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Cystometric studies in neurogenic bladders

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CYSTOMETRIC STUDIES

IN

NEUROGENIC BLADDERS

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PART I ---- PHYSIOLOGY

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Before entering into a discussion of those pathological conditions, especially those of neurogenic origin, which alter the normal function of the urinary bladder, it will be necessary to discuss the present conception of the physiology of the bladder in its normal state.

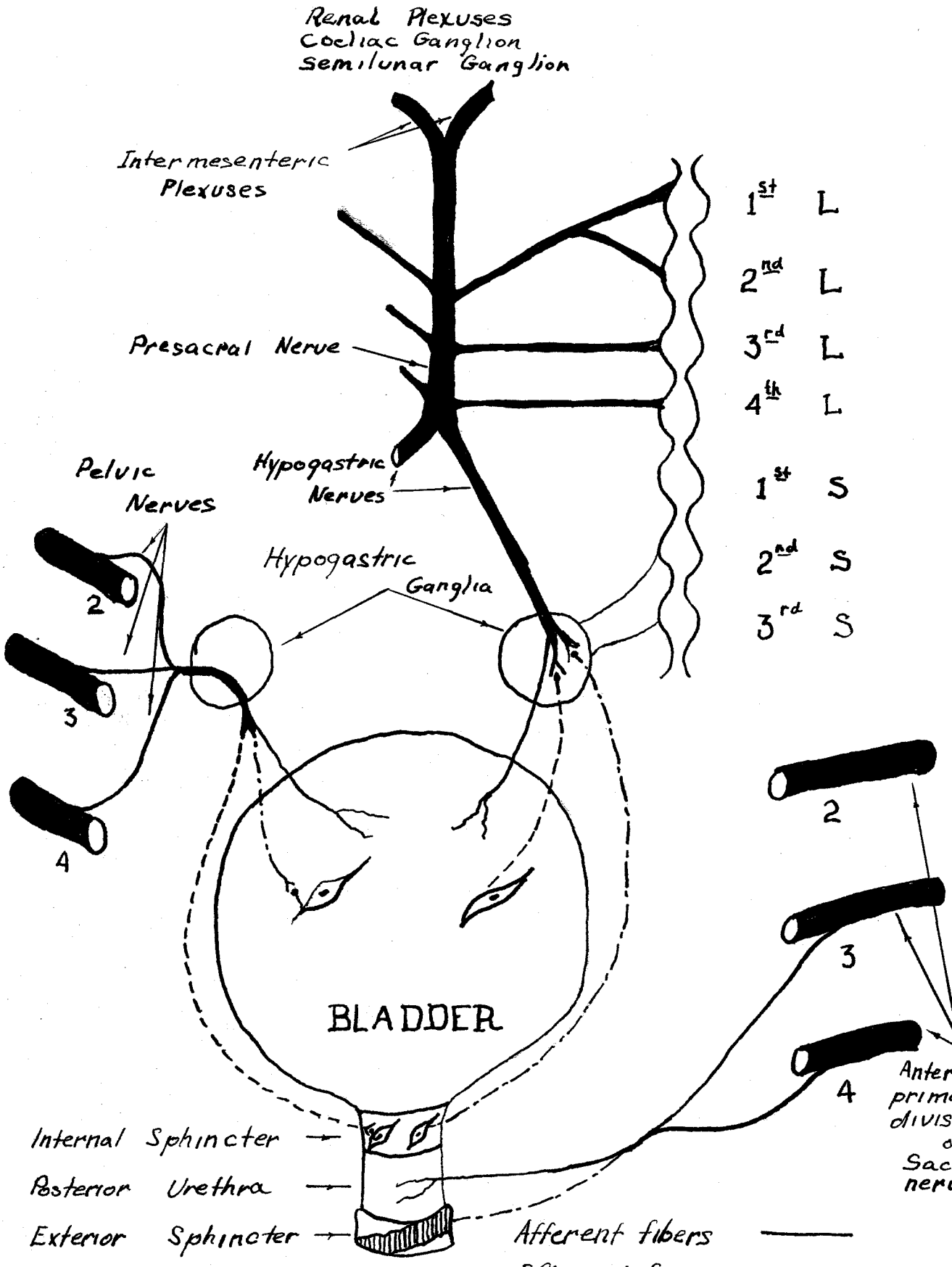
The physiology of the bladder has long been the subject of much speculation and controversy on the part of various investigators since it is, paradoxically, voluntary and involuntary.

