

THE TREATMENT OF HEAD INJURIES

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IN presenting this paper, which has to do with the successful handling of patients who have sustained injuries to their heads, I would like to say, that I believe most patients are satisfactorily treated, a few are overtreated, and a small percentage suffer or even die as a result of faulty diagnosis, faulty judgment, and lack of treatment. The tendency was and is now, although somewhat less so, to overtreat patients who have received such an injury.

At the outset I would like also to emphasize that injury to the brain, and to its coverings, the meninges, the large intracranial sinuses, and to the middle meningeal arteries, is of prime importance, whereas injury to the scalp and to the skull is in itself of minor consequence. So far as I know no one has ever died as a direct result of simple laceration of the scalp or of simple fracture of the skull; it is only when these injuries are complicated by injury to the brain or to the intracranial vascular channels that serious consequences may result. In order to simplify the subject I have divided cases of head injury into two main groups, minor and major injuries:

Minor Injuries

Under this heading should be included all cases in which a blow on the head has been sustained, in which there has been no loss of consciousness nor evidence of involvement of the central nervous system, and in which roentgenologic examination of the skull for fracture has given negative results. Rarely will a patient with a scalp laceration fall into the second group, which consists of major injuries, because of severe loss of blood due to laceration involving one of the principal arteries of the scalp.

Patients with minor injuries to the head can be satisfactorily treated in the office. If there is laceration of the scalp, the edges of the wound should be shaved and the wound thoroughly

cleansed with tincture of green soap and with hot water. Careful exploration should then be carried out for foreign bodies, especially if laceration is the result of flying glass as from the windshield or window of an automobile. The wound should be swabbed with an antiseptic; I prefer tincture of merthiolate, 1:1,000, or tincture of iodine. The edges of the wound should then be approximated loosely with interrupted sutures of silkworm gut, dermal, or of silk. If it is impossible to control bleeding with tying of the suture, each bleeding vessel must be caught and tied or controlled with a suture ligature lest a hematoma develop beneath the edges of the wound. For the first twenty-four hours a firm compression bandage is advisable. The exploration and suture of the wound is best done under local anesthesia, a 1 per cent solution of procaine being used; among children, gas anesthesia is often advisable and, at times, necessary. If general anesthesia is necessary, hospitalization for a few hours following recovery from the anesthetic of course is advised. If the wound is contaminated with dirt or there is suspicion of soiling, a prophylactic dose of tetanus antitoxin should be given.

Major Injuries

This group comprises all cases in which patients have had a lapse of consciousness, are unconscious or in shock, present symptoms or signs of involvement of the central nervous system, have simple, depressed, or compound fractures of the skull, and those whose head injury is one of a group of injuries sustained during an accident, for today more and more people are encountered with multiple injuries as a result of the mechanization of industry and of speedier methods of travel.

All of these patients should be hospitalized and kept under close observation for at least twenty-four hours, during which time the blood pressure, and pulse and respiration rates should be recorded at frequent intervals. The state of con-

*From the Department of Neurologic Surgery, The Mayo Clinic, Rochester, Minnesota. Read before the meeting of the Wabasha County Medical Society, July 5, 1934, Lake City, Minnesota.

sciousness, mental reactions, and movements of the limbs also should carefully be noted.

Of this group of patients with major injuries a small percentage will be in such poor condition that all measures are likely to be without avail, and death will ensue within the first twenty-four hours after admission. One should never lose hope, however, for now and then a patient who looks hopeless will respond to conservative treatment and make a nice recovery. Generally speaking, though, a patient who does not give evidence of immediate response to treatment has suffered irreparable injury to the brain, and all attempts, conservative or radical, are of course incapable of restoring such individuals to health.

Most patients with major injuries to the head are in a state of shock, that is, they are unconscious or only partly conscious, the blood pressure is low or imperceptible, the pulse is rapid and feeble, and the skin is cold and clammy. The pupils may be dilated and react poorly to light. When a patient is seen in this condition the indication, of course, is to combat shock. Shock is best overcome by application of external heat in the form of hot water bottles and blankets, avoidance of drafts, lowering of the head and, if hemorrhage from a large vessel of the scalp is contributing to the lowering of blood pressure, immediate control of such hemorrhage. At times, a pressure bandage is sufficient to control bleeding; however, as blood pressure rises, bleeding is likely to start again, so it is well to catch the bleeding vessel with a forceps or pass a suture ligature around it and tie it in order to prevent further loss of blood. If shock is profound, pituitrin (surgical) given hypodermically or even intravenously may be used with great success. This drug acts quickly, but its effect is fleeting. Ephedrine, on the other hand, acts a little more slowly, but its effect is more prolonged. Warm, black coffee, when given by rectum, also is effective in combating shock. Acacia (6 per cent) may be used intravenously to increase the volume of circulating fluids, but if there has been much loss of blood, a transfusion of citrated blood is the best treatment.

Time should not be taken to care for and to suture the wound thoroughly until shock has been controlled.

Pain may be a contributing factor in the production of shock, especially if there is injury to some other part of the body, such as, for in-

stance, rupture of a kidney or fracture of one of the long bones. Sedatives, preferably codeine hypodermically, should then be administered. Morphine should be avoided in cases of injury to the head because of its depressing effect on the nervous system, especially on the respiratory center; morphine also masks pupillary changes. When morphine has been given, and the patient becomes very quiet and the respirations slow, it is often impossible to tell whether the change in the patient's condition is the result of morphine or whether it indicates the onset of increased intracranial pressure from cerebral edema or from intracranial hemorrhage.

While shock, if present, is being combated, the head should carefully be inspected and palpated for evidence of lacerations of the scalp and for fractures of the skull. The ears, nose, and mouth should be examined for evidence of bleeding that is so often seen with fractures of the base of the skull. Oftentimes one has to rely entirely on the finding of blood in the ear for the diagnosis of a basal fracture, as these fractures are difficult or impossible to demonstrate by ordinary methods of roentgenography.

Inspection and palpation also may reveal a hematoma of the scalp, without laceration, beneath which there may be a linear fracture. Depressed fractures of the vertex are easily diagnosed by palpation alone. As one moves the fingers across the smooth, curved head one may encounter a sharp, firm margin beyond which there is a distinct depression. The depressed bone may be fixed or movable, and rarely is one able to detect bony crepitus as in fractures of other bones of the body.

No one questions the advisability of roentgenologic examination of the head in cases of cranial trauma, but there is a good deal of argument on the part of some as to the time when the patient should be subjected to this examination. At the clinic it is felt that this examination should be made immediately, and all patients with head injuries are sent directly for roentgenologic examination on admission to the hospital. There are many reasons for this practice. The first and most important reason, from both the patient's and the examining physician's standpoint, is that it enables one to tell within a few minutes whether or not there is a depressed fracture; when this is known, future treatment can be outlined intelligently and without undue suspense as

to whether or not the patient has an injury which demands immediate, active, surgical intervention; also, it can be determined whether or not there is a linear fracture extending across one of the middle meningeal grooves. The importance of this latter information will be referred to later. Another very important factor to be considered is the medicolegal aspects of the case. Today, with so many people carrying insurance to protect themselves as well as their automobiles, and the more or less common practice of instituting suit against employers, insurance companies, and others involved in a given accident, it is imperative that the physician protect his patient's interests and his own reputation by having roentgenograms made which can be used in event of litigation. Obviously the best time to have these plates made is at the time the patient is admitted to the hospital. Some patients live only a short time after severe cranial injuries and it might be difficult to obtain roentgenograms.

