TRANSACTIONS

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PHILADELPHIA ACADEMY OF SURGERY

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The Vice-President, DR. WALTER E. LEE, in the Chair

CALVIN M. SMYTH, JR., M.D., Recorder

TOXIC GOITRE WITH PARALYSIS OF EXTRA-OCULAR MUSCLES

DR. EDWARD J. KLOPP and, by invitation, DR. E. G. SHANNON, presented a man aged forty-five, a coal miner by occupation, who was admitted to the Jefferson Hospital, September 29, 1933, with the diagnosis of exophthalmic goitre. His chief complaints were nervousness, excessive perspiration, marked tremor of both hands, palpitation of the heart, staring of both eyes, diplopia and loss of weight. He has been a miner for the past twenty-five years, working from seven to eight hours per day, and has never been financially distressed. In August, 1932, there was an onset of marked nervousness with tremor of both hands and perspiration of the entire body. There was some gastric disturbance in the early part of August, during which he vomitted once. He was treated by a physician who diagnosed his case as "miners' asthma" on account of cough, expectoration and shortness of breath on exertion. He has not been able to work for the past five months.

The spinal fluid was practically normal. The Wassermann and Kahn reactions were negative. Metabolic rate on September 30 was plus 51; October 10, plus 29; October 18, plus 17; October 27, plus 15, and November 17, plus 25.

The patient was prepared for subtotal thyroidectomy by rest in bed and small doses of Lugol's solution. At operation under avertin, supplemented with nitrous-oxide anæsthesia October 31, 1933, nothing unusual was encountered. The most important symptom was the unusual paralysis of some of the extra-ocular muscles.

During his first illness in August, 1932, there was no clinical evidence of any ocular impairment. He was able to return to work in October. In January, three months later, he developed tearing of the right eye, followed by marked œdema of the right eyelid. With the subsidence of these symptoms he found he could not raise the right eyeball and that all objects appeared double except in the lower field. Five months later, the left eyeball was similarly affected and on October 2, 1933, both eyes exhibited an almost complete paralysis of the upward movements of both eyeballs, indicating an involvement of both superior recti muscles. In addition, the inferior oblique muscles were affected as no upward-outward rotation could be obtained. The inferior recti muscles being unopposed, it was noted that both eyes were turned definitely downwards, the right eye slightly lower than the left. (Fig. 1.) The left eye could be elevated slightly, the right eye apparently not at all. With binocular single vision obtained only in the lower field, it was necessary for the patient to tilt his head rather sharply backwards. Since the subtotal thyroidectomy on October 31, a little over a month ago, he feels

that he can now bring his head nearer the erect position and still maintain single vision.

In attempting convergence, the right eye looked straight ahead while the left deviated in slightly. It is also interesting to note that with the eyes rotated to the extreme right the right eye turned downward, while with the