Since a knowledge of the anatomy of the viscus is necessary, before undertaking a discussion of function, we shall review, briefly, the various nerve pathways concerned in bladder mechanics.

The efferent nerves to the bladder, just as in the heart, stomach, and intestines, are of two types, sympathetic and parasympathetic. (1).

The sympathetic portion is represented by the hypogastric nerves which arise from a common trunk on the promontory of the sacrum, the pre-sacral nerve. (fig. 1). This presacral nerve receives medullated fibers from lumbar ganglia 1,2,3,4, comprising about 60% of the nerve. Non-medullated fibers from the intermesenteric plexus make up the other 40%. The spinal origin of the intermesenteric plexus is unknown, but its fibers have been traced to the renal plexuses, the caeliac ganglion, and the semi-lunar ganglion. Thus the nerves from 1,2,3,4, form the lateral roots, and the juncture of the right and left intermesenteric plexuses, the middle root of the pre-sacral nerve. The hypogastric nerves end in their respective hypogastric ganglia from whence their post-ganglionic fibers run to end in the bladder wall.

Fig. I Diagram of Bladder Innervation



The parasympathetic supply arises from the anterior primary divisions of S 2,3. These nerves also run to the hypogastric ganglia, but pass through without synopsing to end in the bladder wall. The motor supply of the external sphincter is the pudic nerve from S 3,4, which runs to the viscus without passing thru the ganglia. (2).

These various nerve elements are connected with the mural nervous system of the bladder, and are seen best at the point of entrance of the ureters into the bladder. When the tissue at this point is sectioned and stained by the silver method of Bielchowsky, the cells are seen clearly as multipolar with their processes extending in various directions over the bladder. These ganglion cells and connecting neurones are found both in the muscle tissue and in the external connective tissue layers of the entire bladder wall. The vesical plexus receives (a) medullated fibers from the sacral nerves, the nerve erigentes of Eckhard, (b) non-medullated and medullated fibers from the hypogastric nerves, and (c) fibers from the sacral portion of the marginal fasciculus. (3) (Fig. 1).

That there are spinal pathways connecting the vesical plexus with the higher centers in the brain is conceded by all the investigators of this field. (2-3-4-5-6).

Langworthy and Kolb, from their experiments on cats, believe this center to be situated in the cephalic portion of the hind brain. Their experiments confirm the work of Barrington. (7-8).

The exact part played by the various nerve pathways in the normal function of the bladder is still a matter of some controversy.

Perhaps the simplest theory of innervation was advanced by Muller. He believed that in the bladder, as in the intestines, one dealt with the law of "crossed innervation." According to this law,

in man it does so after a variable period of time, providing the section is above the lumbo-sacral segments. (4-5).

Elliott in 1907, published a paper in which he stated, "The main control of the bladder is by the vesical plexus -- from the sacral nerves. No good proof exists that the hypogastric plays an inhibitory rale to the detrusor." (10).

Denny-Brown is of the same opinion and in a recent article, advanced the idea that the "micturation center" is not in the brain as Langworthy and kolb believed, but in the lower segments of the spinal cord, namely T 12, L 1, L 2, S 2, S 3. The function of the higher centers are only to decide whether or not micturation would be suitable to the environment. However, he found that stimulation of the hypogastrics resulted in contraction of the internal sphincter, and acknowledges the accepted theory of the motor function of the parasympathetics. He believes that the essential mechanism is the vesical plexus. This plexus develops into an automatic functioning unit after section of both sympathetic and parasympathetic roots. The circumstances which decides its development is the reaction of the bladder to the stretching caused by an increasing volume of contents. (4).