While the roentgenologic examination is being conducted, the patient may be treated for shock, lacerations may be dressed and even sutured, and a search for other injuries carried out. A superficial neurologic examination also may be performed, in the course of which the size and reaction of the pupils, extra-ocular and facial movements, the power of the extremities, and the presence or absence of pathologic reflexes may be noted.

In the absence of a depressed fracture and of signs indicative of localized injury to the brain, injuries to the scalp should be cared for and the patient put to bed under very close observation. If the patient is conscious and his general condition is satisfactory, ice caps to the head and analgesics, such as acetylsalicylic acid or codeine, in small doses, for headache, may suffice. Fluids should be limited to an intake of 1,500 c.c. for the first twenty-four hours and they should be in the form of fruit juices and sweetened drinks. This is to limit reactionary edema and swelling in the brain.

If the patient is not doing well, and irrespective of whether roentgenologic examination has demonstrated a fracture of the skull, a diagnostic and therapeutic lumbar puncture may cautiously be performed. Lumbar puncture should not be done in the presence of compound fracture of the vault or of the base because of the danger of the introduction of bacteria when the current

of cerebrospinal fluid is started down the spinal canal after fluid has been escaping through a rent in the meninges and skull. Patients with compound fractures decompress themselves or lower their own intracranial pressure, and lumbar puncture is, therefore, unnecessary as a diagnostic or therapeutic measure.

Some very excellent neurosurgeons advise against the use of lumbar puncture in cases of cranial injury, but I believe it is one of the greatest aids in selected cases and, when cautiously used, free of danger. I am confident that many lives have been saved and that many patients have been spared major complications as a result of wisely conducted spinal drainage. Spinal drainage and intravenous injection of hypertonic (20 to 50 per cent) glucose solutions are our mainstays in the control of increased intracranial pressure resulting from injury to the brain, and these measures have lowered the incidence of the necessity for operative intervention and the mortality rate in cases of cranial injury. Today it is rarely necessary to perform subtemporal decompression in cases of pure cerebral edema resulting from trauma.

Operative intervention, therefore, is indicated only in cases of depressed fracture and in cases in which signs of localized pressure or irritation are exhibited as determined by neurologic examination. The time interval which elapses between cranial injury and the appearance of symptoms and signs of compression of the brain is very important in the differential diagnosis of the cause of compression. Depressed fractures produce symptoms immediately, whereas hemorrhage requires a few hours in which to produce a clot large enough to give signs of a space-occupying lesion. If symptoms do not appear for a day or two, the most likely cause is edema of the brain from contusion caused by jolting of the brain in the rigid cranium.

In cases of depressed fracture, the fragment or fragments should be elevated and replaced, if the fracture is not compound; if the fracture is compound, it is probably better to remove the fragments because of the danger of infection and of development of an osteomyelitis that would necessitate later sequestrectomy.

In extradural hemorrhage, originating from a tear in one of the middle meningeal arteries, there usually is a definite triad of developments. The patient is momentarily unconscious from a

blow on the head; this is followed by a lucid interval which is supplanted several hours later by a state of unconsciousness and stupor. This history, together with a roentgenogram revealing a fracture extending across one of the middle meningeal grooves, enables one to make a diagnosis of extradural hematoma of middle meningeal origin. It has been taught that, in subarachnoid hemorrhage or bleeding from cortical vessels, the cerebrospinal fluid is bloody, and that, in extradural or middle meningeal hemorrhage, the fluid is clear. That is true as far as it goes; one may, however, find bloody spinal fluid from tearing of a middle meningeal artery and, of course, the two conditions may co-exist. Recently, at the operating table, I was able to demonstrate the passage of blood from a torn, middle meningeal artery into the subarachnoid space and the passage of clear fluid from the subarachnoid space into the trephine opening. This patient's spinal fluid had been bloody on lumbar puncture. He made an excellent recovery following ligation of the torn, middle meningeal artery and removal of a large extradural hematoma.

If, in the absence of evidence of a break in the continuity of the skull, the patient has convulsions involving one extremity or one side of the body, or if he has definite weakness of a member, the opposite side of the brain should be explored for a progressively enlarging, intradural hemorrhage. Preliminary lumbar puncture will reveal bloody fluid under increased pressure.

In cases in which there is leakage of cerebrospinal fluid or in which a fistula develops as a result of a basal fracture extending into the paranasal sinuses, the pharynx or ear, treatment should be conservative. Active intervention in the way of operative procedure is likely to result in meningitis, with a prolonged and complicated convalescence, or even in death. Spinal puncture should not be done for the reasons mentioned before. Instead, the patient should be propped up in bed and methenamine (urotropine) administered so long as the fistula remains patent. The position is important because it allows the brain and its surrounding membranes to settle down against the line of fracture and seal it off, purely as the result of gravity. Methenamine acts as a meningitic antiseptic. When there is leakage of cerebrospinal fluid as a result of basal fracture, the patient should be warned not to blow his nose

because of the danger of ascending infection and of meningitis. In cases in which cerebrospinal fluid leaks through the ear, special attention should be paid to the ear or ears involved. The ear should not be irrigated or tightly plugged; instead, a small pledget of sterile cotton, wrung out of a saturated solution of boric acid, should be left in the external auditory meatus. This should be changed as often as necessary to insure complete absorption of all fluid discharged through the rent in the meninges.

Patients who have sustained severe cranial injury should be kept under observation for some time after they apparently have completely recovered, and they should be warned concerning the possibility of development of post-traumatic sequelæ. Such sequelæ are prone to develop in compensation cases, and it is at times a most difficult problem to decide whether a given patient has real trouble or whether in his desire for a satisfactory settlement of his claim he consciously or subconsciously magnifies his symptoms. However, as soon as the patient is able, graduated exercises should be started, his claim settled, and he should be urged to return to work.

The most common sequelæ to injuries of the head are headache and dizziness, both of which are subjective phenomena that cannot accurately be estimated. Headache in many cases is undoubtedly real and is due to faulty absorption of cerebrospinal fluid, which results in chronic hydrocephalus and in increased intracranial pressure. This condition can be ameliorated by limitation of fluid intake and by careful attention to the bowels, using laxatives, if necessary, to secure daily movements. Occasionally, repeated spinal drainages are necessary until the absorptive mechanism catches up with the secreting mechanism. Dizziness at times is quite troublesome to the patient and a major problem to the physician; it may result from a fracture that has involved the ear and semicircular canals, or it may be a sequel to petechial hemorrhages in the cerebellum. Reassurance, regulation of habits, and a mild sedative, such as one of the many preparations of bromides or of phenobarbital (luminal), often help. Occasionally dizziness is so disabling as to warrant, in selected cases, intracranial section of the eighth (auditory) nerve.

