POLYCYTHAEMIA VERA (RUBRA) COMPLICATED
WITH HYPERTHYROIDISM.

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THE note on polycythaemia hypertonica¹ in the Epitome of Current Medical Literature of October 25th, p. 14, induces me to think the following case may be of interest:

A man, aged 45, came to consult me regarding his high blood pressure. He had been under treatment for this condition for over two years, but his symptoms had increased in severity, and he was now subject to recurrent attacks of vertigo, which rather alarmed him. The history was one of gradually increasing cyanosis during the last eight years, some shortness of breath, and increasing muscular weakness. About April, 1918, some bombs which fell near his house during an air raid greatly upset him. Within twenty-four hours this was followed by swelling of the neck and thyroid gland, which became as large as two duck's eggs. The swelling gradually receded until it became the size of a hen's egg and had remained in that condition. From this time he became nervous and exhibited signs of hyperthyroidism. During the last month he had had several attacks of giddiness with some confusion of mind, lasting a few seconds. There was no history of specific disease.

His face showed a well-marked degree of cyanosis. The superficial temporal arteries were tortuous and thickened. The radial pulse felt hard and gave the impression of high tension. The artery was thickened. The blood pressure, taken on three separate occasions, was always about 120 mm. Hg. The heart was not enlarged. The urine contained a trace of albumin, but microscopic examination showed no casts or anything abnormal. The spleen was enlarged and reached to 3 in. below the costal margin. The right lobe of the thyroid gland was prominent, the swelling being as large as a hen's egg. There was slight exophthalmos.

A complete blood examination was made, with the following result:

Red blood corpuscles...	...	9,600,000 per c.mm.
White blood corpuscles...	...	25,000 "
Haemoglobin...	...	125 per cent.
Colour index...	...	0.8

A differential blood count revealed:

Polymorphonuclear neutrophils...	...	80.8 per cent.
Small lymphocytes...	...	10.4 "
Large lymphocytes...	...	5.6 "
Eosinophiles...	...	0.4 "
Mast cells...	...	2.8 "
Myelocytes...	...	0.0

There was therefore both polycythaemia and leucocytosis.

In the literature on polycythaemia, I could find no case mentioned in which the thyroid gland was involved. This must be an unusual complication. There are apparently two varieties of polycythaemia, each with its own well-marked characteristics:

(a) *Polycythaemia Hypertonica*, which has been described by Geisböck² and also by Stähelin,³ in which there occurs a high blood pressure, hypertrophied heart, an increased number of red blood corpuscles up to six to eight million or more per c.mm., a pulse which is generally regular, but prone to extra-systoles. No increase in size of the spleen. Arterio-sclerosis and nephritis.

(b) *Polycythaemia Vera*, as described by Vaquez and Osler,⁴ and *Polycythaemia Rubra*, mentioned by Clifford Allbutt.⁵ These appear to be the same condition, in which the red blood corpuscles may reach to ten million or more per c.mm. The arteries are thickened and tortuous, but with normal blood pressure and no hypertrophy of heart. The spleen is enlarged. The urine contains a trace of albumin.

Osler says that the three cardinal features are: a change in the appearance of the patient, enlargement of the spleen, hyperglobulism; there is a moderate leucocytosis without any characteristic differential change; the red corpuscles may reach to even thirteen million. As a rule they are normal in appearance and shape.

The blood is always very viscous; this was quite marked in the case here recorded, and some effort was required to draw the blood up into the pipette. This gives one the impression that clotting may readily occur in the capillaries if the pulse becomes slowed and the blood pressure low. A heart which is not hypertrophied must feel the strain of driving such an increased mass through thickened and narrowed vessels. It may be that the attacks of "vertigo and confusion" complained of had some connexion with this increased viscosity. It has been stated⁶ that in epilepsy the coagulation time is much shortened. It is only reasonable, therefore, to assume that the

"attacks" are something in the nature of petit mal; the description seems to fit in with the latter.

