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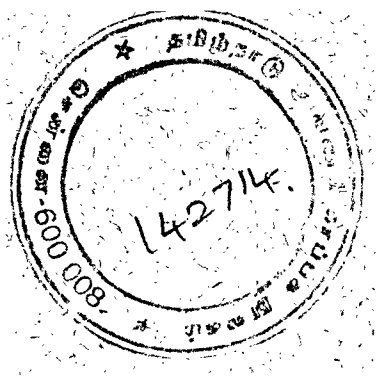
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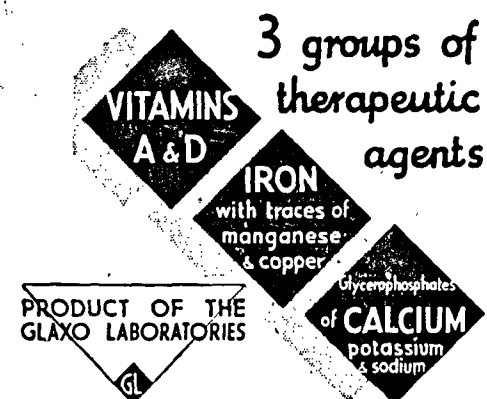
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A MONTHLY JOURNAL OF PRACTICAL MEDICINE & SURGERY

Editor: Dr. P. R. Venkappaya.

VOL. VII

MARCH 1938

No. 3

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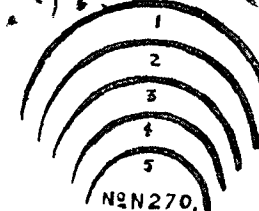
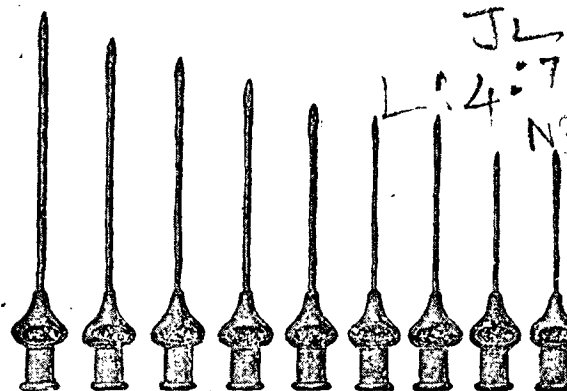


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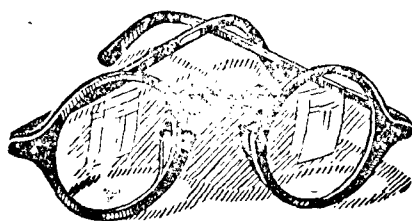
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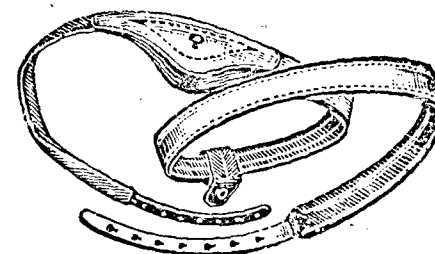
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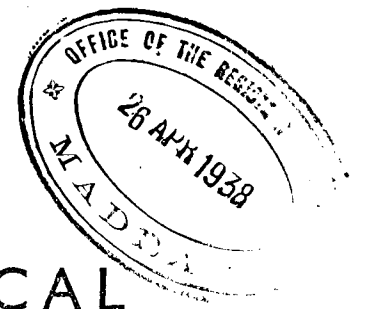
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MEDICO-SURGICAL SUGGESTIONS

VOL. VII

MARCH 1938

NO. 3

THE TREATMENT OF SPINAL CORD INJURIES.

BY LOYAL DAVIS, M.D.

Chicago.

Damage to the spinal cord or nerve roots which accompanies fractures or dislocations of the vertebræ seriously complicates otherwise simple therapeutic measures. In some of these cases the judgment of the physician is tested to its utmost to determine the proper pathologic diagnosis and the most effective therapeutic procedure. The question is not alone that of the indications for or against surgical measures ; rather the problem is one of diagnosis and treatment wherein anatomy, neurophysiology, and neuropathology play a greater part than is at first apparent.

Nearly all of the injuries of the spinal column which are associated with cord injury are produced by violent flexion or extension of the spine. The force may be applied to the lower end of the spinal column, as is illustrated commonly in falls from a considerable height or in automobile accidents ; or to the head, as is illustrated by diving into shallow water. The vertebræ may be dislocated, or, as is more common, there may be a fracture-dislocation. In addition to a fracture of the body of the vertebra, there may be a fracture of one or both articular processes with a forward displacement of one vertebra upon the next below or, the laminæ and processes may be involved. The degree of injury to the spinal cord is dependent upon two factors : the amount of displacement antero-posteriorly and laterally, and the rotation of the vertebra.

True anatomic damage to the cord may vary in degree from that caused by one or more small discrete areas of hemorrhage to larger lesions due to gross hemorrhages. Other foci of damage may be more mechanical in type and vary from partial compression to complete transection of the cord.

From a consideration of the anatomic relationships between the spinal cord and the vertebral column, it is not difficult to understand the mechanism by which the spinal cord is damaged in fractures of the vertebræ. Nor is it difficult to understand how severe

blows or jars to the spine may cause damage to the spinal cord with hemorrhages into its substance. In fact, the wonder is that of all the patients who receive fractures and fracture-dislocations of the vertebræ there are not more instances of injury to the spinal cord among them.

The injured spinal cords may show as diversified a picture as the patients show clinically. At the site of the injury there is an initial edema and hemorrhage, varying from petechial to gross within the substance of the cord. Early one can find swelling of the myelin sheaths, breaking up of the myelin, and later fragmentation of the axons. Phagocytes and scavenger cells of various origins, both from the blood (leukocytes) and from the nerve tissue (microglia, astrocytes), are in evidence, especially near and in the area of major damage. The ganglion cells undergo a change in cases of severe spinal shock even though recovery may occur. In severe local lesions the ganglion cells at the site of injury are involved to a greater degree; but even those cells some distance below the level of the lesion clearly present a picture not unlike that of beginning Wallerian degeneration, a state known as an axonal reaction. These cells usually recover and later may show no microscopic signs of previous damage.

It goes almost without saying that every case of injury to the spine should be subjected to a careful and painstaking neurologic examination. For this purpose a knowledge of some fundamental facts of neuroanatomy and neurophysiology and familiarity with a few important fiber tracts are necessary for the simplest understanding of spinal cord lesions and the resulting symptoms. In addition, however, there are certain facts which must be kept in mind about the vertical localization of motor, sensory, and reflex functions in the different segments of the spinal cord.

If only a few of the motor segmental levels are kept in mind they will serve as an aid in determining the location of a given lesion. For example, the motor segmental innervation of the deltoid muscles, which abduct and externally rotate the arm, is in the fifth cervical segment of the spinal cord. The biceps muscle is innervated by the sixth cervical segment, and the triceps by the seventh cervical segment. Therefore, given a patient with a spinal cord lesion, who can flex but is unable to extend his forearms, one may state definitely that the lesion does not extend higher than the seventh cervical segment. Likewise, the abdominal recti muscles above the umbilicus are innervated by the ninth and tenth thoracic segments, while the same muscles below the umbilicus are innervated by the eleventh and twelfth thoracic segments. Therefore, when the patient is asked to flex his neck with the hands by the side, if the umbilicus moves upward evidence is present of paresis or paralysis of the lower halves of the recti muscles. This is known as

Beevor's sign and aids in determining the level of a lesion at the tenth thoracic segment.

Many anatomic, physiologic and experimental investigations have shown that the skin of the body is divided into zones in relation to the various segments of the body. If one imagines the body in the all-fours position of our ancestors, and then makes transverse sections of the body at regular intervals, beginning at the neck and passing to the coccyx, the segmental sensory distribution of the spinal cord will be understood more clearly. Because in this posture the thumbs and great toes naturally are in an advanced position, it will be readily seen that the corresponding sides of the arms and legs will be sectioned at a higher level than will be the opposite sides. For this reason the radial sides of the upper extremities are represented by a higher segmental level of the spinal cord than are the ulnar aspects. In the same manner the medial aspects of the thighs and legs are of a higher segmental level than are the external sides of the lower extremities. The successive segmental levels in the thoracic region are understood easily, and as one passes caudally the segmental representation of the primitive tail is about the anus. It is apparent at once that a loss of sensation which corresponds to a segmental area is diagnostic not only of a spinal cord lesion but of its level as well.

To these general diagnostic aids may be added the important symptoms characteristic of *upper* and *lower motor neuron lesions*. *Lower motor neuron lesions* occur as the result of injury or disease of the anterior horn cells, the anterior roots or the peripheral nerves, and only of those structures. They are accompanied by a flaccid paralysis, loss of deep tendon reflexes, muscle atrophy, and the reaction of degeneration as determined by electrical stimulation of the muscles. Of these three possible sites of a lower motor neuron lesion, the presence of sensory changes is diagnostic of a peripheral nerve lesion. An *upper motor neuron lesion* is characterized by the presence of a spastic paralysis, exaggerated deep tendon reflexes, absence of muscle atrophy, pathologic reflexes (Babinski, Oppenheim, Gordon, Chaddock) and absence of superficial reflexes (abdominal and cremaster). Such a lesion may occur at any level in the corticospinal tract. The occurrence of cranial nerve paralysis, involvements of other functional systems and the distribution of the motor weakness will determine the level of the lesion. For example, a paraplegia occurs rarely in cortical lesions but is common in spinal cord lesions.

It is unfortunate that there are known criteria by means of which the doctor can differentiate clinically between a complete physiologic and a complete anatomic interruption of spinal cord function. The former may be a more or less rapidly recoverable lesion, but the latter is an irreparable damage from which recovery

does not occur. As in the case with peripheral nerve lesions, a complete physiologic interruption of the spinal cord cannot be differentiated from a complete anatomic section. Both are followed by complete paralysis in the muscles supplied by nerves which originate below the level of the injury. In both, complete sensory loss results below the level of the injured segment, and the reflex changes and bladder disturbances may be similar. Therefore, it becomes necessary to develop a method for the recognition of incomplete lesions.

Although many cases of incomplete anatomic lesions of the spinal cord show complete physiologic interruption, a large number may present only partial paralysis of the muscles below the level of the lesion or preservation of one or all types of sensation. In such cases the indications are quite clear. However, when the loss of motion and sensation is complete the problem is more difficult.

The symptomatology which follows a complete transection of the spinal cord may be divided into three stages :

The first is a stage of muscular flaccidity, corresponding to the period of spinal shock. In this state the paralyzed muscles are toneless and flabby, all the superficial and deep reflexes are lost, and there is a retention of urine and feces. At times retention of urine and incontinence of feces have been observed, and at times the cremasteric and bulbocavernosus reflexes have been elicited.

The second—or stage of reflex activity—begins with the first reflex response to an external stimulus, usually from the sole of the foot. In the full development of this stage a stimulus applied to any part of the lower extremity gives rise to a flexion reflex of the hip with adduction of the thigh, of the knee, and of the ankle. When reflexes can be evoked with ease, an extensive and widespread reflex action can be obtained which has been called a "mass reflex." This consists of a flexion spasm of the anterior abdominal wall and of the lower extremity, evacuation of the bladder, when its contents accrue to a certain amount, and sweating from an area from the skin in the paralyzed region. One of the most receptive fields of exciting reflexes is the genital area, and the cremasteric, dartos, bulbocavernosus reflexes and erection of the penis are commonly present. During this stage, in some cases the knee and ankle jerks can be evoked.

The third stage, that of gradual failure of reflex functions of the isolated spinal cord, usually preceding death, consists of the gradual return to a condition closely simulating the first stage.