Epilepsy is an unfortunate complication that may arise in any case and one whose occurrence is impossible to forecast. If this complication

does appear, and seizures are mild and infrequent, they may be controlled by regulation of the patient's habits and by administration of a saline cathartic when a seizure is anticipated. Some patients require bromides or phenobarbital, either alone or alternately, for indefinite periods. Operation in post-traumatic epilepsy is not indicated unless a foreign body is present or the seizures are Jacksonian in type; even then results are not always brilliant.

Not infrequently patients are seen at the clinic who give a history of cranial trauma, which at the time of the accident was considered quite mild, who have never recovered completely, even though several weeks or even a few months have

passed. In such cases the condition may be due to cerebral compression from chronic, subdural hematoma or from a collection of blood between the dura mater and arachnoid. If neurologic investigation reveals signs of increased intracranial pressure, exploration should be carried out. Evacuation of these accumulations of blood gives very good results.

In conclusion, I wish to state that each case of head injury is in itself an individual problem and should be so managed. When these patients are studied carefully, closely observed, and treated according to the indications in the particular case, the results are very gratifying. Treatment should be as conservative as possible.

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ACUTE MYOCARDIAL INFARCTION, WITH RUPTURE OF THE INTERVENTRICULAR SEPTUM COMPLICATED BY HYPERGLYCEMIA WITHOUT GLYCOSURIA: REPORT OF A CASE*

E. J. Kepler, M. D., J. M. Berkman, M. D., Division of Medicine and A. R. Barnes, M. D., Section on Cardiology:

Dr. Kepler and Dr. Berkman: The patient, a man aged sixty years, was admitted to the Clinic January 2, 1935. Three years previously, following a fracture of the sternum, diabetes mellitus had been discovered. The fracture had healed, and by controlling the diabetes with diet and 30 units of insulin daily the patient's health had remained good. One month before admission, however, he had begun to neglect his diet, he had used alcohol rather freely, and he had discontinued the use of insulin.

The patient had remained in fair health until December 29, 1934, when he had been forced to leave his work on a road scraper because of retrosternal pain. This pain had been projected to the wrists, and it is of some diagnostic significance that when he had reached home he had asked to have his wrists rubbed. Simultaneously with the onset of pain he had become cyanotic and very short of breath. Dr. Nering, who was called to see him at his home, found him to be very ill, and because of

*Presented at the meeting of the General Staff, March 6, 1935.

the dyspnea, pain, and the presence of a large amount of sugar in the urine quite logically felt that the patient's symptoms were due either to the onset of diabetic acidosis, with impending coma, or to coronary heart disease. Obviously the chief therapeutic indication at that time was the use of sufficient insulin to render the urine sugar free. On the following day, even though glycosuria was controlled, the patient's condition had become worse. There had been a sharp drop in the systolic blood pressure to 78 mm. of mercury and in the diastolic to 72 mm. A friction rub could be heard over the precordium. Cyanosis and dyspnea had increased. Because of the difficulties of treatment and some uncertainty as to the diagnosis, Dr. Nering referred him to the Clinic.

On admission to the hospital it was obvious that the patient was critically ill. His temperature was 96°F. and his pulse rate 150 beats a minute. Breathing was labored and he was intensely cyanotic. He could be aroused when questioned, but, when left alone, he lapsed into semistupor. He did not complain of pain, but when asked if he felt pain, he pointed to his sternum.

On examination it was found that the sclerae were slightly jaundiced. Numerous râles were present in the left side of the thorax posteriorly. The abdomen was slightly rigid. A thrill could be felt at the left of the sternum. There was a marked systolic murmur of unusual quality over the entire precordium; its point of maximal intensity was situated in the fourth and fifth left intercostal spaces. The blood pressure was so low that a reading could not be obtained.

Our preliminary opinion was that the patient had had coronary occlusion. This diagnosis was favored because of the presence of shock, the intense cyanosis, and the fact that the dyspnea was not of the type seen in air hunger. The patient was immediately given morphine and was placed in an oxygen tent.

Laboratory studies were then made, but instead of clarifying the diagnosis they made it more obscure. Urinalysis disclosed nothing of consequence except a trace of albumin. The leukocyte count was 12,600 per cubic millimeter of blood. Roentgenologic examination of the thorax, interpreted from bedside roentgenograms gave evidence of bilateral bronchopneumonia, which was more marked on the right side, and moderate enlargement of the heart. Examination of the chemical constituents of the blood showed the values per 100 c.c. to be as follows: sugar, 572 mg.; urea, 236 mg.; creatinine, 4.8 mg.; and serum bilirubin, 3.4 mg. The carbon dioxide combining power of the blood was 28.7 volumes per cent. Electrocardiographic examination gave a rate of 108, sinus tachycardia, left ventricular preponderance, an inverted T wave in derivation III, and a notched QRS in derivation III. A second electrocardiogram on the following day showed a rate of 103, sinus tachycardia, left ventricular preponderance, an inverted P wave in derivation III, an inverted T wave in derivation III, a notched QRS in derivations I and III, and a slurred QRS in derivation II (The QRS interval was 0.14 seconds). In spite of the indeterminate character of the electrocardiograms, the preliminary diagnosis of coronary thrombosis was not changed.

Because of the acidosis, it was felt advisable to attempt to lower the blood sugar even though there was no sugar in the urine. Accordingly, the patient was given 50 units of insulin at once and 10 units every hour until the blood sugar was reduced to 240 mg. By this time it was evident that control of the hyperglycemia was not improving the clinical condition of the patient and no further insulin was given. He continued to fail and died about thirty-six hours after admission.

The salient features in this case, then, were as follows: An elderly patient, who had had diabetes mellitus for three years, during the last month of which the condition had been uncontrolled, suddenly suffered severe pain. This pain was situated behind the sternum and was projected to the wrists. It had however decreased in severity. He was found to have severe glycosuria, which was controlled by the use of insulin. Subsequently he went into a condition simulating shock, which was characterized by a rapid fall in blood pressure, cyanosis, and dyspnea. On admission to the hospital, laboratory studies disclosed marked hyperglycemia without glycosuria, moderate acidosis, and the chemical changes in the blood usually indicative of marked renal insufficiency.

