statements will on investigation put the unused static machine in service they will find it one of the most resourceful means at command, and those who have not one will find an installation a profitable investment for themselves and their patients.

THE ETIOLOGY AND TREATMENT OF HYPERTROPHY OF THE PROSTATE.

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It would seem that if hypertrophy of the prostate is due to atheroma, as is generally supposed, it would occur in more than 25 to 30 per cent. of all males past middle life, and I, therefore, believe that it is due entirely, or nearly so, to congestion or inflammation. Atheroma is frequent without hypertrophy, and *vice versa*.

Acute and subacute inflammations everywhere give rise to hyperplasia, and especially is this true in the prostate, which is abundantly supplied with blood vessels and highly cellular connective tissue. In the liver, kidney, spleen or any other glands of the body, hypertrophy or interstitial hyperplasia is always preceded by congestion or inflammation. These conclusions are the result of clinical observation and not of laboratory research. The general laws of pathology justify them.

The arteriosclerosis theory of Guyon has practically no adherents; nearly all authorities admit that congestion and inflammation will produce hyperplasia, and that this plays an important part in many cases of hypertrophy, the only difference of opinion being as to the manner in which hyperplasia produces hypertrophy.

As a basis for these conclusions, from a clinical standpoint, is the frequency with which hypertrophy occurs in comparatively young people. I have seen it in a boy 8 years old and in a number of men between 30 and 40, one of whom had complete retention for twelve years until operated on at 37. The claim may be made that the enlargement in the young is only a prostatitis. That is true, but when this inflammation has persisted until it produces obstruction it is as truly a hypertrophy as is adenoma of the prostate.

Inflammation is an effort on the part of Nature to repair an injury. The first stage in the process is congestion, which causes exudation of fluids into the tissues and also an infiltration of small round cells. When this infiltration has become a chronic process the new tissue becomes organized, and this is the beginning of the hypertrophy. That it is a true hypertrophy is not at all probable, as it occurs at a period of life when the functional activity of the gland is on the decline, while in other glandular organs it begins at a much earlier period.

Hypertrophy is an excessive growth or development of an organ. True, the action of a muscle tends to increase the circulation, and if long continued there follows hypertrophy or increase of substance, a physiologic process. Increased circulation, plus infection, will cause exudation of small round cells and hyperplasia—a pathologic condition. As this pathologic process continues the elements of the gland, whether glandular or fibrous or both, become increased. The glandular elements are increased first, the fibrous elements secondarily. Naturally, one expects the glands immediately surrounding the urethra to become involved first, and pathologists have shown that the central glands are first involved.

No reason can be given why the glandular form should be so much more frequent than the interstitial other than the continued irritation produced by the retained secretion of the acini, from complete or partial occlusion of the excretory ducts by the small round-celled infiltration.

After the occlusion of the glands the interstitial hyperplasia may cease, but the retained secretion will keep up the irritation of the acini, causing a multiplication not only of the cells but also of the acini. This is a plausible explanation of the occurrence of the adenomatous form in from 80 to 85 per cent. of the cases of hypertrophy. Furthermore, the fibrous and fibromyomatous form is most frequent in the young.

In the case of the boy of 8 the infection followed catheterization during an attack of typhoid, and it has continued to the present time. The boy now has a large fibrous gland, the enlargement being principally in the median lobe and practically free from inflammation. The bladder wall is not only thrown into rugæ, but two diverticula are developing on the posterior wall. There are 360 c.c. of residual urine.

Two other patients under my care at present have prostatic hypertrophy. One, a man, aged 34, has no residual urine, but he has a contracted bladder and urinates frequently. The other, a man, aged 37, has 450 c.c. of residual urine and an infected bladder, with the usual rugæ accompanying hypertrophy. Another patient had complete retention for four years previous to operation at the age of 41, and yet another, aged 59, had had complete retention for fourteen years.

Most of these patients gave a definite history of acute infection in young manhood, and all gave a definite history of chronic prostatitis. Of course, acute specific infection is not a pre-requisite to a chronic prostatitis, but it is the most common cause. There was no sclerosis, nor was there a history of injury to any nerve trunk.

