ports a case of tardive dyskinesia in a subject who received no drugs other than reserpine.

In an attempt to replicate the favorable results reported by previous investigators, six female patients, 42 to 60 years of age, currently receiving no neuroleptics, were selected because of severe dyskinesia of long duration. The average number of symptoms per patient was 5.5. Reserpine was administered in increasing doses to 4 mg per day, a level that was maintained for four weeks. At the end of this period, the response to reserpine was fair in three, and negligible in the other three patients. No subject was found to be substantially improved because at least one of the original symptoms was conspicuously present. Of all manifestations, oral dyskinesia seemed to be the least affected, whereas posture and gait improved most. In some cases, increased motility was reduced, only to be replaced by abnormal posture or tremor. In conclusion, the effects of reserpine on tardive dyskinesia were modest. Owing to the rarity of patients receiving high doses of reserpine, there is little information on the long-term effects of this drug.

GEORGE E. CRANE, M.D.
Baltimore, Md. Spring Grove State Hospital

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JOD-BASEDOW (OR JOD-COINDET)

To the Editor: The recent report by Vagenakis and his associates (N Engl J Med 287:523-527, 1972) regarding Jodbasedow in Boston represents a striking phenomenon. This is particularly evident because of the ubiquitous use of iodides in this population and the previous absence of published reports in this area on induction of thyrotoxicosis by iodides. The authors were as perplexed by their careful observations as are many of the *Journal* readers. Perhaps these findings were fortuitous events, or perhaps they represent an unusual expression of latent thyrotoxicosis in these particular patients. In any event, I suggest that broad limitations not be issued prematurely for the indicated uses of iodides in diagnosis and therapy. The editorial comment by Dr. Utiger (N Engl J Med 287:562-563, 1972), which cautions that "... it would clearly seem wise not to use pharmacologic quantities of iodides therapeutically in any patient who has evident thyroid disease or who has received thyroid-ablative treatment in the past, unless it is for the specific purpose of obtaining its antithyroid effect," is premature and not justifiable at present. Perhaps a wiser admonition would be for physicians to be aware of this unusual action of iodides and to report further objective observations similar to those reported by Vagenakis and his associates.

HERBERT A. SELENKOW, M.D.
Boston, Mass. Peter Bent Brigham Hospital

The above letter was referred to Dr. Utiger, who offers the following reply:

To the Editor: My suggestion that patients who have thyroid disease or have received thyroid-ablative therapy should not receive pharmacologic doses of iodides was made for two reasons. One is that, as described by Vagenakis and his co-workers,[1] hyperthyroidism may be induced in some patients with nontoxic nodular goiter. The frequency of this effect is obviously uncertain, and indeed it may be unusual. More important, I believe, is the hazard that iodide administration may result in hypothyroidism in patients who have Hashimoto's disease[2] and those who have received radioactive iodide treatment for hyperthyroidism.[3] It may occur in patients with other thyroid disorders as well. Certainly, patients with thyroid disease in the first two categories are not uncommon, and in them the frequency of hyperthyroidism during chronic inorganic iodide administration is substantial.[2,3] It is this hazard of iodide therapy that Dr. Selenkow seems to have overlooked.

ROBERT D. UTIGER, M.D.
Philadelphia, Pa. University of Pennsylvania

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To the Editor: Vagenakis and his collaborators have written a fascinating paper on iodide-induced thyrotoxicosis in Boston.[1] However, there is a slight inaccuracy in their historical introduction. They say that "The occurrence of hyperthyroidism after an increase in iodine intake, usually referred to as Jodbasedow, has been recognized since 1900." In actual fact, this condition was clearly described in 1821 by Coindet.[2] Since, in spite of the importance of Coindet's publication, it seems to be currently unknown and does not appear in textbooks on thyroid disease,[3] a brief description of his report may be of interest.