At necropsy nothing of consequence was found except pathologic changes in the heart. The heart weighed 516 gm. and was light reddish-brown. On the surface of the left ventricle and base of the right ventricle was a fibrous exudate which was present on the opposing surface of the parietal pericardium. The consistence of the left ventricle was decreased, grade 3. The color of the cut surface of the left ventricle was also reddish-brown. The coronary arteries were sclerosed, grade 3, and the anterior descending branch was occluded by a thrombus. There was acute infarction of the apex, of the anterior portion of the left ventricle, and of the adjacent interventricular septum, the infarction extending two-thirds of the distance from the apex to the base, and there was aneurysmal dilatation of the apex of the left ventricle and of the interventricular septum. At a point in the interventricular septum about midway between the base and apex, there was an irregular rent about 1 by 0.5 cm. Mural thrombi were present in the left ventricle, on its posterior wall, at the apex, and on the interventricular septum. The foramen ovale was closed. The mitral valve was slightly thickened, but the other valves of the heart were normal. There was very marked arteriosclerosis of the aorta.

COMMENT

This case is of considerable interest for a number of reasons: 1. In spite of the presence of a huge myocardial infarct the patient complained of little pain. The absence of pain may possibly be explained by his temperament. When he was well, he was stolid and phlegmatic; he was fond of boxing and of other rough sports and in general was hyposensitive to pain. 2. The chemical changes in the blood were unusual. It is exceedingly uncommon to find severe hyperglycemia without the presence of sugar in the urine. This chemical anomaly occurs occasionally in renal insufficiency. In this case, however, in spite of the high concentration of the blood urea the clinical picture was not that of renal insufficiency. There was no anemia. The urine was of normal specific gravity and con-

tained no albumin, casts, or erythrocytes. We therefore felt that the high concentrations of sugar and urea in the blood were due to a common cause, which probably would be found in the heart. This interpretation was confirmed at necropsy, at which time it was found that the kidneys were normal both grossly and microscopically. It is likely that elevation of both sugar and urea in the blood was the result of myocardial infarction and the exceedingly low blood pressure incidental to it. It is well known that the excretory function of the kidneys depends on the maintenance of a blood pressure which approaches reasonably close to normal limits. The stupor likewise may have been the result of cerebral anemia secondary to the failing circulation.

Dr. Barnes: The consultants who first saw the patient felt, as they have stated, that he had had an attack of acute coronary thrombosis. When I was called I could not be certain of that diagnosis. The patient was in coma and could not give me any history of his attack. His relatives could help very little in supplying details. The electrocardiogram was not suggestive of acute coronary occlusion, although that need not influence one against the diagnosis of coronary thrombosis in the presence of severe diabetes. In the latter condition, especially if complicated by acidosis, the electrocardiogram may undergo bizarre changes which obscure the features characteristic of acute infarction. By the time I saw the patient the râles in the chest were so loud that I could not hear a friction rub. In retrospect, however, the history, the severe heart failure with a marked fall in blood pressure, and the observation of a friction rub, constitute almost irrefutable evidence of acute myocardial infarction.

The most arresting finding on examination of the heart was the presence of a loud systolic murmur, which was heard over a wide precordial area but which was greatest in the fourth and fifth intercostal spaces. A marked systolic thrill was felt to the left of the sternum over the same area. It was perfectly obvious that this constituted the classic signs of congenital septal defect as described by Roger,¹ and I made such a diagnosis. Not being certain of the diagnosis of acute myocardial infarction and never having seen a similar case, it did not occur to me to postulate that this perforation was due to rupture of the septum. Actually, the diagnosis should be easy to make when known acute coronary occlusion is accompanied by the classic signs of septal perforation, as observed in this case.

Sager² reviewed the literature on coronary thrombosis complicated by perforation of the interventricular septum. He found reported seventeen cases and added one of his own in which the diagnosis was substantiated at necropsy. Sager stated that the smaller the perforation the more intense the murmur. If the rent in the septum is large enough, he said, a diastolic murmur may develop. In all but one of the reported cases rupture of the septum had resulted from occlusion of the anterior descending branch of the left coronary artery. The rent was usually situated in the lower half

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1. Roger, H.: Recherches cliniques sur la communication congénitale des deux coeurs, par inoclusion du septum interventriculaire. *Bull. Acad. de méd.*, Par. 2 s., 8: 1074; 1189, 1879.
 2. Sager, R. V.: Coronary thrombosis: perforation of the infarcted interventricular septum, *Arch. Int. Med.* 53: 140-152 (Jan.) 1934.

of the septum. All cases with this auscultatory evidence of septal rupture have resulted fatally, and the longest duration of life in any case was ten days.

Sager reminded us that rupture of the papillary muscle in the left ventricle must be distinguished from rupture of the septum. In the former condition he stated that "systolic thrills and murmurs are even more bizarre and more pronounced and a diastolic murmur is more common. The physical signs are either maximal at the apex or equally marked over the entire precordium." Few patients survive congenital patency of the septum to an age at which acute myocardial infarction occurs. Moreover, in the former condition the history of acute coronary occlusion will be lacking. Rupture of the septum may occur in bacterial endocarditis, but in such cases the embolic phenomena, a positive blood culture, and the relative youth of the patient will suffice to make the differential diagnosis.

TRANSCRANIAL REMOVAL OF AN INTRA-ORBITAL MENINGIOMA

J. G. Love, M. D., Section on Neurologic Surgery: Last year, Adson and Benedict¹ reported before the Section on Ophthalmology of the American Medical Association a case of hemangio-endothelioma of the orbit in which the growth was removed by the transcranial approach. Since that time two other tumors of the orbit encountered at the Clinic have been uncovered and removed in the course of transfrontal exploratory craniotomy. Dr. Craig will report one of these cases, and I am reporting the other and most recent.

The patient, a white woman aged fifty years, came to the Clinic because of prominence and diminution in vision of her right eye. These symptoms have been progressing for nine months. On examination she weighed 175 pounds (79.4 kg.) and was 5 feet 2 inches (157.2 cm.) in height. Her systolic blood pressure was 178 and diastolic 116 mm. of mercury. There was moderate peripheral arteriosclerosis; otherwise, general physical examination gave essentially negative results. Results of the ophthalmologic and roentgenographic examinations, however, were striking and were the only positive findings suggestive of the underlying pathologic condition. The right eye was obviously more prominent than the left, and exophthalmometer readings for the right eye were 23 mm. and for the left eye 16 mm. There was no obvious impairment of rotations, and the peripheral fields were normal. Bilateral astigmatism was present; vision was 6/60 in the right eye and 6/30 in the left, without correction, the error being greater in the right eye than in the left. Examination of the ocular fundi did not reveal any evidence of edema, or pallor of the disks. There was definite hypertensive sclerosis of the retinal vessels. Roentgenograms of the head and orbits disclosed diffuse bony proliferation involving the wing of the right sphenoid and superior wall of the right orbit, suggesting a contiguous meningioma. In addition, there was some benign hyperostosis

1. Adson, A. W. and Benedict, W. L.: Hemangio-endothelioma of the orbit. Removal through transcranial approach. *Arch. Ophthalm.* 12: 484-492 (Oct.) 1934.

of the frontal bone. Neurologic examination was entirely negative for evidence of involvement of the central nervous system. In view of these findings and the known tendency of the meningiomas of the sphenoidal ridge to extend into the orbit and produce unilateral exophthalmos,² a diagnosis of meningioma arising from the right sphenoid ridge and extending into the right orbit was made.