These cases show that hypertrophy is not confined to the aged or atheromatous period of life, and also show that the condition is frequently found in patients in the prime of life. It must be admitted, however, that hypertrophy manifests itself most frequently in the aged, but the explanation for this is twofold: First, each year lengthens the time for infection and continued inflammation with its increasing obstruction; and, second, it occurs at that period of life when lack of tone and senile degeneration make themselves manifest. There is yet no record of any celibate undergoing operation for hypertrophy, a strong point in favor of infection as a cause.

TREATMENT.

If this theory be true, pathologic hypertrophy is a preventable condition in many cases, and the treatment is prophylactic, palliative and operative. The prophylaxis is prophylactic, palliative and operative. Prophylaxis consists in the application of heat, massage, dilatation and various medicinal remedies. The palliative treatment consists of catheterization, intravesical irrigation, vesical sedatives and urinary antiseptics.

The operative treatment is palliative and radical. Palliative measures, such as cystotomy, are only resorted to in extreme cases in which prostatectomy is not advisable. Prostatotomy, or the Bottini operation, is indicated in a limited number of cases—those in which the median lobe is small—and even these patients will do better with perineal section. The removal of the gland is advisable in all cases in which hypertrophy is sufficient to demand it unless the patient is *in extremis*.

I am in favor of the perineal operation. First, because it is in accord with the best laws of surgery in that it affords perfect drainage from the most dependent point; second, the ejaculatory ducts, seminal vesicles, prostatic urethra, bladder wall and the vesical sphincter are all left intact, continuous irrigation of the bladder may be kept up for twenty-four hours without soiling the linen, the patient can control his urine the second day after the operation, and is out of bed the third or fourth day, avoiding all the unpleasantness of having to lie in bed bathed in his own urine for two or three weeks, as is the case in the suprapubic operation. In the suprapubic operation not only are the prostatic urethra and ejaculatory ducts destroyed, but a large area of bladder wall is torn, thus opening up the blood vessels and lymphatics for infection, with very poor drainage. The argument has been advanced that the saving of the ejaculatory ducts is of little consequence, since the operation is seldom necessary except in men whose sexuality is unimportant on account of their age. In my opinion, however, the preservation of these ducts is of the utmost importance, and especially would this be true in the boy of 8, the young married man of 25, 34, 41, or even the man of 60 or 65. If for no other reason than to save these ducts, the perineal is the operation to be preferred. A number of cases could be cited in which the sexual powers were not interfered with, but, on the contrary, have been completely restored to normal.

BLOOD DEPLETION TO 296,000 IN PERNICIOUS ANEMIA WITH RECOVERY.

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Reports of recovery in pernicious anemia, when the blood depletion reaches 300,000 or lower, are rare. In Quincke's classical case it reached 143,000 and the patient was said to have recovered, at least for a time. Very few cases, however, are reported in the literature approaching this figurative count. The word "recovery" is used tentatively in the case herewith reported, in the sense that two years have elapsed since this patient first came under observation with a return, as we understand generally the meaning of the term, to perfect health and ability to perform arduous manual labor for the past year. During this time the blood counts and hemoglobin percentages have reached normal limits.

As the blood counts approached the normal, normoblasts and megaloblasts could not be found in the smears (entirely absent for the past year), and the marked poikilocytosis present in the early specimens disappeared. Two years is, perhaps, too short an interval to speak of recovery in a disease so characterized by a tendency to relapse as primary progressive anemia, and still, to all intents and purposes, the patient is in perfect health.

J. B. T., male, aged 27, farmer, was admitted to St. Vincent's Hospital as a patient of Dr. W. W. Coldham, with whom it was my privilege to see him, Jan. 4, 1906.

History.—The patient had never been out of the state, and had never been ill until March, 1905. Purpuric history, scurvy, lead intoxication and lues were excluded. Hemorrhoids not present. In March, 1905, the illness began with weakness, palpitation and dyspnea, loss of appetite and extreme pallor. The ankles and subpalpebral folds became swollen later. He partially recovered during the summer following (1905) and was able to resume his work for a short period.

