles—all on the impact of viruses in general practice.

The focal point of all Ian Watson’s work was his day-to-day clinical practice. As a member of the Foundation Council he always believed that the work of the College should remain based on the everyday care of patients and his book stands as a monument to this theme, based as it is on years of meticulous observation. Viology has never been a natural subject for general practitioners, and even today treatment is severely limited. Nevertheless, the man who first reported Shere fever to the world was able more clearly than anyone else before or since to elucidate and understand what all of us see daily in our practices. With his eyes and ears alone he was one of the very few doctors who has ever been able to distinguish the different clinical syndromes of virus diseases at the bedside.

Ian Watson’s work, on which his book is based, achieved national and international recognition. He delivered both the James Mackenzie Lecture and William Victor Johnson Oration, and was awarded the Sir Charles Hastings Prize twice, the James Mackenzie Medal, and the James Mackenzie Prize.

Following a recommendation of the Awards Committee, the decision was taken by the College to publish this book posthumously, both as a contribution to the literature of general practice and as a memorial to one of the College’s greatest Presidents. The unfinished book was left in draft form and every effort has been made to bring the text and references to a standard of which Ian Watson would have approved.

Epidemiology and Research in a General Practice is based on a lifetime of observation and research. It has within its pages lessons for all general practitioners, but most of all it is the personal testimony of a great clinician.

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References

Published in a limited edition, the book can be obtained while stocks last from the Publications Sales Department, Royal College of General Practitioners, 14 Princes Gate, Hyde Park, London SW7 1PU, price £10 plus 50p postage. Payment should be made with order.

Health care in an academic family practice

The theoretical model of family practice is one of primary, comprehensive and continuous care to families. This model is not yet fully documented. In a test of the model’s reality, 130 regular users of an academic family practice were surveyed. Eighty-two (63 per cent) identified the practice as their usual source of care. Projected use of the practice within this subset ranged from 100 per cent for general health examination to 20 per cent for marital/sexual problems. Only 35 per cent of the other 156 members of these 82 patients’ households were said to use the practice as their usual source of care. To the extent that these findings can be generalized to other settings, it can be concluded that the health care utilization patterns of family medicine patients and their immediate family members fall short of the specialty’s expectations.

Abstinence symptoms after withdrawal of tranquillizing drugs: is there a common neurochemical mechanism?

Withdrawal of anxiolytic agents may result in a syndrome characterized in its most severe form by delirium and seizures. There is increasing evidence that anxiolytic drugs produce their pharmacological effects by enhancing brain gamma-aminobutyric acid (GABA) transmission. During long-term exposure to anxiolytics, brain GABA synapses show evidence of adaptive changes which would tend to reduce the effect of the anxiolytic and restore normal GABA function. Abrupt cessation of anxiolytic treatment might therefore lead to an acute reduction in GABA function; this change could underlie the severe symptoms associated with tranquillizer withdrawal.