Right transfrontal craniotomy was performed March 14, 1935. The frontal bone was markedly thickened and extremely vascular. The dura mater was very thin and inseparably attached to the hypertrophied por-

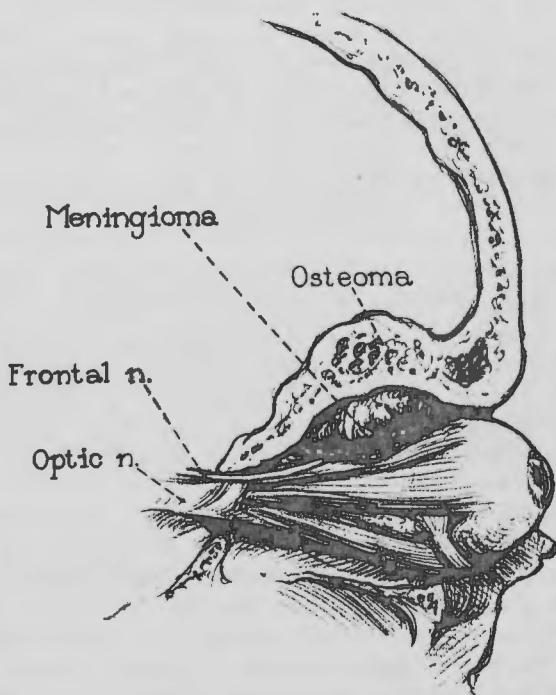


Fig. 1. Relation of the meningioma to orbital contents.

tions of the frontal bone. This necessitated an intradural rather than the usual extradural exploration of the sphenoid ridge and anterior cranial fossa. When the right frontal lobe was elevated, the anterior fossa gave no evidence of the presence of a tumor other than an osteomatoid formation. The sphenoid ridge and right optic foramen were normal, as were also the right optic nerve and the chiasm. In short, there was no intracranial neoplasm. Because unilateral proptosis, with diminution of vision, had been the outstanding symptom and finding, the orbit was then unroofed extradurally by rongeur away the osteoma involving the superior wall of the right orbit, and in so doing a reddish-brown intra-orbital tumor was uncovered. The tumor had completely replaced the orbital fascia, and, when it was elevated, the frontal nerve lying on the levator palpebrae

2. Voris, H. C. and Adson, A. W.: Meningiomas of the sphenoidal ridge with unilateral exophthalmos: report of two cases. *Surg. Clin. N. Am.* 14: 663-672 (June) 1934.

superioris and extra-ocular muscles was exposed without any tissue intervening. The tumor was removed completely, and microscopically proved to be a meningioma. The wound was closed in the usual manner and two Penrose drains were left as a precautionary measure because of the marked vascularity of the bone.

The immediate postoperative condition of the patient was excellent, the drains being removed at the end of twenty-four hours. About thirty-six hours following operation a rapidly increasing weakness of the left side developed, and within an hour, the patient was stuporous and had complete left hemiplegia. By this time she was on the operating table for elevation of the flap. When the wound was opened, a large hematoma was found compressing the right side of the cerebrum, and free bleeding was taking place at the site of spontaneous rupture of a vessel on the surface of the frontal lobe. The vessel was doubly occluded by means of silver clips, and then, after removal of the hematoma, the wound was closed with through-and-through sutures of silkworm gut. Two Penrose drains were left in for twenty-four hours.

Following this complication, the patient made a spectacular and complete recovery. She was dismissed from the hospital on the twelfth day after operation, at which time the wound was completely healed and neurologic examination was negative. Examination of the eyes revealed definite reduction in the exophthalmos, the actual readings being for the right eye 22 mm. and for the left 16 mm. Pupils, reflexes and extra-ocular movements were normal. Vision was 6/20 in each eye, with correction. Examination of the fundi revealed no change from the preoperative condition. Slight edema of the right eyelids was still present.

SUMMARY AND CONCLUSIONS

Roentgenograms which ordinarily are so valuable in the diagnosis and localization of this type of tumor were misleading. They suggested the presence of an intracranial neoplasm, whereas the entire tumor lay within the orbit.

The practical application of the transcranial approach to tumors of the orbit is again demonstrated.

*ANNUAL MEETING, MINNESOTA STATE MEDICAL
ASSOCIATION — MINNEAPOLIS,
JUNE 24, 25 and 26, 1935*

TREATMENT OF SUDDEN ARTERIAL OCCLUSION WITH PAPAVERINE HYDROCHLORIDE: REPORT OF CASE*

E. V. Allen, M. D., Division of Medicine and A. R. MacLean, M. D., Fellow in Medicine, The Mayo Foundation: A minister, a Norwegian aged fifty years, first came to the Clinic in 1932 because of diabetes mellitus of twelve years' duration, and because of occlusion of the coronary arteries which had taken place three months previously. Treatment for the diabetes and heart disease was outlined. The patient was reexamined at the Clinic and admitted to the hospital February 11, 1935. From 1932 to 1935 he had completely neglected the diabetes and, although suffering frequently from attacks of angina pectoris, he had continued to lead an active life until the onset of gangrene, involving the left great toe, in January, 1935.

On examination the distal phalanx of the left big toe was found to be superficially gangrenous. The pulsations of the dorsalis pedis and posterior tibial arteries of the left foot were graded 2.¹ Pulsations were absent in the dorsalis pedis and posterior tibial arteries of the right foot but were normal in the popliteal and femoral arteries on both sides.

The diabetes was controlled in a week by a diet containing 111 gm. of carbohydrate, 65 gm. of protein, and 157 gm. of fat, and by 60 units of insulin, daily. As a result of rest, contrast baths, massage, ingestion of alcohol, and treatment with short wave diathermy, the superficial gangrene affecting the left great toe improved so that, on April 7, only a small area, $\frac{1}{2}$ inch (1.27 cm.) in diameter, remained.

On March 7, at 2 p.m. while the patient was sitting quietly in a chair, he noticed a sudden and severe pain in his right leg. On examination of the leg a few minutes later it was found to be white and cold from the toes to the middle of the thigh. There was complete loss of motor function below the knee and the reflexes were abolished. Anesthesia was present to the level of the knee. Pulsations could not be felt in the arteries of the right leg beyond a level 2 inches (5 cm.) distal to Poupart's ligament. No pulsations could be felt in the arteries of the left leg. Obviously the patient had had sudden and complete occlusion in the lower part of the femoral artery of the right leg and in the iliac artery of the left leg. No symptoms of sudden occlusion of the arteries of the left leg were noted at that time. The patient became agitated: his pulse rate increased to 140 beats per minute, his blood pressure decreased and pulmonary edema developed. The administration of large amounts of morphine and atropine relieved neither the pain in the leg nor the tachycardia and pulmonary edema. One hour after the onset of pain the condition of the right leg

*Presented at the meeting of the General Staff, March 27, 1935.