Certain differences of reaction in incomplete lesions may be pointed out. In complete lesions the flexor type of movements is observed commonly, while in incomplete lesions extensor types of movements are present frequently. In complete lesions, the posture of the lower limbs is one of slight flexion; in partial lesions, extension. As a general rule, partial lesions of the spinal cord show a

condition comparable with that of a decerebrate animal in which there are defense reflexes with marked spasticity. Although an extensor type of response to plantar stimulation has been observed in complete section of the spinal cord, usually such a stimulation is followed by a plantar flexion of the toes, and as a fairly general rule, it may be stated that an extensor type of reflex is strongly indicative of an incomplete lesion. Inasmuch as prolonged states of toxemia, or septicemia from urinary sepsis, or bed sores, have a profound influence in hastening the reappearance of the reflex inactivity in cases of complete section of the spinal cord, it frequently occurs that from the practical standpoint, incomplete lesions are relatively easily recognized by the long persistence of spasticity and the signs of a paraplegia in extension.

Of particular value in the recognition of incomplete lesions is the early appearance of a Babinski sign; the failure to evoke mass reflexes from above the knee; a definite history of the absence of a state of spinal shock; marked tonicities in the paralyzed extremities; the involvement of both flexors and extensors in reflex movements provoked by the stimulation of receptive fields; and, of course, in obviously incomplete lesions, the absence of total paralysis or anesthesia below the level of the lesion.

The most discouraging experiences are upon record in the treatment of spinal cord injuries in the cervical region. We have observed eleven fracture-dislocations of the cervical vertebræ, all of which have been associated with definite symptoms of spinal cord injury. Six of these patients were injured by diving into shallow water; two were in automobile accidents; one patient had a pathological fracture of the fifth cervical vertebra due to a vertebral metastasis; one football player was among this group of patients; and another had injured himself falling down a flight of steps. The highest vertebral level was the fourth cervical and of these there were four while the other injuries involved the fifth, sixth, and seventh vertebræ. Fractures of the laminæ, spinous processes and dislocations anteriorly, posteriorly and laterally impinged upon the spinal canal in each of these patients, all of whom were operated upon, except one whose symptoms indicated an incomplete and spontaneously recoverable injury.

Injuries in the region opposite the cervical enlargement of the cord show characteristic symptoms. In all of our cases there was a more or less complete paralysis of the muscles of the lower extremities and the trunk with a partial paralysis of the muscles of one or both upper extremities. If the fifth and sixth cord segments are not injured the deltoid, biceps, brachialis anticus and supinator muscles are not involved although the muscles of the forearm and hands may be paralyzed. As Thorburn pointed out, these patients lie with the arms abducted, the forearms flexed and rotated outward.

The power of the upper extremities may be preserved in injuries below the first dorsal vertebra although weakness in the triceps can usually be detected. In one of our patients, injured in an automobile accident, a fracture-dislocation of the fifth cervical vertebra had produced an immediate and complete loss of motion and sensation in both arms and legs. Within three weeks a slight degree of motion had returned in his left arm and leg and on this side below the clavicle there was a complete loss to pain and temperature stimuli. Sensation on the right side was intact though motion was completely absent. This interesting group of symptoms, first described by Brown-Sequard, are indicative of a unilateral lesion of the spinal cord.

Severe pain in one or the other shoulder is prominent in injuries of the sixth cervical to the first dorsal vertebrae and there may be root hyperesthesias or anesthetics over the shoulder, in the axilla, or on the radial or ulnar sides of the upper extremities. If the lesion extends up to the fifth or sixth cervical segment the sensory disturbances will affect the radial side of one or both forearms, but if it does not extend above the seventh cervical segment only the ulnar sides of the upper extremities are involved. Priapism is frequent in injuries of the cervical cord and an elevation of the temperature to 107 to 108 degrees is quite characteristic. This type of hyperthermia is not affected by the common therapeutic measures employed to lower the temperature in other conditions.

Lesions of the brachial plexus may often accompany compression injuries of the cervical spinal cord. These are usually of two clinical types. There may be an immediate paralysis of all four extremities, which is quickly followed by the return of motion in one arm and leg. The opposite arm then presents a flaccid paralysis, the corresponding leg a spastic paralysis and there is loss of pain and temperature sensation on that side of the body in which motion has returned to the extremities. The spastic paralysis of the leg may eventually disappear and a residual flaccid paralysis of the upper extremity remains.

In another clinical type, the patient may sustain an injury to the brachial plexus and then several days or weeks later may develop a slowly progressive paralysis of the leg on the same side, with loss of pain and temperature sensation on the opposite side. In these cases it is necessary to determine whether the flaccid paralysis of the arm, distinctly a lower motor neuron lesion, is due to an injury to the nerves of the plexus, to the roots, or to the anterior horn cells of the spinal cord.

If the lesion is one of the plexus itself, the loss of touch sensation will be greater than the loss to pain. If it affects the roots, the loss of pain sensation will be more extensive than the loss to touch. If

only the anterior horn cells are involved, sensation will be unaffected. However, if sensory loss exists with such a lesion of the gray matter, then light and pressure touch as well as pin prick and pressure pain will be absent. Finally, if the paralysis in the leg disappears relatively soon and the arm paralysis persists, it speaks for a brachial plexus lesion as against one of the spinal cord.

We have operated upon eight patients with injuries of the spinal cord due to fracture-dislocations of the eighth to twelfth thoracic vertebrae, but none which have involved the upper thoracic spine. All of these patients were injured in automobile accidents except two who fell from a height and struck in the sitting posture and one coal miner, who was dragged for a hundred feet in a jack-knife position. The rather characteristic symptoms produced by involvement of the twelfth thoracic vertebra are illustrated in the following case as are the dangerous results which are likely to follow unskilful attempts to reduce or "adjust" such dislocations :

A young woman fell down a flight of stair steps and immediately lost control of her legs and sphincters as well as suffering a loss of sensation to all stimuli below the level of the tenth thoracic skin segment. At the time of her injury she was $4\frac{1}{2}$ months pregnant, but successfully delivered her baby at term. For two years she was subjected to attempts designed to reduce a dislocation of the twelfth thoracic vertebra. When she entered the hospital, the motor and sensory findings were unchanged ; the deep tendon reflexes of the lower extremities were increased ; pathological reflexes were present and the lower abdominal reflexes were absent.

A complete spinal subarachnoid space block was present and at operation the spinal cord was found to be compressed by the fractured twelfth thoracic vertebra. The dura matter was thick ; the arachnoid was thickened and inseparable from the underlying traumatized spinal cord.

Loss of the abdominal reflexes and a loss of sensation over the abdominal wall corresponding to the sensory distribution of the segments involved are characteristic of lesions in the lower half of the thoracic spinal cord. Since the upper halves of the abdominal recti muscles are innervated by the eighth to tenth and the lower halves by the tenth to twelfth segments, Beevor's sign may be present. If the patient raises his head from the bed, with his arms at his sides, the umbilicus is pulled upward if the lesion is at the level of the tenth segment because of the paralysis of the lower halves of the recti. Likewise, the upper abdominal reflexes on one or both sides may be preserved while the lowers are absent. Girdlelike root pains are most frequent in injuries of the midthoracic vertebrae.

Injuries of the lumbar vertebrae have occurred in each of our cases as the result of falling from horses ; being thrown from automobiles, or jumping from a height ; but in each instance the force

of the injury has been received on the buttocks or the extended lower extremities. The spinal cord ends opposite the superior border of the second lumbar vertebra and below that level the spinal canal is occupied by the cauda equina. Consequently, the symptoms of a lower motor neuron lesion are frequently characteristic of an injury to the lumbar vertebrae. Flaccid paralysis, loss of the deep tendon reflexes, muscle atrophy, reaction of degeneration and loss of sensation in the area of distribution of the lumbar and sacral segments are the usual symptoms. However, because of the nature of the cauda equina, involvement of motor and sensory function may be very asymmetrical in the lower extremities. In one of our patients who had a compression fracture and an anterior dislocation of the first lumbar vertebra, the most extensive sensory loss was in the second and third lumbar skin segments on the left side. Likewise, many of the muscles of the right leg continued to react to the faradic current, whereas in the left leg faradic reactions were completely lost.

Retention of urine and incontinence of feces are prominent symptoms. Excruciating pains in the back, or radiating pains into the perineum, genitals, and often down the posterior surfaces of the thighs occur frequently as the result of root irritation produced by the fragments of fractured bone.

There are two problems which must be solved in the decision for or against surgical intervention in fracture-dislocations of the spine. First, is there a demonstrable block of the subarachnoid space, which means compression of the spinal cord? Second, is there x-ray evidence of bony encroachment upon the spinal canal? Positive evidence of subarachnoid block usually implies bony encroachment on the spinal canal. It must be emphasized that a complete anatomic lesion of the spinal cord cannot be differentiated from a complete physiologic section. Therefore, unless one has inspected the cord at operation there can be no justification for the assumption that the cord has been divided completely. Surgical intervention may be useless and often harmful in the presence of a complete transverse anatomical lesion of the spinal cord but, as has been pointed out, a complete transverse physiological lesion may be present from compression by a dislocated vertebra. It would appear that the benefit which may be derived from operative interference is based simply upon the question of whether or not there is pressure upon the cord by a dislocated vertebra or fractured fragments of bone. In the presence of such pressure there is no question as to the benefit to be gained by an operation. Perhaps the most valuable aid in addition to the x-ray and a careful physical examination is the employment of the Quackenstedt test of pressure upon the jugular veins.

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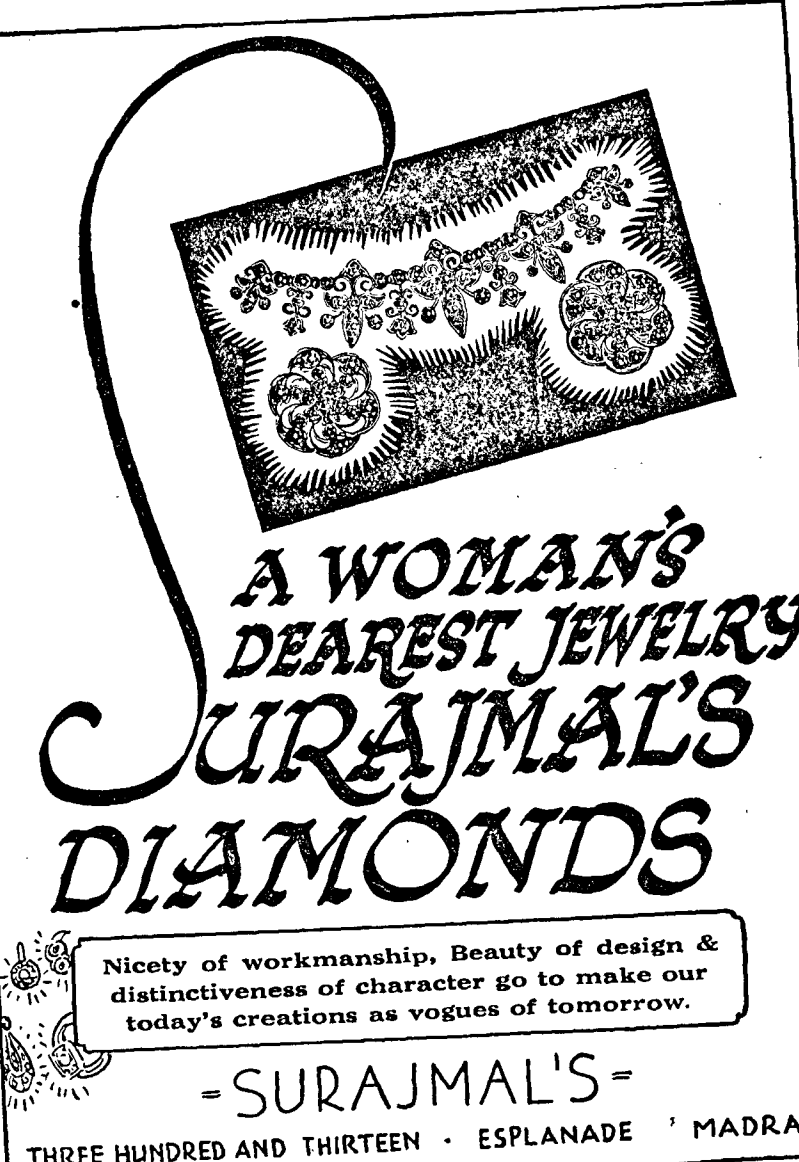
pressure (Queckenstedt's sign). This rise in pressure is transmitted normally to the fluid in the spinal subarachnoid space. Release of jugular compression in normal subjects is followed by a prompt return of the pressure to approximately its original level. When, however, this increase pressure does not take place on compression of the jugular veins, it is indicative of an obliteration of the spinal subarachnoid space. Cases of partial block may be recognized by the slowness with which the pressure changes in the spinal system after compression of the jugular veins; the less than normal rise produced, and the comparatively long time it takes for the pressure to return after release of the jugulars.

