POLYCYTHAEMIA VERA (RUBRA) COMPLICATED
WITH HYPERTHYROIDISM.

BY

EDGAR J. TYRRELL, M.B., B.S.DURH.,
LONDON, E.C.

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. l. xxxiii, 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.

BY

A. D. MCCALLUM, M.B., CH.B. EDIN.,
PRESTON.

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

respirations had ceased, and the pupils were widely dilated. Other medical aid was summoned and artificial respiration continued for three-quarters of an hour without avail.

A *post-mortem* examination was conducted by Dr. Pilkington the following day, and, except for a mass of caseous tuberculous glands in the mesentery, all the organs were healthy. There was no evidence in the pharynx, glottis, or larynx that the boy had recently suffered from diphtheria. The left ventricle of the heart was contracted and empty; the right ventricle was flaccid and contained dark fluid blood.

The boy's parents are healthy, and from his previous history it appears that, except for susceptibility to bronchial colds during the winter, he enjoyed good health.

I may, perhaps, add that at the *post-mortem* examination the stomach was found to contain about half a pint of undigested food. The injection was made at 10 a.m. and the last meal (breakfast), consisting of bacon, bread-and-butter, and tea, was taken at 8.30 a.m.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

CALCIUM CHLORIDE AS A HAEMOSTATIC.

RECENTLY a man, aged 25, consulted me for enlargement of the right tonsil. The organ was the size of an ordinary walnut, and removal was advised. He elected to have this done at once, and also decided against a local or general anaesthetic. The tonsil was easily removed by a spade guillotine; bleeding was slight, and the man walked away a few minutes afterwards. An hour later I was hastily summoned, and found him somewhat blanched, throwing up blood clot every few seconds, and with a pail before him which must have contained a pint of blood at least. Attempts to localize the bleeding spot by examination only caused more violent retching and gagging with more bleeding, and for the same reason spraying with a styptic also failed. The bleeding was venous, and an attempt to arrest by digital pressure failed for the same reason as the other measures. At last I injected into his gluteal muscle a hypodermic syringe of a 1 in 4 solution of calcium chloride, following this with a similar injection into the other gluteus. The amount of the salt injected was 8 to 10 grains. The effect was striking; the bleeding ceased in a minute and a half and did not recur. He passed the night in a half-sitting posture on the couch, and no subsequent recurrence took place.

A week afterwards he came up with a slight but persistent return of the venous oozing, which was at once arrested by injecting 4 grains of calcium chloride into the gluteus as before; it did not recur.

It is to Dr. Grove of St. Ives, Cambridgeshire, that I am indebted for the knowledge of this measure; indeed, many local practitioners will remember the valuable paper he read some years ago, at a meeting of the Cambridge Medical Society, on the uses of calcium chloride in practice.

Histon, Cambs.

L. GWILLIM DAVIES, M.D. Cantab.

ACUTE SUFFOCATIVE CATARRH.

THE case recorded by Dr. C. O. Jones (October 11th, p. 466) recalls two cases which I saw within a couple of years in a working-class practice in Scotland. The first was in a woman of 30 years, who was seven months pregnant. The history was that, after washing down the outside stair leading to her house about 9 p.m., she was attacked with sudden breathlessness and was with difficulty able to ascend the stair. A neighbour assisted her into bed and sent for me. When I arrived she was half reclining on her right side and gasping for breath as if she had a very severe attack of asthma. A quantity of pink froth was coming from her mouth; I calculated that from beginning to end she brought up about a pint. The whole chest was full of râles; the pulse was rapid and feeble. She died within an hour of the onset of the attack. Her family doctor informed me that, with the exception of an attack of pleurisy some years before, she had always enjoyed good health.

The second case was in a man aged 28. The attack came on while he was asleep. There was the same dyspnoea as in the first case, and a large quantity of froth was brought up, but it had not a pink colour. There was a history of chronic chest trouble which I took to be

tuberculous, but the râles all over the chest prevented my being able to locate it. I sent the man into hospital at once, but he died within a few hours of the onset of the attack.

Birmingham.

ROBERT ANDERSON, M.D.

Reports of Societies.

TUBERCULOSIS AND LIFE ASSURANCE.

At a meeting of the Assurance Medical Society held on November 5th, with the President, Dr. F. PARKES WEBER, in the chair, Dr. OTTO MAY read a paper on some aspects of the tuberculosis problem in life assurance, namely, (i) the significance of a tuberculous family history; (ii) the question of "contact-infection"; (iii) the assurance of individuals with a personal history of tuberculosis. Under the first head he dealt with the universal experience that tuberculosis "runs in families." The frequency with which three, four, or even more members of a family died of this infection was far greater than could be accounted for by any fortuitous incidence of the disease. But the explanation was still a matter of debate.

Even if an inherited "predisposition," or "lack of resistance," were accepted, it must not be assumed that each individual of a tuberculous family was specially susceptible to this disease. For this reason we must guard against the assumption that a tuberculous family history necessarily rendered the proposer an impaired life, acceptable only at special rates. The question was one for decision by the actuary rather than the medical man. Some light was thrown on this problem by an investigation by Rusher and Kenchington, with the assistance of the late Dr. E. M. Light.¹

They investigated the mortality in a group of some 80,000 proposers with a family history of tuberculosis, the total experience comprising 622,462 policy-years, with an average observation period for each proposer of nearly eight years. The data were grouped in various ways, according to age at entry, sex, nature of insurance (whole life and endowment), and details of family history—whether the tuberculous deaths were in lineals (father and mother), collaterals (brothers and sisters), or lineals and collaterals.

The results, expressed in ratios of actual to expected deaths, varied considerably in different groups, but the chief findings might be roughly summarized as follows:

1. The mortality experienced in the early years of assurance was heavy, especially for young ages at entry. In other words, the effect of a definite tuberculous family history shows itself mainly in the younger ages at entry and the early years of assurance.

2. After the first five years of assurance the excess mortality is less marked, especially in the case of endowment assurances, in which it comes well within normal limits. The latter fact suggests the importance of "selection" by the assured—of the "bad life" more or less consciously choosing a whole-life assurance rather than an endowment.

3. As regards the relative importance of lineals and collaterals in this respect, the authors "have no hesitation in expressing the opinion that at least as much attention—if not more—must be given to a history of tuberculosis among brothers and sisters as amongst parents."

4. The degree of tuberculous family history is of much less importance than the age at entry, and may, in fact, be said to have no practical value for assurance purposes over age 35 at entry.

In 1914 appeared an elaborate medico actuarial investigation, embodying the combined experience of the leading American companies with regard to various classes of special risks. The findings with regard to tuberculous family history were particularly interesting, as in most of the groups, in addition to the age at entry, the weight, and in some cases the height, were taken into account.

The logical conclusion from these data was that, in estimating the importance of a positive family history of tuberculosis, two additional factors must always be taken into account: (1) age at entry, (2) weight and build.

In proposers under 35 the risk should be treated as increased unless all other circumstances were unimpeachable—weight (not below good average), occupation, habits, and past history (freedom from any suspicion of tuberculous disease). If any one of these factors was unfavourable in a proposer under 35, a considerable debt should be placed on the policy; if two factors, for example, weight and occupation, were unfavourable, the

¹ *Journal of the Institute of Actuaries*, 1913.