1. Pulsations are graded from 0 to 4; the latter indicates normal.

had not changed. Papaverine hydrochloride $\frac{1}{4}$ grain (0.016 gm.) was injected intravenously at 3:20 p.m. Two minutes later a delicate blush was apparent in the toes which had previously been pale. Four minutes after injection of the papaverine the entire foot was pink and pain had decreased appreciably in the leg. Improvement of circulation continued, and, forty minutes after injection, the leg had recovered all function and sensation was normal. The pain had vanished and the color of the foot was normal. No change was noted in the arterial pulsations, but aside from this there was no evidence that the arteries had suddenly been occluded.

At 8:00 p.m., six hours after the symptoms of arterial occlusion, the patient complained of pain in the left foot and calf. Dyspnea and signs of pulmonary edema were still present. On examination the foot and lower part of the left leg were pale, cold, and insensitive. The area of cyanotic discoloration on the big toe had increased. In an hour anesthesia and coldness had involved the leg to 3 inches (7.5 cm.) above the knee.

Suitable treatment for the cardiac condition was instituted, and papaverine hydrochloride, $\frac{1}{4}$ grain (0.016 gm.), was again injected intravenously, but symptoms and findings referable to the left leg did not change. However, the temperature of the right leg, in which there had been sudden arterial occlusion a few hours previously, increased 2° C. in ten minutes.

Morphine in large doses had little effect on the terrific pain. Intermittent suction and pressure (Pavaex treatment) was applied to the left leg at 10:30 p.m., and the pain was relieved completely in fifteen minutes. Papaverine hydrochloride was given four more times in the next twenty-four hours but without benefit. In the following four and a half days a total of seventy hours of treatment was given with the intermittent suction and pressure apparatus. When this treatment was first discontinued pain recurred in an hour. The period of relief gradually decreased until, shortly before operation on March 11, it was necessary to carry on treatment continuously in order to relieve the pain. The distal parts of the extremity slowly became gangrenous. On March 11, the condition of the heart was greatly improved and amputation of the left leg was performed through the upper part of the thigh. Spinal anesthesia was used. There was very little bleeding during the operation and the larger arteries and veins were completely thrombosed.

Following operation the patient's condition was fairly good except for an increase in the pulse rate to 140 beats per minute. There was no recurrence of pulmonary edema. The condition of the right leg remained excellent, the color, temperature, sensation, and movement being consistently satisfactory. Forty-eight hours after operation the body temperature and pulse rate increased. The patient became anxious, then delirious, and finally died on March 15, four days after operation. At necropsy the left iliac artery was found to be occluded completely. The right iliac artery and upper portion of the femoral artery were patent although a moderate degree of arteriosclerosis was noted.

COMMENT

The return of circulation to the right leg as a result of treatment with papaverine² following sudden occlusion of the right femoral artery was the most striking and dramatic that we have ever observed. Circulation was not reestablished to the left leg for reasons which we will consider later. What is the rationale of papaverine therapy and the explanation of the results following its use? This question can be answered only if there is clear understanding of the sequence of events following sudden arterial occlusion.

For some time we have felt that the profound ischemia that follows sudden arterial occlusion was far too marked to be explained by simple occlusion of an artery. It seemed unreasonable to us that sudden occlusion of a popliteal artery, for example, should cause gangrene of the foot when we frequently see individuals with chronic occlusive disease of the popliteal arteries without any great degree of impairment of the arterial circulation. Always when we have seen cases of acute arterial occlusion in which severe pain has begun suddenly, we have been dissatisfied with our explanation of the cause of the pain. Recently we have believed that it was caused in a large degree by widespread arterial spasm. This belief is supported by a number of observations of other investigators. Seifert³ and others have observed arterial spasm in the involved artery during operation for removal of an arterial embolus. After observation of patients with arterial embolism, Seifert³ concluded that pain occurring at the onset of arterial embolism was due to arterial spasm. He noted also that simple arterial ligation, which was frequently necessary in the treatment of war wounds, did not ordinarily provoke the type of pain which is associated with arterial embolism.

In a study of experimentally produced embolism, Gosset, Bertrand, and Patel⁴ observed that an embolus was fixed at the point of lodgment by arterial spasm. They observed likewise that the lowered temperature of an extremity in which a main artery had been occluded by an embolus was replaced gradually by a normal temperature a few hours afterward. This is indirect evidence that the original diminution in warmth was caused by arterial spasm that relaxed in the course of a few hours to allow reestablishment of circulation. Excellent experiments have been reported by Mulvihill and Harvey⁵ who ligated the external iliac arteries of dogs. The temperature of the extremities was reduced to that of the room in from two to six hours. This lowered temperature persisted for several hours, when there was a gradual or rapid increase in temperature so that, in an average period of about thirteen hours after ligation of the artery, the corresponding extremity was again about as warm as before opera-

2. We do not know the optimal amount of papaverine hydrochloride for intravenous injection. Recently we have observed vasodilation and no harmful effects following injection of $\frac{1}{2}$ grain (0.032 gm.) into the veins of two patients and 3 grains (0.2 gm.) subcutaneously into one patient.
3. Seifert, E.: Die Deutung des Schmerzes bei der arteriellen Embolie. Deutsch. Ztschr. f. Chir. 232: 187-198 (May) 1931.
4. Gosset, A., Bertrand, Ivan and Patel, Jean: Sur la physio-pathologie des embolies artérielles des membres (recherches expérimentales). Ann. d'anat. pathol. 9: 841-862 (Nov.) 1932.
5. Mulvihill, D. A. and Harvey, S. C.: Studies on collateral circulation. I. Thermic changes after arterial ligation and ganglionectomy Jour. Clin. Investigation. 10: 423-429 (Aug.) 1931.

tion. If sympathectomy was performed when the temperature of the foot was decreased as a result of arterial ligation, there was an immediate increase to the approximate level of warmth before the iliac artery was ligated. If sympathectomy was performed before arterial ligation, no change in the temperature of the extremity resulted from ligation. Herrmann and Reid⁶ reported that, in ten cases of sudden arterial occlusion, intermittent negative and positive pressure, effected by the apparatus described by them, reestablished the circulation. We feel that these excellent results are brought about by inducing collateral arteries to function at a higher level.

These observations indicate rather conclusively that widespread arterial spasm, or failure of collateral arteries to dilate, substantially effects the diminished arterial supply which follows sudden arterial occlusion. It follows also that the best therapeutic procedure for sudden arterial closure is relief of arterial spasm or the inducing of collateral arteries to assume an increased function in the transportation of blood. The procedure of choice is sympathectomy, but this is ordinarily too extensive a procedure when sudden arterial occlusion has occurred.