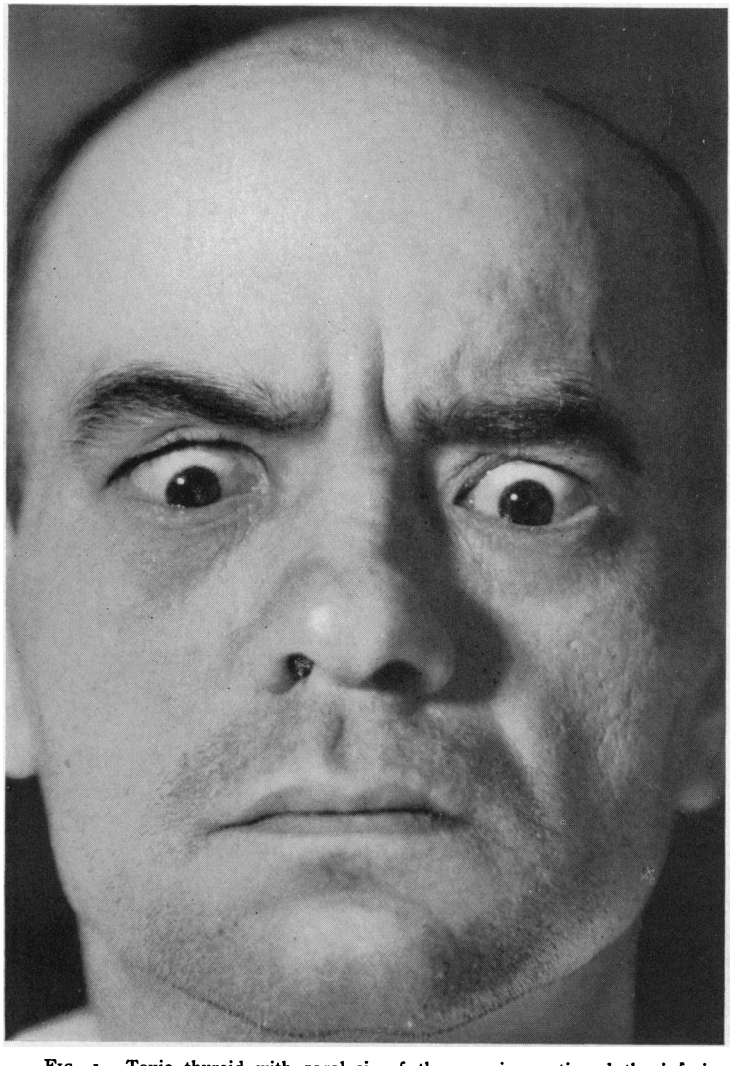

FIG. 1.—Toxic thyroid with paralysis of the superior recti and the inferior oblique muscles of both eyes with slight impairment of function of the externi and interni muscles.

eyes rotated to the left, the left eye was similarly deviated downwards, indicating an over-acting superior oblique muscle. In addition to the paralysis of the superior recti and inferior obliques, the function of the externi and interni muscles was slightly impaired. The fields for white and color were within normal limits and the eye-grounds, aside from some angiosclerosis, were negative.

The ocular symptoms that may accompany Graves' disease naturally draw the interest of the ophthalmologist. The symptoms were striking and important and it seems appropriate in connection with this report, before touching upon the unusual muscular complication in this case, to briefly enumerate them.

The explanations for the development of this condition are varied and as follows:

That it is due (1) to engorgement of orbital vessels; (2) to contraction of unstriated muscle fibres in the orbit, running from the equator of the eye to the orbital septum; (3) to deposition of fat in the orbit with œdema of ocular muscles. According to Foster-Moore[6] the last explanation is the most tenable.

The speaker remarked that paralysis of the extra-ocular muscles in toxic goitre is rarely seen. Cases have been reported by Ballet,[7] Liebrecht,[8] Buschan,[9] Manheim,[2] Lang and Pringle,[10] West[11] and others, in some of which other cranial nerves have been involved. Palsy of the intrinsic muscles—sphincter of the iris and ciliary body—has not been recorded nor cases of associated movements of the eyes, with the exception of cases of paralysis of convergence by Schmidt-Rimpler[12] and Vossius.[13] The occurrence of paralysis of, first, the right superior rectus and five months later, of the left superior rectus, would not support the view that this case was one of an associated paralysis of the superior recti. Bristowe[14] reported a case of ophthalmoplegia externa in a young man three years after the symptoms of Basedow's disease had developed. Later loss of smell and taste occurred. Warner[15] had reported a case of binocular external ophthalmoplegia with palsy of the facial and trigeminal nerves. Voss[16] has reported two cases of palsy of the extra-ocular muscles in exophthalmic goitre. Brain[17] has recently reported five cases of enlarged thyroid and muscular disturbances. He offered no explanation of the relationship between the two conditions.

The interesting question arises as to the cause of the defective ocular movements. Various theories are advanced but the most acceptable is that of Foster-Moore,[6] who believes that they are due to changes in the muscles rather than in the nerve of supply, a view, as he states, "which is supported by the distribution and by the fact that definite changes have been found in the muscles." This change, as he noted in one of his cases, was due to fatty infiltration of muscles.

BIBLIOGRAPHY

[1] Dock: Osler's "System," vol. 5, p. 862, 1915.
[2] Manheim: Der Morbus Gravesii. Berlin, p. 75, 1894.
[3] Fuchs: Fuchs' Text-book of Ophthalmology, p. 818.
[4] Willbrandt and Saenger: Die Neur. des Auges, vcl. 1, p. 50, 1900.
[5] Falta: Die Krankungen der Blutdrüssen, Berlin, p. 50, 1913.
[6] Foster-Moore: Medical Ophthalmology, p. 174.
[7] Ballet: Gazette Hebd., p. 558, 1888.

[8] Liebrecht: Bemerkenswerthe Fälle von Morbus Basedowii aus der Prof. Schöllerschen Klinik, Klin. Monatsblat f. Augenheilk. S. 492, 1890.
[9] Buschan: Die Basedowsche Krankheit, Leipsig u. Wien, 1894.
[10] Lang and Pringle: Trans. Ophth. Soc., U. K., vol. 6, p. 105, 1886.
[11] West: Trans. Ophth. Soc., U. K., vol. 6, p. 79, 1886.
[12] Schmidt-Rimpler: Nothnagel's system, 1898.
[13] Vossius: Beiträge zur Augenheilkunde, vol. 18, 1895.
[14] Bristowe: Brain, p. 313, 1886.
[15] Warner: On Ophthalmoplegia Externa Complicating a Case of Grave's Disease. Med. Times and Gaz., p. 540, 1882; Lancet, vol. 2, p. 104, 1882.
[16] Voss: Deutsche Med. Wochenschrift, August 13, 1903.
[17] Brain: British Med. Jour., November, 1932.