Often the character of the dislocation or rotation of the fractured vertebra as shown upon the x-ray films is in itself sufficient indication for surgical intervention. However, occasionally, symptoms of cord injury may be present in the absence of x-ray evidence of bone pathology. In such instances we have come to depend entirely upon the presence or absence of a subarachnoid space block and in each of our patients operated upon a manometric block was demonstrated. It is justifiable to perform successive Queckenstedt tests to follow the persistence or disappearance of the block. This is true particularly if there are clinical evidences of improvement in the symptoms. Likewise, in the presence of demonstrable x ray evidence of bone pathology and in the absence of a subarachnoid space block, one is justified in waiting for a spontaneous improvement of symptoms. In other words, the demonstration of compression of the spinal cord is the indication for surgical treatment and not the supposed extent or character of the spinal cord damage.

The mere fact that there are signs of neurologic damage in injuries of the spine does not, however, constitute a surgical indication. In cases of spinal injury with minor neurologic symptoms, or signs of a recovering lesion, and no evidence of subarachnoid space block, treatment by the usual orthopedic measures is probably the method of choice.

With manifestations of neurologic involvement of a definite degree, where there is compression sufficient to cause a subarachnoid space block, there exists a definite indication for laminectomy. It is conceivable that certain cases may present themselves with major neurologic signs and x-ray evidence of severe bone injury (crushing and fragmentation with spicules driven into the spinal canal) which do not give evidence of a block on manometric study. These cases should certainly be operated upon.

There remain some patients who have a questionable amount of permanent neurologic damage, questionable block, and questionable x-ray evidence of encroachment upon the spinal canal. It is in these cases that perhaps the definitive judgment of the neurologist



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and surgeon is tested to its utmost. With these patients it is probably better to err on the side of a possible unnecessary operation.

With the knowledge that the spinal cord does not regenerate once it is anatomically severed, even if one believes in certain cases that the displacement and fragmentation of the bone has been so great as most certainly to transect the cord, these patients should not be denied an opportunity for recovery. In some, recovery will occur when the pressure is relieved, and no one can tell by clinical examination alone that a complete irreparable lesion is present.

Occasionally a patient presents himself with evidences of spinal cord damage at a time after the injury to the spine when the bony damage has completely healed. The question of surgical intervention in these cases is at times a delicate one. Here so much depends upon the nature of the lesion, the degree of disability it is causing, and the course of its progress that no inclusive rules can properly be laid down. In these cases every effort should be made to arrive at an accurate pathologic diagnosis before submitting the patient to an operation. Then probably only those cases with lesions which offer some chance for surgical relief should be operated upon.

The time of operation after receipt of the injury is important and is dependent upon the demonstration of a subarachnoid space block in addition to the physical condition of the patient. One may with justification wait for several hours until the immediate state of general shock has subsided before a laminectomy is performed; but this period should not be extended to the point of waiting for the cessation of symptoms of improvement in the presence of a block. Though considerable recovery may occur spontaneously, the mortality of the operation of laminectomy in competent hands is too low to justify such conservatism.

First-Aid Care. It is imperative that trauma to the spinal cord be recognized at the scene of the accident because careless transportation may be responsible for irreparable damage to the spinal cord. The public must be instructed that an injured patient unable to move his arms and legs or legs alone should not be moved except under the direct supervision of a doctor.

Attempts to lift the patient's head to enable him to drink water, thus flexing the chin onto the chest in the presence of a fracture-dislocation of the cervical vertebræ may destroy whatever cord function remains. If the patient can move his arms, hands, and fingers freely, but is unable to move his legs, he may be rolled over face downward, without lifting on the presumption that the thoracolumbar region is injured. If the arms and legs are paralyzed, the patient should be moved cautiously by three or four persons; one exerting gentle traction on the head and one on the feet while the others carry him, lifting beneath the shoulders, trunk, and hips.

In all cases the back should be carefully supported. If transported on the back, a rigid stretcher or even a solid one is better for these cases than a soft bed, in which they may sink and thus increase the fracture deformity. In fractures of the cervical spine, the head should be kept as if one piece with the shoulders when moving or turning the patient. If these precautions are rigorously observed, the injured patient may be transported in safety.

GENERAL TREATMENT.

Care of the Urinary Bladder. The care of the bladder in these cases is a question concerning which there is probably more discussion and less unanimity of opinion. For a better knowledge of the subject it is well to understand the mechanisms behind the production of the so-called cord bladder. The neurogenic bladder may be defined as a bladder malfunctioning because of a derangement in the normal physiologic nervous mechanism which controls the emptying and filling of the organ. Because of the disruption of this mechanism in injuries of the spinal cord one is faced with various types of malfunction. There may be complete incontinence or retention with overflow. In some cases a so-called automatic bladder may develop, with more or less complete evacuation at intervals.

It must be remembered that the innervation of the bladder is all derived from the lower lumbar and sacral levels of the cord and that theoretically there may not be so much interference with bladder function in cord lesions above this level except for the loss of voluntary control over voiding. Practically, this works out in a satisfactory manner in handling the bladder disturbances of these patients. Centres in the sacral region of the cord below the site of the lesion keep up a slight tonus in the detrusor, and apparently give rise to an automatic voiding reflex when the bladder becomes distended. While much weaker perhaps than the normal voiding reflex, this reflex may be increased by stimulation reaching the cord below the level of the lesion. This accounts for the emptying of the bladder as part of the mass reflex excited in some complete lesions of the cord by pinching or stroking the legs.

Urinary infection alone will prevent a return of function in the spinal cord. The bladder should not be allowed to distend until overflow occurs and dribbling takes place. The stagnant urine distends an already paralyzed bladder wall beyond the possibility of recovery of its contractile power. Further, an ascending urinary tract infection occurs which endangers the life of the patient, and his possible chances for recovery. Likewise, frequent catheterizations cannot be performed without traumatizing the urethra and, even in the most careful hands, introducing the danger of infection. Therefore, a retention catheter should be introduced and left in the bladder for from ten days to two weeks at a time. A clamp should be applied and opened at intervals of four to five hours, depending upon the

rapidity with which the bladder fills. This procedure will aid materially in establishing an "automatic bladder." The bladder may be irrigated daily with warm boric acid solution. Experience has shown that the use of the retention catheter is the most successful method of preventing urinary sepsis. In cases which are operated upon after urinary infection has occurred, a suprapubic cystostomy may be the only means of treating a serious bladder infection.

Decubitus Ulcers. It is extremely important to guard against decubitus lesions of the skin in the anesthetic areas of the body and extremities. The most successful method is to place the patient upon a water or air mattress which allows an equal distribution of the patient's weight. As has been pointed out, the loss of sensation is variable as a result of spinal cord lesions and involvement of the cauda equina is characterized commonly by sensory loss about the buttocks which has given rise to the term "saddle anesthesia."

The skin must be kept scrupulously clean and dry. Alcohol rubs and the application of cocoa butter should be given freely. As soon as is deemed wise, the patient should be turned on the side and propped there by pillows, so that strain on the paravertebral muscles is avoided and the same relative position of the spinal column is maintained. Harsh, stiff linen sheets which burn the skin must be avoided. The linen should be changed immediately if it becomes soiled from an incontinence of urine or feces. Nurses should be instructed about the case with which hot water bags may produce blisters of the insensitive skin, which are commonly the beginning of a large spreading ulcer.

If they develop or are already present, decubitus lesions may be treated with excellent results by daily exposure to ultraviolet light. The necrotic edges of large ulcers should be debrided and all undermining pockets of necrotic tissue removed. The combination of Dakin's solution irrigations and dry heat are also efficient in the treatment of this type of lesion.

After care. The aim of treatment of spinal cord injuries should be to restore the patient to an economic and social independence. It is a common error to look upon the disabilities which result from these injuries as hopeless. Many of the crippling deformities and contractures which are observed commonly after injury to the spinal cord can be entirely avoided or to a great extent improved.

Gentle massage of the muscles, the use of radiant heat, and the whirlpool bath are all agents which have proven helpful. Passive movements may be carried out very well in conjunction with massage. They help to stretch contractures which have already occurred and to prevent those which invariably occur in an inactive or denervated muscle. They also increase the range of motion in an already stiffened joint and help to keep a mobile joint active so that when the time comes it is ready to perform its part as an effector

mechanism. Such movements help to re-educate the muscles in performing normal movements. These exercises should be carried out slowly, gently, and never with quick, jerky movements. Each separate passive exercise should be individualized, and the patient be required to make the attempt to perform the movement simultaneously or to attempt to hold the part in the position imposed upon it.

The use of electrotherapy in spastic paraplegic patients accomplishes very little as compared to the results which may be obtained in the flaccid paralysis. Electrotherapy is indicated to prevent the atrophy of muscles and fibrosis, to increase nutrition and to conserve the functional capacity of paralyzed muscles until sufficient return of function has taken place to permit of active motion.

Splints have been found of little or no use in the treatment of spastic paralysis in the lower extremities. Deformities and contractures may be prevented in part from developing by massage and passive movements of joints. When the patient is confined to bed the extremities should be kept in extension and in abduction by the use of sandbags and pillows. The tendency for these patients to lie upon their sides constantly, with the extremities flexed, must be prevented. On the other hand, marked flexor and adductor reflex spasms may occur in spite of all efforts to prevent them. Constant supervision and careful nursing may prevent the grotesque, disabling deformities which may otherwise occur. When contractures have developed splinting is of little value, and operative intervention is indicated. Section of the obturator nerves, posterior root sections and tendon divisions have been employed in these cases to make the patient more comfortable, and his nursing care less laborious.

Much can be done by the intelligent use of splints in cases of flaccid paraplegia. While the patient is in bed, foot drop may be avoided by the use of sandbags against the soles of the patient's feet and a cradle over the legs to keep the weight of the bed clothes off the feet. These require careful and constant adjustments, and it may prove more desirable to use light crinoline posterior molded splints which may be removed easily for massage and passive movements.

When the patient becomes ambulatory the most satisfactory splint to prevent foot drop is a simple one which can be attached to the shoe easily. As in usually the case, the remainder of the extremity also requires support. A modification of the Thomas caliper splint may be employed; a spring lock at the knee joint enables the patient to flex the knee when he sits. When he is standing or walking this lock closes and the knee is kept in extension. The additional foot drop splint may be attached very simply to this type of apparatus.