In the treatment of viscosity iodine has been suggested. My patient says he has been treated with potassium iodide for over two years, with apparently no effect on the viscosity. It must be remembered also that there has been considerable hyperactivity of the thyroid gland during the last eighteen months. If iodine be one of the chief active ingredients of thyroid secretion when the thyroid gland is overacting, this man's blood must have become saturated with iodine—and still there is excessive viscosity.

McCarrison says⁷ blood changes of a fairly definite character occur in endemic goitre. The number of erythrocytes is diminished as well as the haemoglobin index. The coagulability of the blood is increased; the total number of leucocytes is reduced. The blood changes of exophthalmic goitre resemble those of endemic goitre.

It is reasonable to think, perhaps, that the administration of thyroid gland substance may be of benefit in these cases. In the case quoted above there are symptoms of hyperactivity of the gland and the thyroid is considerably enlarged; but is it not a fact that in hyperactivity the internal secretion is perverted in character? It is excessive in quantity but deficient in quality. It would be interesting to know whether thyroid has ever been used in the treatment of polycythaemia, and, if so, what was the result.

REFERENCES.

- ¹ Epitome of Current Medical Literature, BRITISH MEDICAL JOURNAL, October 25th, 1919. ² Geisböck, *Deut. Arch. f. klin. Med.*, v. 1, lxxxiii, 1905, p. 395 quoted by Sir Clifford Allbutt. ³ Stähelin, *Berl. klin. Woch.*, January 16th, 1911 (quoted by Sir Clifford Allbutt). ⁴ Osler, *Principles and Practice of Medicine*, 8th edition. ⁵ Clifford Allbutt, *Diseases of the Arteries*, Part I. ⁶ Turner, J., *Journ. of Mental Sci.*, October, 1907 (quoted by Sir Clifford Allbutt). ⁷ McCarrison, R., *The Thyroid Gland in Health and Disease*, 1917.

FATAL ANAPHYLAXIS FOLLOWING PROPHYLACTIC
INJECTION OF DIPHTHERIA
ANTITOXIN SUBCUTANEOUSLY.

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THE death here recorded was apparently an instance of severe anaphylaxis. Sir William Osler mentions the rarity of the condition and alludes to the symptoms of fatal cases, namely, extreme distress, dyspnoea, cyanosis, respiratory failure, and collapse. He warns against inoculation of persons who have been previously inoculated with antitoxin, persons subject to asthma, and persons susceptible to the proximity and smell of horses. There is no evidence of this boy having been previously inoculated with antitoxin, and on my arrival at the house he was playing on a coal cart drawn by a horse. The bronchial colds mentioned later may have partaken of the character of asthma. Sir William Hale-White mentions two fatal cases occurring rapidly after the injection of diphtheria antitoxin.

On September 25th, 1919, I gave an apparently healthy boy, aged 8 years, a subcutaneous injection of diphtheria antitoxin; he collapsed and died within five minutes. The injection was made at his home, and was given as a prophylactic, his mother having been removed on the previous evening to Preston Isolation Hospital, suffering from faucial diphtheria. The serum used was supplied by the Municipal Health Department of Preston, and was marked on the cover as potent until January 1st, 1921. The dose given was 2,000 units. The injection was made subcutaneously through the skin, previously painted with tincture of iodine, to the inner side of the angle of the right scapula, and the syringe used was an all-glass Burroughs, Wellcome and Co.'s serum syringe, which had been previously boiled for five minutes. Following the injection of the boy, his three younger sisters were injected with the same dose and in a similar manner without, at the time of writing (September 28th), any bad result. Within two minutes of the boy's inoculation he complained that "it had gone to his stomach," and he immediately ran out of the kitchen of the house, where the injection was made, to the w.c. in the yard. A minute later I heard him call out "Daddy! Daddy!" and his father immediately ran out to him and as quickly returned and informed me that his boy was very ill and was apparently choking. I followed the father into the yard and met him carrying the boy in his arms from the w.c. The boy's face was deeply cyanosed, and he was apparently collapsed. I carried him quickly into the kitchen, placed him on the sofa, with his head hanging over the edge, and after pulling out his tongue and swabbing from the pharynx a considerable collection of mucus, commenced artificial respiration. At this time the heart beats and