DR. THOMAS A. SHALLOW remarked that one could hardly believe that the same lesion which produced the bone changes in this man was responsible for the blindness and deafness. When we consider those factors about the eyes and ear, we are struck by this fact that in oxycephalus, which is a condition known as tower head, there is an early fusion of the sutures at the base of the skull. As a result, the subsequent development of the brain makes pressure and destroys some of the basal structures, particularly the first, third, fourth and sixth nerves. This belief is fortified by Doctor Keeler's Baranay test and by Doctor McAndrew's study of this child's eyes. There is quite a dispute about the consistency of the bone. Some agree that the bones are soft, others say that they are hard, and a review of the pictures shown by Doctor Davis indicates there has been softening at some time. There is no history of fracture, so here we have a condition which resembles Paget's disease and the reports of other cases show there is only bending as in this patient, but there is softening and, at a later stage, hardening of the bones.

When we consider the pathology in osteitis fibrosa cystica, which is caused by hyperparathyroidism and find that this produces osteoporosis and cystic change in the bone, we cannot help, by comparison, to classify osteopetrosis as hypoparathyroidism, since excessive lime-salt deposits are found in this disorder.

SOLITARY CYST OF THE FALCIFORM LIGAMENT

DR. HENRY K. SEELAUS remarked that among the 107 reported cases of solitary non-parasitic cysts of the liver, only three have been recorded as occurring in the hepatic ligaments, two in the falciform ligament, one by Dujarrie and the other by Wakely and MacMyn, and the third, in the round ligament by Bevan. The present report constitutes the fourth recorded case.

The patient, a girl, aged seven, was admitted to the children's ward of the Jefferson Hospital in the service of Doctor Bauer, July 11, 1933, with a diagnosis of hepatic cirrhosis with ascites. The present illness began three years ago with an enlargement of the abdomen which had gradually but progressively increased. In November, 1931, she was operated upon in another city, the operative note being that the liver was increased to ten times its normal size with an accompanying ascites. Nothing further was done and

the incision was closed without drainage, healed without complications and the patient sent home in ten days. Following her discharge from the hospital the abdominal enlargement increased and she was referred to the Jefferson Hospital. On physical examination the abdomen was markedly enlarged, distended and rounded, the superficial veins, including those of the umbilical region, being very much dilated. There was fluid present, the characteristic wave being elicited. An X-ray film of the abdomen confirmed the findings regarding fluid but there was so much fluid present that it prevented any further interpretation regarding any abdominal masses. A film of the pericardium and heart was negative for any evidence of Niemann-Pick's disease. The bromsuphalein retention study of the liver indicated normal hepatic function; there was a positive indirect van den Bergh reaction and the quantitative van den Bergh study was .42 mg. per 100 cc. of blood. Under the impression that we were dealing with an instance of juvenile hepatic cirrhosis with ascites the abdomen was opened under ether anæsthesia through an upper Moynihan incision. Instead of finding an enlarged liver with ascites we encountered a large cyst adherent to the under surface of the liver and the great omentum. Being mindful of Wangensteen and Scott's work on shock following the sudden removal of large amounts of fluid from the peritoneal cavity we incised the fibrous wall of the cyst and with the aspirating set evacuated the cyst by gradual decompression, removing 3,850 cc. of a brownish, muddy fluid. The collapsed cyst was then easily shelled out from the folds of the falciform ligament and the under surface of the liver. The gall-bladder and the extrahepatic bile-ducts were entirely separate from the cyst and after its removal the two leaflets of the falciform ligament were brought together with a few interrupted catgut stitches. The oozing from the raw surface of the liver was controlled by pressing a hot moist gauze pad against it. The abdomen was closed with one cigarette drain carried down to the hepatic area. The patient reacted well from the operation and was discharged from the hospital three and a half weeks afterwards.

The fluid contained albumin and blood, no bile, a few pus-cells and many red blood-cells and débris.

Pathological Examination by Doctor Crawford.—Specimen consists of a cystic mass which has been emptied of its contents weighing 575 Gm. and measuring 19 by 17 by 15 cm. The external surface is white, smooth and glistening, and there are several ragged areas about 2 cm. in diameter, scattered over the surface. The cyst wall varies from ¼ cm. to 1½ cm. in thickness. The wall is composed of a very dense grayish white tissue. The inner surface is smooth and covered with a slimy, brownish-yellow material.

Histology.—Examination of sections from the wall of the cyst reveals that it is composed of very dense fibrous tissue; in some places, the tissue is hyalinized. No other structure such as muscle tissue is observed, and in the number of sections examined no evidence of an epithelial lining to the cyst could be demonstrated.