Exercises and Muscle Re-education. Very often the paraplegic patient is looked upon by his physician as hopeless. Many times

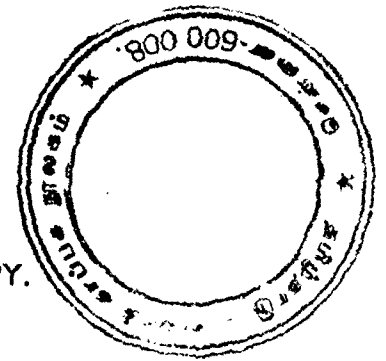
massage, active and passive movements, electrotherapy and splinting may be carried out quite effectively until the patient is ambulatory. It is necessary to teach him to use his recovering muscles to his best advantage. He must be taught to walk again. While one may feel that this will be accomplished gradually in spite of what may be done, there is no question but that progress has been more rapid as the result of directed muscle exercises and re-education.

One of the simplest devices to teach the patient to use his extremities may be made of small iron pipes and is similar to a baby's "walker." The patient stands within this frame, which is on rollers, and rests his weight upon his arms, which grasp its sides. Another excellent method of re-education in walking is to have the patient walk in an aisle bounded by iron pipes, which may be grasped easily, and upon which he may rest his weight as he progresses. The floor of this aisle consists of a board upon which are painted numbered square spaces, varying in size. The patient attempts to place his feet within these spaces and as he progresses the wider spaces are utilized. The results of this method may be increased by having the patient observe the movements of his legs in a mirror.

It has also been found that the movements of the lower extremities may be performed more easily in slightly warmed water. This is true of the spastic as well as the flaccid paralysis. A Hubbard tank is an excellent means of carrying out these under-water movements. This may be installed in the basement of the home at relatively little expense, and is extremely valuable.

The few examples given of methods of re-education of the muscles of the lower extremities can be multiplied many times. In more advanced cases a stationary bicycle may be used or a rowing-machine employed to advantage. Just as in the re-education of the upper extremities, educative exercises which are productive intrigue the patient, and secure his whole-hearted co-operation.

(*Illinois Medical Journal*, Sep. 1937.)



ARTIFICIAL FEVER THERAPY.

A GENERAL REVIEW.

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(Presented before the Annual Session of the Arizona State Medical Association, April 1-4-1937).

Heat, applied in different ways to the human body, has been a therapeutic measure since early times. The present-day concept of artificial fever for the treatment of disease is, however, comparatively new, dating from Von Juarregg's report in 1918 upon the use of malaria for the treatment of general paresis. Since that time much work has been done to place fever therapy upon a scientific basis.

In recent years many methods for inducing artificial fever have been introduced. Inoculation with the parasites of relapsing fever and rat-bite fever, injections of a typhoid-paratyphoid vaccine, injections of sulphur in oil, diathermy, radiotherapy, and the inductotherm, an electrically heated blanket, heat cabinets, hot baths, and a method which we have described and called the "blanket method," have been used for elevating body temperature.

The essential factors appear to be the height and duration of fever, rather than the modality employed. Comparative therapeutic results have been obtained with many of these methods. The investigative work which has been done in this field of therapy has led to a simplification of a technic and a lessening of the hazards of fever therapy.

In 1934, we noted that if a patient is wrapped in blankets in such a way that no portion of his body is exposed except the face, and if warm fluids are given by mouth, the patient's temperature will rise gradually and within 4 to 5 hours will reach 40° C. (104° F). Making use of this principle, we have developed a simple method for the production of artificial fever which we have called the "blanket method."

Briefly, the technic of this method is as follows: the patient is wrapped in thin bath-blankets in such a way that no part of the body is exposed except the face. The heels are protected by gauze dressings and heavy woolen socks. The patient lies upon a canvas sheet, a rubber sheet and 2 or 3 heavy woolen blankets. A common electric body-bake containing 12 40-Watt carbon electric lights is placed over him, and 2 heavy woolen blankets are brought over the bake forming a closed compartment. The front end of the bake is closed with a blanket so that only the head is exposed.

Within 2 hours after turning on the lights, the patient's temperature rises to 40°C. (104° F.) The bake is then removed, and the patient is wrapped, but not tightly, with the canvas and rubber sheets and over these the heavy woolen blankets.

The patient's temperature is taken orally at ½-hour intervals until it reaches 38° C. (99.5° F.), then at 15-minute intervals until it reaches 40° C. (104° F.), and after that every 5 minutes by axilla until the treatment is concluded. The thermometer is left in the axilla throughout the rest of the treatment. Warm fluids containing 40 gm. of sugar and 10 gm. of salt to the liter are administered throughout the treatment; about 200 c.c. are given every half-hour. Morphine, gr. ¼, is given hypodermically when the temperature reaches the desired height; this may be repeated in a ⅙ gr. dose in 2 hours if necessary.

The temperature is regulated during the treatment by loosening or removing the blankets if it tends to exceed the desired height. Any degree of fever can readily be reached by this method, and the temperature is easily controlled.

The usual treatment consists maintaining the temperature at the desired height for 5 hours, and terminating the fever by removal of the blankets. The patient is bathed and given an alcohol rub. If he is not nauseated, a light supper may be given.

During a single treatment, 3,000 to 4,500 c.c. of fluid are given. If the patient does not take fluids well by mouth, 1,000 c.c. of normal saline and 5 per cent glucose may be given intravenously at the conclusion of the treatment.

Mortality: We have employed this method of obtaining artificial fever at the Mount Zion Hospital and the University of California Hospital in over 1,800 instances. Under ideal conditions and with our present knowledge hazards from this type of therapy have been reduced to a minimum. At the University of California Hospital where 1,500 treatments have been given, one fatality occurred in a badly deteriorated parietic. Our results have been in striking contrast to those from material therapy in which the mortality has varied from 3½ to 10 per cent. Fever therapy under any circumstances should be regarded as a serious procedure, and should be given with the greatest of care.

Contraindications: The candidate for fever therapy should be in good physical condition. No serious cardiac or renal lesion should be present. Active tuberculosis or other respiratory infections, obesity, marked debility from wasting disease, and hypertension are contraindications for fever therapy. Advanced age, i.e. over 65 years, should be considered an added hazard. If the patient is not a good risk for fever therapy, and it is urgently needed, the treatment should be modified to meet the conditions.

Untoward Reactions: Following are the untoward reactions which may be encountered.

1. **A rapid rise in temperature to dangerous heights:** This is most apt to occur when high temperatures are obtained, such as 40° to 41°C. (104° to 106° F.). The patient's temperatures may rise suddenly to 41.5°-42.5° C. (106.7°-109° F.) and the patient may go into a state of heat collapse. If the temperature is accurately recorded every 5 minutes and properly regulated, it is not likely that the rapid rise will occur. The pulse is also a good indication of the patients' condition. As a rule, the pulse rate does not exceed 140-150 per minute at the height of the temperature; a rise above 160 per minute is an indication to discontinue treatment.

Such a rapid rise of temperature is not usually serious if it is detected and combatted promptly. Immediate measures to lower the temperature should be instituted, such as cold sponges, cold colonic flushes, and an intravenous injection of 1,000 c.c. of 5 per cent glucose in normal saline solution. It has been our experience that this reaction occurs most frequently in badly deteriorated parietics or in individuals who have been greatly debilitated by their disease.

2. **A secondary rise in temperature** occurs occasionally several hours after treatment is completed. This should be watched for and combatted.

3. **Nausea and vomiting** are not uncommon symptoms during and immediately following treatment. They are not likely to occur if sufficient fluids are administered during the treatment.

4. **Hematemesis** occurred in 2 of our cases.

5. **Auricular fibrillation** developed in 1 case.

6. **Tetany** is occasionally seen.

7. **Herpes simplex** is of common occurrence, and usually follows the first treatment although sometimes the second and third. It is more likely to appear when high temperatures are induced.

Indications for artificial fever: Krusen recently reviewed the reports on fever therapy which have been published during the past year. He included over 100 such reports which indicated that fever therapy was used for a wide variety of conditions. Artificial fever is not a panacea. According to our present knowledge it is, however, of proved value in the treatment of syphilis, gonorrhea, chorea, interstitial keratitis, certain forms of chronic arthritis, and bronchial asthma.

In treatment of syphilis: My own interest in fever therapy has been centered mainly in its use in the treatment of syphilis. The exact way in which fever produces beneficial results in syphilis is not known, but there is considerable evidence to show that high temperatures have a direct effect upon the *treponema pallidum*. Warren and his co-workers have shown that the *treponema pallidum* is destroyed in vitro if a temperature of 39°C. (102.2°F.) is maintained

for 5 hours, of 40°C. (104°F.) for 3 hours, of 41°C. (105.8°F.) for 2 hours, and 41.5°C. (106.3°F.) for 1 hour. Bessemans and Thiry recently showed that in humans it required a tissue temperature of 42°C (107.5°F.) sustained for 1 hour, or of 40°C. (104°F.) sustained for 2 hours, to render the *treponema pallidum* avirulent and immobile. They heated syphilitic lesions by means of hot water and diathermy, and determined the tissue temperature by means of a needle thermocouple. Primary and secondary lesions treated in this way involuted promptly, while the unheated lesions progressed in the usual manner. Many others have shown that in rabbits heat alone causes the disappearance of the *treponema pallidum* from syphilitic lesions, and can cause these lesions to heal promptly. Artificial fever can prevent the successful inoculation of *treponema pallidum* in experimental animals.

It is evident from the effect of heat upon the *treponema pallidum* in experimental animals, in the test tube, and in human tissues, that high temperatures which can be easily and safely produced artificially should be beneficial in the treatment of human syphilis. In addition to the direct action of high temperatures upon the invading organisms other factors such as increased tissue resistance undoubtedly play a part in the beneficial effects obtained.

In 1934, we treated 33 patients in the early phase of syphilis with fever alone. The effects upon the dark-field examinations, the clinical course of the lesions, and the serologic tests were noted. While in 94 per cent of the cases the dark-field examinations were rendered negative and the lesions involuted promptly, it soon became evident that the fever did not sterilize the patient of *treponema pallidum*. The blood Wassermann reaction was not reversed to negative in any of the patients; and clinical relapse occurred in 3 cases shortly after the treatment was discontinued. These findings are similar to those of other workers.

Recently Neymann reported good results in early syphilis by combining drug and fever therapy.

The use of artificial fever in other forms of systemic syphilis, including the "Wassermann fast" group, has not, in our experience, been promising.

The effects of fever upon general paresis, tabo-paresis, tabes dorsalis and other forms of neurosyphilis is at times striking. Artificial fever therapy is a valuable adjunct in the treatment of all forms of neurosyphilis.

An analysis of the results which we obtained in a series of 55 patients with neurosyphilis is presented. The study represents our experience up to May, 1936. In this group there were 37 males and 21 females; their ages varied from 18 to 71 years, although most of the patients were between 35 and 45 years of age. The average age was 39.6 years. The cases were classified according to the type of neurosyphilis. All patients were in the late phase of syphilis. There

were 12 with general paresis, 8 with tabo-paresis, 14 with tabes dorsalis, 7 with the meningovascular type; 17 were asymptomatic.

All received antisyphilitic drug therapy both before and after fever therapy. The large majority were resistant cases; treatment had been continued for a considerable period without material effect upon the spinal fluid findings before fever therapy was instituted. The average period of observation was 1.6 years before fever therapy, and 1.4 years after. An attempt was made to give each patient 10 weekly fever treatments, each consisting of a temperature of 40°C. (104°F.) or over, maintained for 5 hours.

Serologic results - spinal fluid: Among 55 cases, in 35 or 63 per cent the Wassermann reaction of the spinal fluids changed from positive to completely negative; in 13 or 23.4 per cent, it became less positive; in 3 cases it became more positive; and in 4 cases it did not change. Thus the spinal fluid in 86.4 per cent of the cases either became negative or improved.