Learmouth, during a lumbar sympathectomy on one of his patients, applied farodic stimulation to the distil cut ends of the hypogastrics and by observing the bladder through a cystoscope, found that the internal sphincter and trigonal region contracted but the bladder wall underwent no demonstrable changes. MacDonald, however, reports a case in which stimulation of the hypogastrics caused a definite contraction of the detrusor. Elliott made a similar observation but on a patient in which the sacrals had been destroyed. He explained the phenomena by showing that in a case of that kind, the reflex bladder set

the nerve bundle which carried motor impulses to one set of muscles carried also inhibitory impulses to the corresponding set of antagonistic muscles. Thus, in the case of the bladder, motor impulses to the bladder wall would be accompanied by inhibitory impulses to the sphincters. He showed that stimulation of the pelvic nerves (parasympathetic) lead to a contraction of the detrusor and a relaxation of the sphincter. Also if the pelvic nerves were cut bilaterally, the tone of the detrusor decreased, and the sphincter would no longer open. The hypogastric nerves are the motor fibers to the sphincters and inhibitory fibers to the detrusor. However, section of the hypogastrics lead to no demonstrable difficulty in emptying the bladder, whereas, had their effect been that of antagonistic to the pelvic nerves, we should expect to get a contracted bladder, with atonic sphincters. (3).

Rose and his co-workers also believe in the reciprocal innervation of the bladder by the sympathetic and parasympathetic, and in addition, the nervus pudendus which controls the external sphincter. They consider that the sensory impulses from the bladder are carried by a variety of pathways. Thus the "First desire to void is carried by the pudic nerve to the cord and from there to the brain; but the feeling of distension is carried by the sympathetics to the higher centers." Rose considers cerebral influence to be one of inhibition to the detrusor muscle. (6,11,1).

Langworthy and Kolb, advance the theory that the center for controlling bladder tone is not in the lumbo-sacral segments, but rather in the brain. They hypothesis a "micturation center" for control of bladder function as a whole in the region of the cerebral motor cortex. (9).

Denny-Brown, however, denies the existence of this "micturation center" in man. As evidence, he points out the fact that the cat bladder does not, become reflex after section of the spinal cord, whereas

up by the vesical plexus was augmented by the hypogastrics due to an outgrowth of fibers to the decentralized motor ganglia. These findings, however, do not necessarily conflict with the original conception of hypogastric nerve function, since this nerve contains both motor and inhibitory fibers, and while only the inhibitory fibers, innervate the normal bladder wall, motor fibers may grow out to assist the vesical plexus in the bladder devoid of sacral control. (11, 2, 32).

Of the hypogastric nerves Denny-Brown says, "In any case, we do not have to bring the pre-sacral nerve into any scheme of essential nervous mechanism of micturation." He bases this statement on the observation that in many patients, lumbar sympathectomy does not result in frequency or incontinence. (4), (17).

We are now in a position to discuss the phenomena of micturation. Rose believes that the weight of accumulated urine within the viscus causes a mild involuntary bladder wall contraction, the resulting increased pressure being recognized in the posterior urethra. This impulse, or feeling of a "first desire to void" is carried via the pudic nerve to the higher conscious centers. If the conscious centers find micturation appropriate to the environment, the inhibitory influence of the cerebral cortex is removed and the perineum is voluntarily forced down to align the external and internal sphincters. (6).

This downward movement of the perineum was shown by Denny-Brown to be mainly the result of the relaxation of the levator ani muscle. (13)

The next step is the contraction of the trigonal muscle which bridges from the trigone to the posterior urethra. This movement forces down the internal sphincter to a level of the trigone and allows urine to run slowly into the posterior urethra. (6),(14),(15).

According to Rose, the urine running slowly through the urethra

causes an efferent stimulation which connects in the cord with the involuntary nervous system, stimulating the bladder to more forceful contractions. Thus the bladder wall becomes thicker so that the force becomes increasingly greater, until the bladder empties.

The contraction of the prostatic muscles, the bulbo-cavernosum, the perineii, and the levator ani brings about the terminal ejections of urine from the urethra, after which the bladder relaxes and the internal sphincter closes. Since the bladder wall is in constant rippling motion, that is, dilating and contracting, any external force which may be applied is taken up, and not transmitted to stimulate a forceful bladder contraction. (6).