Jean-François Coindet was born in 1774 and practiced medicine in Geneva, a city that at that time had a large number of goitrous inhabitants. Coindet was familiar with the centuries-old method of treating goiter with burnt sponge and seaweed. In 1811, Courtois discovered purple crystals in vats that had been used to distill seaweed to produce saltpeter for Napoleon's army. This material was proved to be an element and named iodine by Gay-Lussac and Sir Humphrey Davy.[4] Coindet wisely deduced that iodine might be the active principle against goiter in both sponge and seaweed. In 1820, he reported that if he gave 6 to 10 drops of tincture of iodine three times a day to goitrous patients, many of the goiters disappeared in six to 10 weeks.[5]

The above events are well known, and Coindet is generally credited with being the first to introduce iodine therapy for simple goiter. What has apparently remained obscure is that in the following year he described certain complications among 150 patients whom he had treated with his new remedy.[2] These included accelerated pulse, palpitation, insomnia, increased appetite, rapid emaciation and muscular weakness. Sometimes, a decrease in the size of the goiter was concomitant with the other symptoms. In some of his patients these manifestations persisted for a long time.

The complications described by Coindet are quite typical of the signs and symptoms of thyrotoxicosis and thus should be considered the first documentation that the disease can be produced in patients with nontoxic goiter when large doses of iodine are administered. Indeed, since the publications of Graves[6] and Basedow[7] did not appear until 14 and 19 years later, respectively, one could argue for an eponymic designation of spontaneous thyrotoxicosis as "non-iodide-induced Coindet's disease" rather than that of iodide-induced hyperthyroidism "Jod-Basedow".

So far as I have been able to ascertain, although Coindet lived until 1834, he did not write further on the use of iodine in goiter.

MONTE A. GREER, M.D.
University of Oregon
Portland, Ore. Medical School

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HYPERTHYROIDISM WITHOUT CALLUSES

To the Editor: Of the many signs and symptoms of thyroid disease used to estimate overactivity and underactivity of the thyroid hormones' effects on peripheral tissues, one that has intrigued me seems largely unappreciated. Certain shin signs – namely fineness, warmth, increased sweating and smoothness – are well known in hyperthyroidism, and careful study of the patient's shin permits appreciation of these findings. However, one will also note that the calluses often observed in euthyroidism will gradually disappear within a few weeks of the onset of hyperthyroidism and will gradually reappear as the patient resumes a euthyroid state, provided hand use continues unchanged. Serial observation of callus changes permits one to estimate whether a patient is hyperthyroid, – often even how long she might have been so and whether she is recurrently hyperthyroid or euthyroid. Many patients easily recognize changes in calluses on their hands – making this an often useful sign in history taking and follow-up observation of the thyroid patient.

Farmington, Conn. RICHARD ALLEN DICKEY, M.D.

HYPOCHOLESTEROLEMIA IN IDIOPATHIC HIRSUTISM

To the Editor: Dr. J. F. Dingman has reported a decrease of the serum cholesterol level after metyrapone administration.[1] Okuno and Nakayama[2] observed that the serum cholesterol concentration was abnormally low in patients with untreated congenital adrenal hyperplasia (simple virilization) and elevated after corticosteroid therapy.

In two sisters with idiopathic hirsutism we similarly found a pronounced hypocholesterolemia that disappeared after corticosteroid therapy (Table 1). These results are in perfect agreement with the

Table 1. Effects of Corticosteroids on Serum Cholesterol and Urinary 17-Ketosteroids (17-KS), 17-Hydroxycorticosteroids (17-OHCS) and Plasma Testosterone in Two Sisters with Idiopathic Hirsutism.