The changes in the colloidal gold curve were not as striking, although in 25 cases, or 47.1 per cent, it became normal; in 40 per cent it became less positive; in 2 cases it became more positive; and in 4 cases there was no change. The colloidal gold reaction is slower to change under treatment than the other elements in the spinal fluid. Taking the group as a whole, the gold curve in 81.1 per cent of the cases either became normal, or improved and gave promise of becoming normal with continued treatment. The cellcount responded promptly to treatment; in 32, or 91 per cent of 33 cases in which the cell-counts were abnormal they were reduced to normal. In 2 cases there was a decrease in the number of cells although not to normal; and in only 1 case did the number of cells increase. The proteins in the spinal fluid were reduced to normal in 25 cases, or 59.5 per cent, in 15 cases, 35.7 per cent, they were reduced but not to normal. In 2 cases there was an increase in the proteins.

Serologic results—blood: The blood Wassermann and Kahn reactions did not respond as well as did the spinal fluid elements. In 16 cases, or 28 per cent, the blood Wassermann reactions became negative; in 9 or 15.5 per cent, it became less positive; in 12 or 21 per cent, it remained positive; in 2 it changed from negative to positive; and in 8, or 14 per cent, it was negative before fever therapy was instituted and remained so. In 43 per cent of the cases the test either became negative or less positive. The Kahn reaction of the blood in general paralleled that of the Wassermann, although it reversed somewhat less easily.

Changes in clinical status: For the most part the clinical improvement corresponded to serologic improvement, although the clinical change took place long before the serologic findings changed. The maximum serologic change did not become apparent for a year or

longer, whereas the clinical symptoms tended to disappear within the first few months after treatment was instituted.

Of the 20 cases of general paresis and tabo-paresis, only 2 patients had to be institutionalized. Five of the remaining were markedly confused mentally, but following treatment they improved sufficiently so that they were able to return to work. The rest of the cases were milder in type, and the disease in these patients has apparently been arrested. If the cerebral damage has been great, no amount of treatment can restore the patient to a normal condition. In many instances, although the disease is apparently arrested, the patient continues to show progressive mental deterioration. Therefore, therapy should be instituted as early as possible in the course of the disease.

In tabes dorsalis, optic atrophy and ataxia were little relieved. One outstanding effect of fever therapy was the elimination of lightning pains and crises. In practically every patient with severe pain, this symptom was relieved during the treatment. The relief was only temporary in some cases, lasting a few weeks or months; in others it was permanent.

Although artificial fever therapy was originally introduced for the treatment of neurosyphilis, it is of greatest usefulness in gonococcal infections: The gonococcus is susceptible to temperatures that can be safely attained in human beings. Different workers have reported conflicting data on the exact deathtime of the gonococcus. However, Boak, Carpenter and Warran carefully studied the effect of heat on 130 different strains of gonococci in test-tubes. They found that gonococci generally are destroyed at a temperature of 41.1° C. (106° F.) to 41.6° C. (107° F.) in from 6 to 27 hours.

A review of the literature on the effects of artificial fever therapy in gonorrhea and its complications show that this form of treatment yields excellent results. Our own experience in the treatment of gonorrheal arthritis, epididymitis, prostatitis, urethritis and salpingitis, is in accord with that of others.

Acute gonorrheal arthritis responds with surprising promptness. The pain frequently leaves after 1 treatment, and most of the cases clear completely after 4 to 6 treatments. The temperature attained for treatment of this type of infection should be higher than for treatment of neurosyphilis—usually 41° C. (105° F.) for 5 to 7 hours. The treatments are given at shorter intervals, i.e. every 4 to 5 days, for best results. Chronic gonorrheal arthritis responds less well; some patients obtain complete relief but the majority we are only partially benefited.

Epididymitis, prostatitis and salpingitis respond well to fever therapy; urethritis is somewhat more resistant.

Treatment of other diseases: We have had some experience in the treatment of various other types of arthritis; although some of the

patients received temporary benefit the results in general have been disappointing.

A number of patients with multiple sclerosis have been treated. The large majority received no benefit; only 2 or 3 seemed improved.

In mycosis fungoides, psoriasis, Hodgkin's disease, and lympho-granuloma inguinale, fever therapy was found to be of no value.

Comment: Artificial fever can, under certain conditions, be produced in patients with little difficulty and without hazard. This accomplishment is the result of years of work and study. Fever therapy has a definite place in our therapeutic armamentarium.

The exact way in which high temperatures produce their effect is not completely understood. There is probably a direct action upon the invading organisms as well as upon the infected host. In some way, fever hastens nature's attempt at cure.

It is of little importance whether fever is developed within the body i.e. by inoculation with malaria, by typhoid injections or by diathermy; or whether it is produced by placing the patient in a hot bath or a heat cabinet. The only factor of importance is that the elevation of temperature be of sufficient height and duration. The method of choice is the simplest and least hazardous.

Methods employing physical means are preferable to those using biologic agents because they are more controllable, and because their effect upon the patient is less debilitating. At the present time we can employ fever as we do any other therapeutic agent. We can prescribe the exact height of temperature desired, and the rise in temperature can be stopped at any point that is indicated. The treatment can be altered to meet the various conditions that are encountered in clinical medicine. If a patient is a poor risk, lower temperatures can be induced and the treatment stopped immediately if the patient does not tolerate it. These advantages are of great importance.

Artificial fever can be induced in patients by means of simple and expensive equipment. While high temperatures can be induced easily, we must never regard this form of therapy lightly. The patient with a temperature of 40° to 41° (104° to 106° F.) is acutely ill and requires the utmost care. The treatment is a hospital procedure, and should be conducted only by trained personnel.

Lastly, I wish to point out that fever therapy is not a panacea for all diseases. It has unquestionable value in the treatment of all types of gonococcal infections and in neurosyphilis; its value in the therapy of other diseases remains to be proved.

Summary: 1. A review of the subject of artificial fever is presented.

2. The blanket method of inducing artificial fever is described.

3. The serologic and clinical results in a series 58 patients with neurosyphilis treated by means of artificial fever and drug therapy, are given.

4. The value of artificial fever therapy in gonococcal infections is discussed.

—(South Western Medicine Sept. '38.)

TUBERCULOSIS OF THE THROAT.

HUGH J. EVANS, M.D.

Tulsa.

I am presenting this paper, not with the idea of adding anything new or startling, but merely wish to review five cases which I happened to observe while in the Clinic at the Eye, Ear, Nose and Throat Hospital in New Orleans. The prime object of the paper is a plea for an early diagnosis of this condition.

Tuberculosis of the throat is simply one of the many manifestations of this devastating disease, but it is one that most particularly concerns the otolaryngologist.

The first recorded article on this subject was that by Morgagni prior to 1767. Since then a great deal has been written, more indeed since the invention and use of the laryngoscope by Gorcia in 1855.

This disease is caused by the tubercle bacillus and is almost always secondary to pulmonary tuberculosis. Spencer states that he has never seen a primary infection during twenty years of his practice. Arrowsmith, Levy, Hastings, Sir St. Clair, Thompson, and Dworetzky corroborate Spencer's remark. Dworetzky reports the disease most frequent between the ages of twenty and forty-five years. It is extremely rare in children. Males are more subject to it than females, due probably to greater exposure and the most extensive use of tobacco and alcohol.

The subjective symptoms may be summarized by the findings of the one hundred cases, sixty five males and thirty-five females, reported by Taylor and Nathansen. They found hoarseness in eighty-five, laryngeal pain in eighteen, dysphagia in seven, aphonia in nine, dryness in the throat in nine, and tickling in the throat in four. To this list may be added dyspnea, which Spencer found as a frequent complication.

Objectively we find conditions varying from anemia of the larynx and epiglottis to marked redness and deep infiltration of the interarytenoid region. Tuberculomata may be present in any part of the larynx or pharynx. Ulcers vary from superficial to quite deep seated and extensive lesions. Edema, causing great alarm and much discomfort, may be encountered.

In making a diagnosis it is essential to have the co-operation of the internist and his confirmation of the pulmonary findings. In making a differential diagnosis, we have to rule out syphilis, carcinoma, chronic catarrhal laryngitis and rarely actinomycosis.

In treating tuberculosis of the throat we have to bear in mind that this condition is not in itself a true entity, but a complication usually secondary to pulmonary tuberculosis. The capable otolaryngologist recognizes this fact after he has made the diagnosis and solicits the aid and co-operation of a competent internist who assumes responsibility for the care of the chest condition.

The local treatment of the throat may be divided into medical and surgical. One type may be used with one individual and another type with another individual. This means that different patients respond to different forms of therapy and the success of the treatment depends upon the care and the technique provided.

Surgically, we are justified in removing extensive infiltration areas and curettement of tuberculous ulcers as advocated by Herking, Lake and Krause. This type of work can now be done by the electric cautery under local anesthesia with less trauma, less loss of blood and less shock to the patient. The cautery snare is used for amputation of the diseased portion of the epiglottis.

The purpose of the electric cautery is to produce an eschar rather than to try to eradicate all the tuberculous tissue. Following the eschar, granulation tissue produces new blood vessels and more nutrition to the affected part. This fact adds to the popularity of the electric cautery but it must be remembered that it has to be used with judgment and skill. It is considered better technique to do repeated cauterizations rather than produce too extensive eschares.

Vocal rest is one of the most important factors in curative treatment. This means absolute vocal rest, requiring the patient to use the pad and pencil.

Topical applications to the throat at the site of the lesion are best tolerated in an oily base. Many drugs have been tried and many have been discarded. Chaulmoogra oil, used first by Lukens in 1921 who made the statement that it was the best local treatment up to the present time. This oil is known to have relieved dysphagia after one application. It may be injected into the larynx or it may be applied by direct swab.

Formalin is also recommended for local treatment, beginning with three per cent, the strength may be increased up to fifty per cent as the patient's tolerance permits.

Lactic acid has been used but it is quite painful and its use has been curtailed.

A few drops of compound tincture of benzoin on loaf sugar allowed to dissolve in the mouth often relieves that dryness and tickling sensation in the throat which is at times quite annoying.

The pain experienced by swallowing may be so severe that the patient prefers starvation to the excruciating difficulty of taking nourishment. This demands specific treatment in order to preserve the general condition of the patient. Cocain, metycaine, procaine, or butyn sprayed into the throat just before eating allows the patient to overcome this painful swallowing. Wigglesworth, in the Veterans' Hospital, uses orthoform and anestheseine, equal parts insufflated into the throat with good results.

Mild inhalations of camphor and menthol and compound tincture of benzoin, give relief in some cases.

Blocking of the internal branch of the superior laryngeal nerve is perhaps the most effective means in controlling painful dysphagia. This idea was originated by Hoffman in 1908. It consists of an injection of eighty-five per cent alcohol into the nerve as it leaves the thyrohyoid foramen in the thyroid cartilage. This is a safe procedure and gives relief for several weeks. Medically we depend upon the application of a topical anesthetic for dysphagia or if necessary, upon the injection of the laryngeal nerve with alcohol. Vocal rest is essential.

Surgically, the use of the electro cautery with the production of an eschar in the diseased tissue, is the accepted method.

CASE NUMBER ONE.

P. M. White adult male, age thirty-four; markedly emaciated; height five feet, eight inches; weight one hundred ten pounds.

Chief Complaint: Hoarseness; difficulty in swallowing; loss of weight (twenty-one pounds in past month); onset sudden, cold and pharyngitis following exposure while working.

Past History: One of the twelve children, all of them alive except one who died from a skull fracture. Parents living. No family history of tuberculosis.