With these apparent facts in mind we had decided that an excellent therapeutic procedure would be to interrupt sympathetic impulses by administration of a spinal anesthetic agent when the arteries of the lower extremities were suddenly occluded. At this time we read an article by Denk,⁷ who treated ten patients with arterial embolism by administering papaverine, which is an antispasmodic. In six cases the patients recovered completely. Two failures were explained by the long period elapsing between the onset of embolism and the beginning of treatment; in another case there was severe arteriosclerosis, and in the fourth case the failure could not be explained. Denk believed that the results of treatment with papaverine equaled those following embolectomy.

In the case which we are reporting the results following the use of papaverine were truly startling and unequivocally excellent as far as the circulation to the right leg was concerned. Failure to reestablish circulation to the left leg appears to us to be attributable to the high arterial closure and the previous diminution in circulation to a point at which gangrene had occurred. Under the circumstances it appears that adequate collateral circulation was not available.

We do not believe that the administration of papaverine is the final solution in the treatment in all cases of sudden arterial occlusion. However, it is simple, and the results, when they occur, are prompt. We see no harm in injecting papaverine intravenously in all cases of sudden occlusion of the arteries of the extremities. If the method fails, embolectomy, treatment with intermittent suction and pressure, or other measures can be used. It is of interest that in this case intermittent negative and positive pressure likewise failed to produce reestablishment of circulation to the left leg.

6. Herrmann, L. G. and Reid, M. R.: The conservative treatment of arteriosclerotic peripheral vascular diseases. *Ann. Surg.* 100: 750-760 (Oct.) 1934.

7. Denk, Wolfgang: Zur Behandlung der arteriellen Embolie. *München, med. Wchnschr.* 81: 487-489 (March) 1934.

Prompt treatment is important in all cases of sudden arterial occlusion, and accordingly prompt recognition of the condition is essential. Why is this so if the arterial spasm is transient, as shown by the experiments of Mulvihill and Harvey, and Gosset, Bertrand and Patel? We cannot answer definitely, but we believe that ischemia resulting from occlusion and unrelieved spasm injures the intima of arteries and veins, so that when spasm abates, widespread thrombosis occurs.

Finally, we suggest that treatment with papaverine be tried in cases of occlusion of the coronary, cerebral, mesenteric, and pulmonary arteries. Denk has suggested injection of papaverine in pulmonary embolism and deTakats⁸ reports an excellent result in one case.

SUMMARY AND CONCLUSIONS

The intravenous injection of papaverine hydrochloride effected reestablishment of circulation in one extremity, but failed to do so in the opposite extremity, in a case of sudden arterial occlusion.

The good results appear to be based on the antispasmodic property of papaverine, that is, on relief of arterial spasm.

TRANSURETHRAL SURGERY IN 1934

Gershom J. Thompson, M. D., Section on Urology: The number of patients entering the Colonial Hospital with a chief complaint referable to the urinary tract in the last year has increased markedly. Members of the urologic service in addition to being mainly responsible for these cases see in consultation with members of other medical and surgical services many other patients whose chief complaint is not urologic.

Table 1 gives in more or less detailed fashion the various procedures carried out in the cystoscopic rooms. Many of the patients on the urologic service, of course, did not require cystoscopic examination or transurethral operation, hence not all the patients cared for are included in the total of 1,449 in whose cases a total of 2,649 procedures, exclusive of anesthesia, were carried out.

A considerable portion of this work requires general or regional anesthesia, and anesthetic agents were given 1,161 times; this included spinal anesthesia in 739 cases, general anesthesia in 386, sacral or caudal block in seven and anesthesia induced by the intravenous injection of barbiturates in twenty-nine. This last named form of anesthesia, using either evipan or theonembutal, is very satisfactory for short operations and has been used extensively in the first few months of 1935.

The number of patients subjected to transurethral manipulation of ureteral calculi dropped somewhat, and this may be attributed to the fact that not many patients with small sized stones were seen at the Clinic. Included in the twenty-three cases in which stones were removed are some in which the calculus exceeded 1 cm. in its smallest diameter.

The removal of vesical calculi by litholapaxy was accomplished in forty-three cases (table 1). Some of these stones were good sized and in many cases multiple stones existed. In fifteen additional cases stones of

8. deTakats, Gaza: Personal communication to the authors.

small size were removed with a forceps passed through the cystoscope; in these cases it was not necessary to use the lithotrite.

Included in table 1 under "miscellaneous procedures" are two cases in which congenital urethral valves that caused urinary obstruction were excised, and one case, previously reported with Dr. Helmholz,¹ in which ureteral meatotomy for congenital ureteral obstruction, followed by use of the ketogenic diet, resulted in elimination of infection of the urinary tract. Thirty-two patients with prolonged chronic prostatitis who had abscesses of the prostatic duct were subjected to transurethral operation according to methods described elsewhere.²

Table 1
Transurethral surgery

	Operations		
	1932	1933	1934
Overdistention	30	28	40
Implantation of radium	7	8	9
Fulguration:			
Urethral cysts	71	188	30
Tumor of bladder	29	44	55
Dilatation of stricture	48	19	13
Manipulation of ureteral stones	57	44	40
Cases	39	35	30
Successful	28	28	23
Litholapaxy	12	9	43
Prostatic resection	323	513	696
Sphincterotomy	21	16	3
Incision or fulguration of abscess of prostatic duct		44	32
Miscellaneous procedures	10	9	32
Vasectomy			171
Urograms			166
Cystoscopic examinations		1238	1319
Total procedures	1893	2160	2649

Of the total number of patients, 630, or 43 per cent, were operated on for prostatic obstruction. This is an increase of 39.6 per cent over the number of patients submitting to the same operation in the preceding year, and since it constitutes such a large proportion of the work it seems fitting to confine the rest of my remarks to this group of prostatic cases.

Included in these 630 cases, in which a total of 696 transurethral prostatic resections were performed, are twenty-three in which the patients had submitted to the same operation at this Clinic in former years. In five of these cases the original growth was malignant and recurrence could, of course, be expected. In the other eighteen cases, in which recurrence was not expected, it was necessary to remove 8.2 gm. of tissue, on the average, in order again to relieve the symptoms; in seven of these cases the total weight of the tissue removed at both visits exceeded 25 gm. The question arises, should these patients have been subjected to prostatectomy either at their first or second visit? It was answered without much hesitation by the patients themselves, who on their return refused to have anything other than transurethral resection. Considering the increased

1. Helmholz, H. F. and Thompson, G. J.: The rapid therapeutic effect of ketonuria after ureteral meatomy in chronic pyelitis: report of case. *Proc. Staff. Meet. Mayo Clinic.* 9: 209-213 (April 11) 1934.
2. Thompson, G. J. and Cook, E. N.: Chronic prostatitis and prostatic calculus. *Jour. Am. Med. Assn.* 104: 805-807 (Mar. 9) 1935.

risk and increased stay in the hospital required for prostatectomy, we did not feel inclined to urge its performance. In each case of recurrence the stay in the hospital required for prostatic resection was less than a week, and there was no mortality.

The mean weight of tissue removed per case during the year was 11.6 gm.; in seventy-three cases the amount exceeded 25 gm.