Case No.	Age	Drug	Cholesterol	17-KS	17-OHCS	Testosterone
	yr		*mg/100 ml*	*mg/day*		*ng/100 ml*
1	18	None	83-120	19.4	8.4	105.0
		Dexamethasone, 2 mg/2 days	173	1.7	9.0	37.5
2	15	None	125	13.5	9.9	78.0
		Dexamethasone, 2 mg/2 days	160	1.6	6.7	7.5

suggestion of Okuno and Nakayama that in these cases hypocholesterolemia may be due to excess utilization of cholesterol for steroid synthesis. A further interesting and unexplained observation is the fact that the mother and brother of the two patients exhibited cholesterol values of 165 and 145 mg per 100 ml respectively, which are slightly decreased as compared to normal controls of the same age, but without clinical endocrinologic signs.

GIORGIO NOSEDA, M.D.
EVA SCHLUMPF
University Medical Clinic
Berne, Switzerland Tiefenau Hospital

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B AND T LYMPHOID CELL LINES

To the Editor: The dialogue (N Engl J Med 287:989-990, 1972) between Rubin's group and that of Aisenberg and Block concerning the origin of nongranulocytic leukemia from T or B cells has both theoretical and practical importance. Our experience in establishing over 800 human lymphoid cell lines may provide additional insight.[1]

To our knowledge all normal human lymphoid cell lines have B-cell characteristics such as the membrane receptor for complement and the ability to synthesize immunoglobulins. The cells are stimulated by Epstein–Barr virus (EBV), and the established cells contain the virus or its genome. These cells are large and blastoid and have a normal chromosome constitution. They also clump together and do not form rosettes with sheep red blood cells.

On the other hand, no normal lymphoid cell lines with predominant characteristics of T cells have been reported.

A number of abnormal lymphoid cell lines have been derived from the blood of patients with myelogenous or lymphatic leukemia, or myeloma. One cell line (MOLT) from a child with acute lymphatic leukemia[2] consists of small cells with characteristics of T cells. These cultured cells do not clump or form so-called or spontaneous rosettes[3] with red blood cells of sheep, goat, pig or horse, have an abnormal chromosomal constitution and have no receptors for complement. The cells do not secrete immunoglobulin and have no detectable EBV genome. A cell line (RPMI 8422) from a young woman with acute lymphatic leukemia has an abnormal karyotype and some characteristics of both T and B cells. This cell line has both small and large blastoid cells despite evidence of monoclonal origin. On one hand, the cells do not clump together, have no complement receptor sites, do not secrete immunoglobulins, and EB virus has not been detected in them. On the other hand, the cells do not form rosettes with sheep red blood cells.

Abnormal lymphoid cell lines established from the blood of patients with myeloma secrete light chains or complete immunoglobulin, but do not form clumps and do not contain EB virus, and the average size of the cells is smaller than that of normal lymphoid cell lines.

It is interesting that the cells of the cell lines MOLT and RPMI 8442 do not exhibit phagocytosis of dead cells as is commonly observed in the normal lymphoid cell lines with B-cell characteristics.

Cell lines are easier to establish from chronic lymphatic leukemia, and the cells are more nearly pure B cells, but the cells from other patients with that disease do not become established and may represent cells that are more like T cells. It is also very difficult to establish cell lines from some malignant lymphomas and from patients with Hodgkin's disease. The addition of EB virus is not helpful.

In our opinion an entire spectrum of malignant lymphoid cell lines will be discovered with varying expression of the characteristics of T cells or B cells, as well as some cells without the characteristics of either.

G. E. MOORE, M.D.
J. MINOWADA, M.D.
Albany, N.Y. State of New York Department of Health

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THE HUNTER SYNDROME IN A 46 XX GIRL

To the Editor: We wish to report a case of clinically expressed and biochemically characterized Hunter syndrome, in a girl of normal 46 XX karyotype. The X-linked recessive mode of inheritance of this mucopolysaccharidosis is documented by several extensive pedigrees.[1] The disease is believed to occur exclusively in hemizygous males. Females homozygous for the disorder are unknown, since affected males (with rare exception)[2] do not reproduce.

In normal phenotype of Hunter heterozygotes, the portion of the X-chromosome that bears the Hunter gene or its normal allele conforms to the Lyon hypothesis of random inactivation.[3,4] The hetero-