Physical Examination: Poorly developed, poorly nourished, anemic adult male. Eyes sunken, face and neck wrinkled. Tongue presents a geographic appearance. Pharynx congested and pillars inflamed. Epiglottis shows ulcerated area involving the tip and extending to the base in the mid-line. Swollen edematous finger-like flaps remain in the lateral border. This whole area is reddened and swollen. Arytenoids are red and edematous. Cords hard to see due to swelling of the arytenoids, but they seem to be normal. Chest: No rales heard.

X-ray: Extensive active pulmonary tuberculosis both lungs.

Follow-up: When last seen about two weeks later his condition was about as described above.

CASE NUMBER TWO.

J. M. white adult male, age 40 years; weight one hundred forty pounds.

Chief Complaint: Hoarseness past two months. Pain in throat. Difficulty in swallowing. Loss of weight (two pounds in past two months).

Past History: One of six children, three of whom died in infancy, of cause unknown. No family history of tuberculosis. Patient is the father of two children in good health.

Physical Examination: Patient is a fairly well developed adult male. Pharynx red. Tongue coated. Epiglottis congested and indurated. Arytenoid swollen and edematous. Cords appear normal except for slight limitation of motion. Chest is negative clinically.

X-ray Report: Extensive infiltration upper half both lungs, probably active. Pulmonary tuberculosis.

Follow up: Patient last seen about two weeks later. Condition about the same.

CASE NUMBER THREE.

F. G., coloured adult female, age twenty-five; weight ninety pounds; height four feet, eleven inches.

Chief Complaint: Pain in throat. Anorexia. Hoarseness upon getting tired. General malaise. Spitting up blood.

Family History: One brother at home now with active tuberculosis. Mother living and well. Father killed in an accident.

Past History: Patient slept with a sister who died of tuberculosis. Came to clinic and was treated for above symptoms. Weight at that time was seventy-eight pounds.

Physical Examination: Poorly nourished adult female. Teeth good condition. Pharynx red. Post-nasal discharge. Arytenoids swollen and red. Epiglottis fixed and pale. Cords pale.

X-ray Report: Early pulmonary tuberculosis both lungs. Most marked in the right.

Follow-up: Patient seen four weeks later in clinic and she looked fine. Weight ninety-four pounds. Appetite good. No throat symptoms. No hoarseness.

CASE NUMBER FOUR.

A. O., white adult male, markedly emaciated, age sixty six; weight one hundred seven pounds.

Chief Complaint: Hoarseness. Chronic recurrent sore throat. Loss of weight. Anorexia.

Past History: Usual childhood diseases. No history of tuberculosis in family, but was treated about a year ago for tuberculosis and was told he had no active condition.

Physical Examination: Anemic, emaciated adult male. Eyes dull and sunken. Nasal septum deflected to right, Pharynx red, vessels prominent. Epiglottis entirely missing, leaving an ulcerated area at base of the tongue. Arytenoids red and swollen. Cords appear normal in appearance and function. Chest sunken and ribs prominent. Expansion limited. No rales heard.

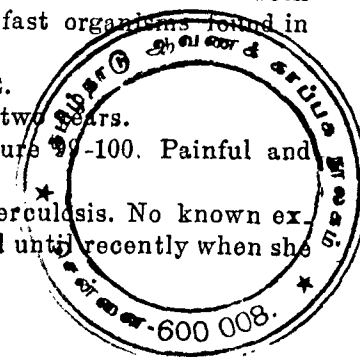
X-ray Report: Fibrosis both lungs but no evidence of active disease. X-ray report from another hospital showed fibrosis in both apices and through both lungs, in 1936. Acid fast organisms found in the sputum at this time.

CASE NUMBER FIVE.

A. M. K., white adult female, age thirty-two years.

Chief Complaint: Hoarseness. Temperature 99-100. Painful and difficult swallowing. No loss of weight.

Past History: No family history of tuberculosis. No known exposure to tuberculosis. Has always been well until recently when she



had a sore throat with lots of pain upon swallowing. Delivered her third baby eight days before admission to hospital. Her condition has been progressively worse since delivery.

Physical Examination: Patient is a poorly developed adult female suffering with severe sore throat. Complexion pale and she is anemic. Heart: No enlargement or no murmurs. Lungs clear, no rales heard. Tongue coated. Uvula, both anterior pillars, pharyngeal tonsils and posterior border of veli palatini covered with greyish white confluent adherent membrane. Epiglottic tip also shows similar type of membrane. Unable to see cords due to extreme pain to the patient. Biopsy from anterior pillar showed tuberculoma.

X-ray Report: Extensive involvement both lungs of pulmonary tuberculosis.

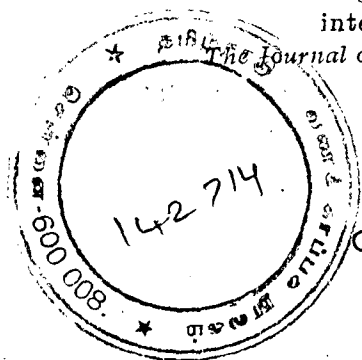
Follow-up: Patient died about four weeks after the childbirth.

CONCLUSION

In conclusion may I mention these ideas which are referable to tuberculosis of the throat:

1. Laryngeal tuberculosis should be diagnosed early in order to get results.
2. Laryngeal tuberculosis is nearly always secondary to pulmonary tuberculosis.
3. Laryngeal tuberculosis occurs rarely in children and most often is found between the ages of twenty and forty-five, and is more common in men than in women.
4. Tuberculous throat lesions are often precipitated by pregnancy.
5. Tuberculosis of the throat can be cured, but only by early diagnosis and by the co-operation of the patient, the internist and the laryngologist.

The Journal of the Oklahoma State Medical Association, Sep. 1937.



ORTHOPEDIC MANAGEMENT OF ANTERIOR POLIOMYELITIS.

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The season is far enough advanced to tell us definitely that we are in the presence of a vastly greater number of cases of infantile paralysis than can be accounted for by ordinary season incidence. There were almost three times as many cases reported in July 1937, as have been reported in any previous month in Oklahoma; substantially more, in fact, than the total reported for any preceding year. While this is not cause for mass hysteria, it behooves us to take stock

of the situation, in light of our past experience, so that we may be better prepared to handle this vast increase in cases. Our attack on infantile paralysis must be in three major fields: first, preventive; second, treatment and cure of the acute disease; third, management of the paralysis.

In regard to prevention, I think it is fair to say that, so far, we have no definitely proven preventive measure which has stood the test of time. In the second phase; namely, the acute disease, there is no specific treatment either to check the disease or prevent paralysis. There are many measures which may have some value. Suffice it to say that results have been very disappointing.

We have not, therefore, made any very substantial progress in treatment, prior to the paralytic stage. Management in the third stage presents an altogether different story. There is no longer room for doubt of the fact that careful, conscientious, and adequate treatment of the paralyzed muscles will result in minimizing the residual paralysis. Since it is in this field that such extensive progress has been made, at the risk of repetition, let me proceed to recapitulate the orthopedic management of this disease.

It is possible to divide this management into three rather poorly delineated sections.

First: The period from the onset of the paralysis to the subsidence of the acute symptoms in the muscles themselves. We have rather arbitrarily set this period as about six weeks from the onset of the disease.

Secondly: The period which covers the time during which active recovery of function of paralyzed or weakened muscles is obtained. Again, arbitrarily, we may say that this period will run from six weeks to about eighteen months, the major portion of recovery occurring in the early months.

Third: The period covering the time of rehabilitation by surgical procedures; such as stabilization, tendon transplants, and other similar methods used to provide substitutes for permanently paralyzed muscle groups; roughly, from eighteen months after the acute disease *ad infinitum*.

Our interest must centre in the first two sections, and more primarily in the first; namely, the period of acute muscular involvement. During this stage we have one aim only. This is to provide complete rest of the paralyzed part, with the extremity immobilized in its most functional position. The immobilization must begin with the onset of the paralysis and must continue until all muscular tenderness has disappeared. Thus, with the onset of the disease, the child should be put at absolute bed rest, regardless of the extent of the paralysis. Of course, during the early stage, the child will probably be sick enough to remain in bed. However, it is not sufficient to keep him in bed only during this acute febrile stage. Involvement

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of the abdominal muscles and muscles of the back is very common, and bed rest is essential in order to prevent over use of these muscle groups. All activity should be discouraged as long as there is any pain or tenderness demonstrable in the muscles. Our general aim, therefore, is rest of the entire organism with specific rest and support to the involved muscle groups. The involved extremity should be supported in the position of election, preferably by light, well-fitted plaster casts. Plaster casts have the advantage of fitting the extremity to which they are applied, and of being fool-proof, so that it is not feasible for the parents to remove the support in order to examine the extremity, move the joints, bathe the skin, etc., a tendency which is well nigh impossible to overcome. If casts are not available, supportive splint are entirely adequate, provided that it be emphasized to the parents, or others in charge of the case, that it is not permissible to remove the splints for any reason, other than to secure more proper fitting.

The cast or splint should be carefully fitted to give support in the correct position. In the lower extremity, the foot is placed at 90°, the knee slightly flexed, the hip slightly abducted and in neutral rotation. If the splint is for the leg only, and does not include the hip, some attachment must be applied to prevent rotation of the leg at the hip or knee. In the upper extremity, where deltoid paralysis is particularly common, the arm should be at 90° with the body, the elbow at 90° and the wrist in a slightly cock-up position. Obviously it is much better to provide well-fitting casts or braces. However, if this is not feasible, the more nearly any support fulfills the foregoing requirements, the more adequate it will be.

During this initial stage, there is absolutely no value, and there is undoubted harm in middlesome treatment of the extremities. *There is absolutely no place for massage, manipulation, or exercise in the early management of infantile paralysis. The only proper treatment in this early stage is rest.* By this is meant, rest of the paralyzed muscle, rest of the body, and rest of the mind. No other regime will give satisfactory results. The question arises as to the point of termination of the period of absolute rest. If an error is to be made, let it be in maintaining the rest over too long a period, rather than cutting this period short. An arbitrary limit of six weeks has been set. This, of course, is a definite figure for a variable period. The exact time can best be estimated by the condition of each individual case. Thus in the last week of the initial period, the plaster splints may be removed on occasion and the extremity examined. If there is the slightest muscular tenderness, immobilization must be maintained. When the tenderness has disappeared, we may gradually slip into the second stage of treatment, at which time more active management is desired.

As the transition occurs from the initial stage to the secondary stage, the time arrives for careful initiation of massage, manipulation, and exercise, particularly muscular training. *Let me emphasize again, that the utmost care must be used in order to prevent overactivity.* At this stage, the child is inordinately tired of his long recumbency and may easily become over-enthusiastic. The parents are anxious to see immediate results and oftentimes encourage the child perform beyond his strength. The only safe criterion at this stage is fatigue. *Treatment must never be carried to the point of over-fatigue of the child.* Thus, in the ordinary case, support may be removed for a little gentle massage and careful motion, great pains being taken to support the extremity and to protect against the pulling effect of gravity. Weight bearing must be absolutely prohibited, if the lower extremities have been at all involved, or if there is major weakness in the abdomen or back. A safe policy is to prohibit weight bearing entirely for six months following the onset of the disease. The six months' period must be spent in conscientious effort to provide gradually increasing exercise for the weakened muscles. It must be emphasized again that throughout this entire period of six months, the extremities must be supported by continuation of apparatus for a very substantial period during the twenty-four hours. Each case must be sharply individualized in this respect.

Another major crisis arises with the beginning of weight bearing. Here again, it is possible, by a few hours of over-fatigue, to undo the labor of many weeks. If there is major weakness, support must be provided by braces. This is particularly true in the lower extremities, back, and abdomen. The least tendency toward deformity must be anticipated and corrected by supportive apparatus. Particular points to guard against are, deformity of the feet, hyper-extension of the knees, and lordosis of the spine, with protuberant abdomen. Properly fitted braces will be of great help in preventing these deformities from becoming crippling.