The average period of hospitalization was definitely shorter than in former years; 58.1 per cent of patients stayed one week or less, an additional 28.9 per cent left in the second week, and only 13 per cent remained longer. The mean period of hospitalization was eight and one-tenth days. The hospital period for the group as a whole was shortened by the reduction in the number of cases in which preliminary suprapubic cystostomy was performed; in twenty-one cases such an operation was done here and in twelve cases cystostomy had been performed elsewhere. Thus a total of 5.2 per cent of the group, as compared with 12.2 per cent in the two preceding years was subjected to preliminary suprapubic cystostomy.

Table 2
Postoperative maximal temperature

Degrees, F.	Resections	Per cent of total resections
97.	2	0.3
98	58	7.6
99	306	44.0
100	181	26.0
101	75	10.8
102	36	5.1
103	27	3.9
104	13	1.9
105	2	0.3
106	1	0.1
Total	696	

There has been a great deal of discussion among urologists in the last year concerning the incidence of febrile reactions and complications following prostatic resection. Caulk and Patton³ concluded after an extensive study that punch instruments do not cause as much destruction of tissue as those resectoscopes which depend on high frequency electric currents for their cutting action. At the Clinic the Braasch-Bumpus punch is used almost exclusively, and it is interesting to note (table 2) that in 52 per cent of our cases the postoperative temperature did not exceed 99° F.; in an additional 36.8 per cent it did not exceed 101° F. at any time during convalescence. Twenty-six patients, or 4.1 per cent had a chill coincident with the fever. I⁴ have called attention elsewhere to measures instituted for the prevention of complications. We have been fortunate in almost eliminating an extremely painful one; namely, epididymitis; which occurred in only eight of the 630 cases in 1934, an incidence of 1.3 per cent. This has been made possible by strict attention to methods of drainage by urethral catheter rather than by routine vasectomy, for this latter operation was performed in only 24.6 per cent of the cases.

3. Caulk, J. R. and Patton, J. F.: The influence of the type of current upon the postoperative complications in transurethral surgery. *Jour. Urol.* 30: 537-558 (Nov.) 1933.

4. Thompson G. J.: The prevention of complications of transurethral prostatic resection. *Urol. and Cutan. Rev.* 38: 847-851 (Dec.) 1934.

Litholapaxy was done at the same time as prostatic resection in twenty-eight cases and in seven others a vesical tumor was removed trans-urethrally at the same time as prostatic resection.

Other data, of interest chiefly to urologists, will be presented at some future time. I might mention in closing that during the year there were but two deaths, a mortality of three-tenths of one per cent for the series of 630 prostatic cases.

THE INCREASE IN CIRCULATION RATE PRODUCED BY
EXOPHTHALMIC GOITER COMPARED WITH THAT PRO-
DUCED IN A NORMAL SUBJECT BY WORK*

W. M. Boothby, M. D., Section on Clinical Metabolism and E. H. Rynearson, M. D., Division of Medicine: During the last two years we have carried out a series of 160 determinations of the circulation rates of seven women and four men who had exophthalmic goiter. In all experiments on circulation rate there is quite a spread in the determinations made under apparently identical conditions, even with good technic, and in exophthalmic goiter this spread is definitely larger than the same observers obtain for normal subjects. Although these variations do not greatly affect the general picture of the average circulation rate in exophthalmic goiter if a sufficient number of determinations are made, the discrepancy between reported determinations in the same case, nevertheless precludes utilization of the method for diagnostic or for other clinical purposes.

The results of this study may be stated briefly as follows: The circulation rate is increased in cases of exophthalmic goiter. On the average, the greater the intensity of the disease, as measured by oxygen consumption, the greater the increase in circulation rate.

The increase in circulation rate among patients with exophthalmic goiter who are not treated with iodine, according to the investigations of Liljestrand and Stenström, is very much greater than occurs among normal individuals from an increase in oxygen consumption as a result of work.

The relative increase in circulation rate among patients who are treated with iodine is definitely less for a given increase in oxygen consumption than among those who have not received iodine. However, even under these conditions, the increase in circulation rate is on the average, both in our own cases and in those of Fullerton and Harrop, slightly greater than that produced among normal individuals by a degree of work that causes a similar increase in oxygen consumption.

These facts suggest the hypothesis that in exophthalmic goiter, especially when the patient is not treated with iodine, there is present in the system a peculiar circulatory stimulant that increases the circulation rate to a greater degree than occurs in a normal subject from a corresponding increase in oxygen consumption as the result of work. In exophthalmic goiter, either this peculiar circulatory stimulant is decreased in amount or its effectiveness is lessened by efficient medication with iodine.

*Abstract of paper to be published elsewhere.

SARCOMA OF THE LARYNX: REPORT OF TWO CASES*

G. B. New, M. D., Section on Laryngology, Oral and Plastic Surgery: Sarcoma of the larynx is rarely observed. Examination has been made in six cases at the Clinic since 1910 as compared with more than 800 cases of carcinoma of the larynx seen in the same period. In 1933, Figi reported four cases and thoroughly reviewed the literature so that this will not be attempted.

In the last year, 1934, I operated on the two patients with sarcoma of the larynx whose cases I am reporting elsewhere. Both patients recovered from the operation and were dismissed in about two months. As in malignant tumors in the antrum of Highmore and pharynx, sarcomas are not reported as frequently as they were reported a decade or two ago, apparently because the pathologists believe that many of these tumors are epithelial rather than mesodermic in origin.

The treatment of sarcoma of the larynx must depend on the situation of the growth, its method of attachment, the type of growth, and its activity. It may vary from a fibrosarcoma, grade 1, which is a very inactive type, to a fulminating type of sarcoma. It may be pedunculated although a very active growth, or diffuse although a very inactive malignant growth. The sarcomas of the larynx that we have seen here at the Clinic, have been either of low grade or encapsulated so that we have removed the tumors by means of thyrotomy, followed by the use of surgical diathermy and irradiation. I have seen highly malignant laryngeal tumors with diffuse involvement in young adults, that have been reported as squamous cell epitheliomas, grade 4, but have been rapidly growing tumors acting clinically like sarcomas. In these cases, irradiation has been employed rather than any surgical procedure. Under this treatment the tumor has temporarily entirely disappeared but I have never seen a patient who has had such a tumor and who has stayed well. In these cases of more extensive laryngeal tumor in which thyrotomy is to be done, I feel that a two stage operation is advisable. Preliminary tracheotomy is performed, and then, after the patient has convalesced from this, exploration of the larynx is made by means of thyrotomy. More extensive tumors should be removed by laryngectomy. The prognosis in cases of sarcoma of the larynx, because of the large percentage of low grade tumors of mesodermic origin is, in general, better than in cases of epithelioma. In the two cases on which this note is based enough time has not elapsed since the operation to warrant a report as to outcome, although at the time the patients were last heard from there had been no recurrence.

*Abstract of paper presented at the meeting of the General Staff March 13, 1935. To be published in full elsewhere.