The illness which brought him to the hospital began about one month before admission (early in December, 1905) with malaise, constantly increasing weakness, constant vomiting after taking food, and dyspnea. There had never been a history of urinary retention or loss of weight.

Examination.—When admitted, the patient was in a drowsy, semi-comatose condition, and unable to answer questions intelligently. The pallor was marked, and of a decided yellow tint. There had been no loss of weight. The panniculus was well preserved. More or less general edema was present, involving, especially, the extremities and face. The eyes were nearly closed. The liver was slightly enlarged, and the spleen was just palpable. Superficial lymph nodes not increased in size. Urine: Examination showed nucleo-albumin present in slight amounts, serum-albumin in considerable quantity, and a slight amount of albumose; otherwise negative. The urine was voided involuntarily during the first few days.

Blood: Jan. 4, 1906. Hemoglobin, minus 10 per cent. (Dare). An average of three counts from ear and finger give 296,000 red cells per c.mm. Poikilocytosis was marked (shadow forms, microcytes, macrocytes, and lance shaped forms were abundant). Few normoblasts and megaloblasts were present. White cells, 4,800; normal differential counts.

Treatment.—The treatment in the beginning consisted of sodium arsenite, gr. 1-10, hypodermically, every four hours, because of the patient's gastric disturbance and his inability to swallow during the first few days in the hospital. Strychnia and brandy were administered hypodermically for the impending collapse, and 500 c.c. normal saline solution were injected subcutaneously, every four hours, for two weeks. Nourishment per rectum. The sodium arsenite was continued during his stay.

Subsequent Blood Examinations.—Jan. 7, 1906. Hemoglobin, 10 per cent. (Dare); red blood cells, 582,400; color index, 0.8; marked poikilocytosis, with abundance of normoblasts; fewer megaloblasts; few myelocytes. The increase in the number of normoblasts and degenerate forms of leucocytes after three days of arsenic medication was very suggestive.

January 10. Hemoglobin, 15 per cent. (Dare); red blood cells, 972,000; color index, 0.7; marked poikilocytosis, but fewer normoblasts and degenerated leucocytes than before.

January 19. Hemoglobin, 20 per cent. (Dare); red blood cells, 1,286,300; color index, 0.7; leucocytes, 3,860; normal differential counts; poikilocytosis still marked; normoblasts diminishing; no megaloblasts found.

The improvement was rapid from this time on and the patient left the hospital five weeks after admission, Feb. 8, 1906.

Subsequent History.—The patient was able to resume work as a farmer in May, 1906. On Nov. 28, 1906, the hemoglobin was 81 per cent (Dare); red blood cells, 2,768,000; color index, 1.4; one normoblast was found in one slide, and a few atypically shaped red cells, but no microcytes or macrocytes.

The blood examination made on Oct. 19, 1907 (one year later) showed hemoglobin, 92 per cent. (Dare); red blood cells, 3,792,000; color index, 1.2; leucocytes, 3,733; differential counts within normal limits. No normoblasts were found; no poikilocytosis.

On Dec. 28, 1907 (two years from beginning of attack), the hemoglobin was 94 per cent. (Dare); red blood cells, 4,438,000; color index, 1.0; leucocytes, 4,320; normoblasts and megaloblasts absent. No poikilocytosis.

Subsequent Treatment.—The first three months after returning home he took small doses of Fowler's solution and dilute hydrochloric acid. Medicine has not been necessary for a year and a half. December, 1907, he weighed 181 pounds.

This case emphasizes the following points:

1. The immediate prognosis in certain cases of pernicious anemia, with blood depletion below 400,000, although serious, is not hopeless.

2. The prognosis depends on the degree of red cell regeneration in the bone marrow, the age of the individual and the potency of the hemolytic poison being important factors.

3. Arsenic exerts a rapid specific influence on the degree of red cell regeneration in certain cases of per-