It is, in my opinion, extremely important to keep a very accurate chart of the extent of impairment of various muscle groups. I may hasten to add that no effort should be made in this direction during the acute stage of the disease. With the removal of the initial support, however, and as some exercise is undertaken, it will be possible to secure the co-operation of the child and make a careful outline of the extent of involvement at that time. It is possible to secure charts for this purpose which list each separate muscle group. On successive dates, a careful recheck should be made in order to determine progress of recovery. Under ordinary circumstances, there should be gradual improvement in the strength of the muscle groups. Any definite retrogression is a possible indication that the case is being overtreated.

It is, of course, impossible, in this brief discussion, to give in detail the various methods of physiotherapy. A few words may be in order in regard to the more publicized treatments. Hydrotherapy is of extreme advantage in the subacute stage of the disease. It is definitely contra-indicated during the acute stage; namely, the first six weeks. There is, of course, no specific therapeutic value of the water, its only function being supportative, largely in permitting active motion of the extremities which are too weak to overcome the pull of gravity. It is an adjunct to treatment.

In conclusion, allow me to quote a paragraph from the pamphlet printed by the *Oklahoma Commission of Crippled Children*. 'In checking the progress of treatment of infantile paralysis in the past ten years, it appears that there has been tremendous progress in management. Statistics from older epidemics reveal the fact that the greatest majority of cases developing paralysis had definite residual paralysis at the termination of treatment. In more recent series, it appears that the number of cases with residual paralysis has been sharply curtailed. This has been almost entirely due to more conscientious and intelligent handling of the paralyzed muscles. There is little, indeed, to offer in the treatment of the acute febrile disease. There is much to offer in the management of the case as the acute symptoms subside. It is in this field of endeavour that efforts must be concentrated, until such time as something specific has been ordered which will check the paralysis at its incipency. Careful management will permit temporarily paralyzed muscles to regain their function and will prevent development of deformities in those extremities which have permanent paralysis.'

—*The Journal of the Oklahoma State Medical Association, Sept. 1937.*

DIAGNOSIS OF ACUTE APPENDICITIS IN CHILDREN.

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Chicago.

Abdominal pain and distress together with gastrointestinal symptoms are extremely common in infants and children. Although the incidence of acute appendicitis is comparatively low the possibility of the latter should always receive serious consideration. The seriousness of acute appendicitis may be said to be inversely proportionate to the age of the child. In the younger ones, perforation and peritonitis develop rapidly.

The diagnosis of acute appendicitis is one of the most difficult and important problems encountered in the practice of medicine among children.

Statistical studies show that 50 per cent of all cases of appendicitis occur during the first twenty years of life, and of these, 2.5 per cent occur in the first 5 years, 6 per cent between 6 and 10 years, and 16.5 per cent between 10 and 15 years, and the remaining 25 per cent between 15 and 20 years.

There seems to be a slight seasonal variation with the increase occurring during the summer months, due to the prevalence of gastroenteritis at this time.

The symptoms and signs in the first 5 year period differ distinctly from those in the older groups and will be considered separately. This group is also deserving of special mention because of the high mortality, being eight to ten times greater than that in the 5 to 15 year group, and is due chiefly to the delay in diagnosis and the rapidity with which peritonitis develops. In a large series, 90 per cent were found to have peritoneal involvement at operation. Co-operation and information are difficult to obtain in the younger child and the physical findings are indefinite until peritoneal irritation and perforation occur.

Generalized symptoms and behavior are more diagnostic than local findings. Severe illnesses in infants are not associated with restlessness, and when a child is restless, unable to sleep, cries a great deal, exhibits a pallor and assumes a typical posture while lying in bed with the right leg flexed on the abdomen, and has increased costal type of breathing, one may well consider appendicitis as the cause. Vomiting is quite a constant symptom and occurs soon after the onset of pain. The bowels may be normal or constipated. The temperature is rarely elevated higher than 101 degrees, and the leucocyte count, which may be of diagnostic importance, rarely exceeds 20,000.

In the differential diagnosis, one may consider intussusception, but the distinct suddenness of onset of pain, the shock as evidenced by pallor and clammy perspiration, together with the relaxed abdomen and the findings of a sausage like tumor and the current jelly like bloody mucus obtained on rectal examination, will, in most cases, establish this diagnosis.

Acute mesenteric lymphadenitis cannot be accurately differentiated from acute appendicitis although the tenderness is usually less than the pain experienced by the patient. The process may or may not involve the appendix and it may be well to make the diagnosis of appendicitis in lymphadenitis rather than to consider a lymphadenitis as such and later find signs of a ruptured appendix.

In certain cases of hypertrophic pyloric stenosis the onset may be rather sudden. The projectile type of vomiting together with definite gastric peristaltic waves and the occurrence of a pyloric tumor should however establish this diagnosis.

Primary peritonitis is a rather rare condition which may develop from rupture of the mesenteric lymph nodes or by direct passage of the inflammatory products through the intestinal wall. In this condition, the child appears extremely ill, the abdomen is distended and rigid, and signs of general intoxication are present. The leucocyte count is frequently very high, reaching 75,000 at times.

Colic is perhaps, the most common abdominal pain in infancy. It is paroxysmal in character, recurs from day to day, pressure and massage seems to decrease the pain and complete relief is usually obtained when the bowels are evacuated together with the passage of flatus. This together with the absence of fever establishes the diagnosis.

In the older children, the group of 5 to 15 years of age, the symptoms and signs of acute appendicitis are quite similar to those encountered in adults. I believe it is worth while to restate and emphasize the sequence of symptoms. Pain is the initial symptom in an acute attack of appendicitis, later followed by vomiting but practically never preceded by it. The pain in the early stages is colicky in nature and usually referred to the region of the umbilicus and upper portion of the abdomen. This pain may be explained by the obstruction and tension within the lumen of the appendix and is transmitted through the sympathetic nervous system. Later, with peritoneal involvement the pain localizes in the right lower quadrant and becomes quite different in character. It is usually less severe but constant. This pain is due to parietal involvement of the peritoneum and the impulses are transmitted over the afferent branches of the cerebrospinal tracts. Local findings are absent when the appendix is retrocecal or retroiliac until the process is well advanced because these parietal spinal nerves are protected by the cecum and ileum. Similarly localization is absent when the appendix lies in the pelvis because of the lack of peripheral nerves to those segments of the abdominal wall.

Therefore, it is evident that the early pain of appendicitis is quite different from the later one as to character, cause of production and nerve innervation. The later pain is due to parietal peritoneal inflammatory reaction and is transmitted over the cerebrospinal tracts. Early in appendicitis we have the pain of obstruction later followed by that of infection.

With these facts in mind, one proceeds to examine the child for the presence of localized tenderness, muscle spasm and rigidity. By gradually gaining the confidence of the little patient and using gentleness during the procedure, one is able to elicit the true findings. The examining hand should be warm and all parts of the abdomen and lower part of the chest carefully palpated before any attempt is made to detect tenderness or rigidity in the right lower quadrant. Before attempting deep palpation, one may often elicit a

hyperesthesia of the skin of the right lower quadrant. Muscle spasm and rigidity of the right rectus may be present over its entire length up to the costal margin.

The finding of a definite point of tenderness is, to my mind, of great diagnostic importance. We must keep in mind that the appendix may occupy a number of different positions in the abdomen. In case of insufficient descent of the cecum, the tenderness is found to be high. Where rotation has not been complete, that is in the retrocecal or retroiliac type—this point of tenderness may be in the flank or in the region of the right kidney. Not uncommonly, due to exceptional length of the appendix in childhood together with a long mesocecum, it is often found below the brim of the pelvis. On this account, the point of tenderness may be at McBurney's point—in the right flank—or may not be elicited at all unless a careful rectal examination is made when one may find a decided difference between the right and left sides. Pain may often be elicited in cases where the appendix is situated posteriorly, by applying gentle pressure through the abdominal wall toward the psoas muscle and at the same time supporting the right leg. By suddenly withdrawing support to the leg and thereby causing the psoas to contract against the inflamed appendix, definite pain may be produced.

The vomiting in acute appendicitis is as a rule not persistent or forceful, although cases are met with where the vomiting assumes alarming proportions.

Anorexia is usually present even to the degree of aversion to food. I think we may say that we rarely or never see a child with acute appendicitis complaining of hunger or expressing desire for food.

Careful observance of the posture and general behavior of the child often gives valuable information. The child may not appear acutely ill. The color may be normal or there may be a pallor to the face due to nausea. Restlessness may be due to distention of the appendix or to pain. Respiration is definitely costal in character rather than abdominal and the patient assumes a position in bed which will insure maximum relaxation of the abdominal muscles as evidenced by flexing of the leg on the abdomen. This protective attitude can often be demonstrated if the child is carefully assisted out of bed and asked to take a few steps. He will then favour the right side by keeping the right leg flexed at the hip and knee and walk with a considerable limp.

In the pelvic type of appendicitis, rectal examination is of particular value. The left side should be examined first, to obtain normal feel, then the right side. Tenderness, tension or a mass may often in this way be elicited. In such cases, inward rotation of the flexed right thigh will produce a pain (the s.c. "obturator" sign).

Chills are rarely if ever present at the onset of appendicitis. The temperature is usually low, 99 to 100 degrees, and rarely

exceeding 101 degrees. A differential leucocyte count may or may not be indicative. The total count rarely exceeds 20,000 but the polymorphonuclears may exceed 80 per cent. Of course, it is a well known fact that a drop even in a moderate leukocytosis may indicate perforation and a "silent" period until peritonitis sets in.

Diarrhea is encountered in certain types, particularly in those where the appendix is retrocecal and the inflammatory process extends to the cecum and adjacent ileum. On the other hand, appendicitis is occasionally seen as a complicating disease during or following severe infectious enteritis. Similarly dysuria or hematuria may be present in cases where the appendix is in close proximity to the ureter or bladder.

The differential diagnosis of acute appendicitis in children involves many more problems than in adults. Many conditions occur in children which may be mistaken for appendicitis.

Pneumonia with or without pleurisy often presents symptoms suggesting acute appendicitis; 25 patients out of 145 cases of pneumonia admitted to Boston City Hospital were sent there under diagnosis of acute appendicitis.

The general appearance of the child with pneumonia is rather striking and different from that of acute appendicitis. "The child appears sick, face is usually flushed, increased grunting respiration, temperature is usually above 101 to 104, leukocytosis is high, frequently above 20,000, the respiration is abdominal in type, the respiratory excursion of the abdomen is greater than that of the chest."

The child with appendicitis is usually pale, lays quiet in bed assuming a posture to insure abdominal relaxation, - the temperature is low, leukocyte count usually below 20,000 the respiration is costal in type; the costal respiratory excursions greater than the abdominal. The value of comparing the costal and abdominal respiratory excursion has been demonstrated by the work of McClure.

In border line cases and particularly in the early stages of central pneumonia the x ray findings of the lungs are of great importance.

Abdominal pain is a frequent complaint in many forms of sore throats, particularly so in pharyngitis of s.c. grippy infection. The pain is colicky in nature; may be mild or severe. All parts of abdomen seem to be tender but the pain is usually greater than the tenderness. That is the reverse of appendicitis. The differential diagnosis may be exceedingly difficult to make, particularly as we frequently see acute appendicitis associated with or following upper respiratory infections.

Symptoms of appendicitis often occur in the acute infectious diseases. Measles, particularly in its early stage, may present such a picture. Here careful search for Koplik's spots together with

leukopenia establishes the diagnosis. In this connection I would like to quote from Ronaldson, who made a recent study at three of the large contagious hospitals in London: "The appendix is frequently involved in the pathological processes which are met with in the acute infectious diseases; but suppurative appendicitis is rare; appendicular symptoms are less common than might be expected, and localizations are exceptionally rare."

Pyelitis may simulate retrocecal appendicitis. Temperature is higher than general condition would indicate. Repeated urine examinations must be made and the amount of pyuria taken into consideration.

Summary: Acute appendicitis is rather rare in infants and children under 4 years of age.

Any young child with restlessness, abdominal pain, vomiting and low-grade temperature must be considered a potential case of appendicitis.

The pain in appendicitis is first of obstruction and tension within the lumen of the appendix and is registered through the sympathetic pathways. This pain does not shift nor change into the second pain. This later pain is of independent origin and is transmitted over afferent branches of the cerebrospinal tracts.

General postural characteristics, certain methods of examination particular to children, are of importance in the diagnosis.

In early diagnosis and the avoidance of cathartics lays our hope of decreasing the mortality of appendicitis in children.

—*Illinois Medical Journal Sept. 1937.*

Medical News & Notes.

THE DR. B. S. SHROFF MEMORIAL GOLD MEDAL OF THE BOMBAY MEDICAL UNION.—The following subject has been selected by the Bombay Medical Union for competitive thesis for the above Prize for 1938:—

"Tuberculosis in general, paying special attention to its prevention and treatment."

The award will be in the form of a Gold Medal called the Dr. B. S. Shroff Memorial Gold Medal of the Bombay Medical Union.

The competitor must be (i) a duly qualified member of the Medical Profession holding a degree or degrees and diplomas from Indian and other Universities created by statute, or (ii) a duly qualified member of the Medical Profession holding the diploma of Membership of College of Physicians and Surgeons of Bombay.

The thesis must be sent in six typed copies so as to reach the Honorary Secretaries Bombay Medical Union, Blavatsky Lodge Building, French Bridge, Chowpatty, Bombay, on or before the 15th August 1938.

The thesis should be designated by a motto instead of the writer's name and should be accompanied by a sealed cover containing the name of the writer and his Post-Office Address.

The name of the Prize, the year of competition, the subject of the thesis and the writer's motto should be subscribed on the cover.

No study or essay that has been published in the medical press or elsewhere will be considered eligible for the Prize, and no contribution offered in one year will be accepted in any subsequent year unless it includes evidence of further work.

The accepted thesis shall be the property of the Bombay Medical Union.

All other thesis shall be returned if not accepted provided the return postage expenses are paid in advance by the writer.

In the award of the Prize to the successful candidate, the decision of the Committee shall be final.

SORAB J. POPAT, M.B., B.S.,
M. B. THAKORE, M.B., F.R.C.S.,
Hon. Secretaries.

Bombay, 15th February 1938.

Bombay Medical Union.

THE KISTNA DISTRICT SUBSIDISED RURAL MEDICAL PRACTITIONERS' ASSOCIATION: A meeting of the above Association was held at Masulipatam in the District Headquarters' Hospital on Saturday 19-2-38. The members passed the following resolutions unanimously:

1. Requesting the Editor, Medical Practitioner, Madras not to increase the rate of subscription for his journal beyond Rs. 2-8-0 in cases of the Rural practitioners as per resolution No. 8 and 9 of the provincial R.M.P. conference of 10-7-37.

2. Protesting against the action of Kistna District Board who ousted the R.M.P., Pallevada (Kaikalur Taluq) simply because he was asked to undergo refresher course for a period of 3 months by the D. M. O. Kistna and requesting him to reinstate the practitioner immediately as his orders were not consistent with the terms of the agreement.

3. Requesting the Government to sanction an honorarium of Rs. 30 and an assistant vaccinator when desiring to enforce vaccination and public health work on the R.M.Ps.

4. Requesting the Government that the interests of the R.M.Ps. may be safe guarded when the G.O. No. 3066 p.h. of 15-11-37 is given effect to by the District Boards, since it is feared that the latter are likely to utilise the same to remove the Dispensaries on some political spite and that the opinion of the practitioner may be given due weight while proposing to transfer the dispensary.

5. Requesting that the District Boards may be made to supply raw Indian drugs to R.M.P's of Indian Medicine and thus enable them to manufacture genuine medicines since the drugs now supplied are found to be most ingenuine.

WEST GODAVARI DISTRICT MEDICAL ASSOCIATION: At a meeting of the Managing Committee of the above Medical Association on 27th Feb. 1938 in the Higher Elementary School Building at Tadepalligudem, it was decided that the Andhra Medical Conference should be held at Ellore on May 1, and that the West Godavari District Medical Association be the Reception Committee. It was also resolved that the President of the Conference should be invited from among the Doctors, the Hon. Dr. T. S. S. Rajan, Minister for Public Health, Dr. A. Lakshmanaswami Mudaliar, Superintendent, Maternity Hospital, Madras, and Dr. Muller, Superintendent Tuberculosis Sanatorium, Madanapalli.

The date of the anniversary of the District Association was fixed as the 30th April, 1938, and it was decided to request the Surgeon-General to preside.

CUDDAPAH DISTRICT MEDICAL ASSOCIATION: The monthly meeting of the Cuddapah District Medical Association was held on 19th Mar. '38 at the Government Headquarters Hospital, Cuddapah, under the presidency of Dr. T. S. Harinarayana Panthulu, District Medical Officer.

After light refreshments and tea, Dr. G. Jagannadha Rao read a paper on "Affections of Kidney in Children."

Dr. E. J. Koshi (Assistant District Medical Officer), Dr. Srinivasan (Rural Medical Practitioner, Anantharajupet), and some other members took part in the discussion.

CHINGLEPUT DISTRICT MEDICAL ASSOCIATION: A meeting of the District Medical Association was held on 19th Mar. '38 with Dr. K. Narayanaswami Aiyar, in the chair.

Dr. R. Varadarajan, Secretary, welcomed the President.

Dr. P. S. Srinivasan, M.L.C., Conjeevaram, was elected Vice-President and Dr. (Miss) C. Asirvatham of the Chingleput Government Hospital, was elected to the Committee.

Dr. R. Varadarajan moved a resolution that the Association condemned the perpetration of the class system in the honorary scheme of House Surgeons and Physicians.

Dr. Srinivasan moved that the classification of the honorary House Surgeons and Physicians be taken away from the scheme.

The first resolution being withdrawn, Dr. Srinivasan's resolution was passed.

Dr. P. S. Srinivasan proposed the vote of thanks.

Dr. T. Satakopan, M.D., Honorary Physician, General Hospital, Madras, addressed the members of the Association on "Certain Types of abdominal pain."

The medical practitioners of the town then entertained the guests at a social.

BEZWADA DISTRICT MEDICAL ASSOCIATION: The tenth anniversary of the Bezwada Medical Association was celebrated on 26th Feb. '38 evening in the local Museum Hall under the presidentship of Dr. Ch. S. John, M B B S., E. N. T. Diplomate (Vienna).

The proceedings commenced with the presentation of the Secretary's report by Dr. K. S. Sambasiva Rao and of the Treasurer's report by Dr. J. Dakshinamurti.

Dr. John, in the course of his address, congratulated the enterprising spirit of the Andhra Pharmaceutical Works, the X-Ray Institute and the Diagnostic Clinic and Laboratory, which were an asset to the medical profession at Bezwada. Dr. John suggested that a surgical clinic, a nursing home and a medical library be started which were long overdue in the city.

Dr. H. C. Halge then read an interesting paper on 'Acute Abdomen.'

Dr. D. S. Ramachandra Rao, President of the Association, hoped that the honorary system now to be adopted by the present Government would give ample scope to the new recruits.

Dr. T. V. S. Chalapathi Rao then spoke on the importance of the Diagnostic Clinic and Laboratory.

The Bezwada Medical Stores entertained the doctors at a dinner party in the Museum Hall.

CHITTOOR DISTRICT MEDICAL ASSOCIATION: A meeting of the District Medical Association was held on 26th Feb. '38 at Chittoor under the presidency of Dr. D. Krishniah, District Medical Officer.

Dr. G. Srinivasa Aiyar read a paper on the deficiency of vitamin A with reference to the nature and distribution of the cutaneous manifestations.

With the President's concluding remarks the meeting terminated.

Review.

A MODERN LABORATORY IN BOMBAY.

It was the intention of the head of this enterprise, known as the Chemical, Industrial and Pharmaceutical Laboratories Ltd., (or 'Cipla') to found an industry capable of expanding until it shall assume national importance. Consequently the 'Cipla' Laboratories is carrying out intensive research in addition to introducing to the Indian public a range of medicines, drugs and serums of as high a

quality as those produced by the foremost overseas laboratories. The laboratory was inaugurated in April 1937.

All the equipment of the laboratory was obtained from Germany and the arrangement and working of the various departments were carried out personally by the Managing Director of 'Cipla', Dr. K. A. Hamied, B.Sc., M.A., Ph. D (Berlin) A.I.C., F.C.S., (London) and by the Technical Director Herr C. A. Rothenheim, a highly qualified Pharmacist and apothecary from Germany, who has had experience in a big German Hospital of the most up-to-date manufacturing methods for the production of medicines, drugs and serums of all descriptions. The laboratories is situated in Bellasis Road and a high degree of care is taken to protect the same from possible contamination by dust by watering the garden every now and then. The 'Cipla' laboratories consists of many departments, namely, Main Laboratory, Testing Laboratory, Ampule filling room, Sterilizing room, Physical test room, Fuming Chamber, Tablet making laboratory Tube filling plant and a Vacuum plant which is the largest of its kind in India. They have put into the Medical Market several products. When I went round the place I was very much pleased at the high efficient working of the different departments and I wish the medical public to patronise the laboratory products. The prices are cheaper than foreign imported goods. I congratulate Dr. K. A. Hamied and Mr. C. A. Rothenheim and wish the laboratories every success.

THERAPEUTIC NOTES.

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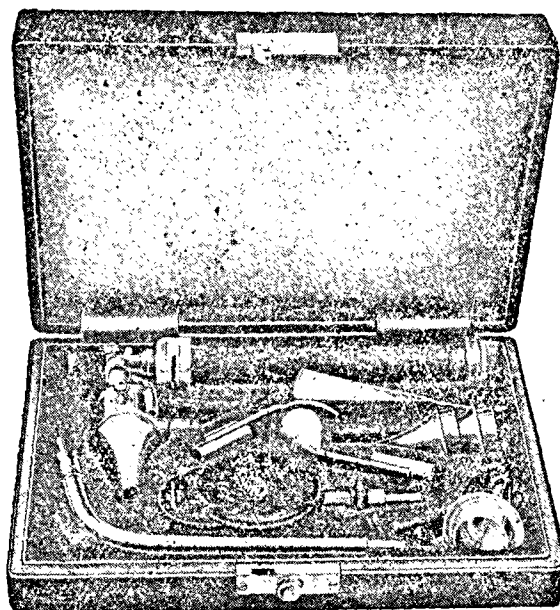
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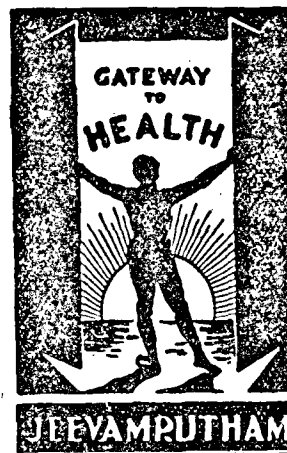
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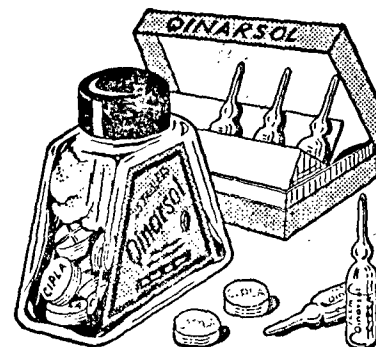
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