It is important to note that Denny-Brown, Munro, and Robertson have a slightly different conception of bladder function. They have found that the reactionary contractions of the detrusor is produced by the distension of the bladder wall, and that these contractions are controlled by a restraining effect which is subconscious at low bladder levels. The process of adaptation intrudes upon consciousness only at increased volumes of urine within the viscus. They believe the sensation of a desire to void to be due to a process of adaptation, that is, a control of impulses going to the vesical wall, and have shown that a willed effort to micturate produces bladder contractions similar to those arising spontaneously. (4), (16).

These investigators hold a quite different opinion, than that of Rose's, of internal sphincter activity. They have shown by means of delicate recording apparatus, that each contraction wave of the bladder is accompanied by an outburst of impulses from the vesical plexus to open the sphincter. Instead of the sphincter being pulled open by the action of the trigonal muscle, they have demonstrated that the true

involuntary sphincter is a tubular shaped muscle which relaxes from above downward as the detrusor contracts as a result of a relationship between the two muscles by the vesical plexus. (4).

The external sphincter cannot be opened until the bladder contracts, its action being to reinforce the internal sphincter; it can, however, be closed voluntarily; whereas the involuntary sphincter opens and shuts slowly, the external sphincter snaps open and shut. (4).

Learmouth calls Denny-Brown's theory of micturation "The mechanism of micturation on desire to urinate." He considers voluntary micturation due to voluntary relaxation of the internal sphincter causing an automatic contraction of the bladder. The other workers in this field deny that the internal sphincter can be voluntarily relaxed, and regard its action as wholly involuntary and due only to contraction waves set up within the bladder. Learmouth further states that the parasympathetic is the only peripheral pathway necessary for the voluntary interruption of flow, as opposed to the view held by others that it is the contraction of the perineal muscles, and especially the levator ani that is responsible. (2), (4), (6).

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PART II --- THE NEUROGENIC BLADDER

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PART II -- THE NEUROGENIC BLADDER

Having considered the essential nervous mechanisms concerned in normal bladder function, we shall now consider the problem of the neurogenic bladder which, in the course of clinical work, is frequently encountered.

"The actually proven presence or absence of a neurogenic bladder makes a given clinical diagnosis certain or eliminates it entirely. Such a finding is an important clue and directs the internist or neurologist toward the proper diagnosis. Occasionally there are cases which, because of the absence of any urinary symptoms, the question of a neurogenic disturbance of the bladder is not considered. Yet, such a disturbance may exist in spite of the absence of any urinary complaints. The neurogenic disturbance of the bladder, because of the process of compensation, does not give any subjective symptoms until the late stages. As the disease progresses, the compensatory mechanisms having failed, the patient begins to have symptoms of improper drainage such as frequency and incomplete emptying. Since these symptoms are also present in vesical neck obstructions, the neurogenic cause of such symptoms may be frequently overlooked. The urologist is then called upon to determine whether the pathologic involvement is neurogenic or obstructive." (18).

In the past, diagnosis of a neurogenic bladder could be made only by the use of a cystoscopic examination. While cystoscopic examination, frequently is adequate in advanced states, it does not serve to differentiate a cord involvement from an obstructive lesion in the earlier stages of a neurogenic bladder.

In a neurogenic bladder, the physiologic status of the

detrusor is markedly altered, even in the early stages of the disease, since the normal tonus of the muscle is dependent upon the reception of normal nervous impulses over clear nerve pathways. The state of tonus of the detrusor is, therefore, an indication of neurogenic involvement. (18).

The determination of such a change in tonus is made by the use of the cystometer. It is an apparatus designed to record the change of tonus in the bladder wall as the bladder is filled with water. The cystometer, in its simplest form, consists of a syringe, equipped with a two-way valve so that water can be drawn out of a flask when the plunger is drawn back, and forced into the bladder, by means of a catheter or cystoscope, when the plunger is pushed in. A side tube running from the syringe to bladder line, connects with a mercury manometer. Since the system is kept closed, any variation in intracystic pressure is shown by a rise or fall of the mercury column. Since the intracystic pressure oscillates within a fairly wide range, a constant point is used, namely, the highest rise of the mercury after each injection of fluid. The pressure after each injection is then graphed against the cc of fluid injected into the bladder. (19), (See Fig. 2.).

There are two basic types of a neurogenic bladder -- the hypertonic and the hypotonic. The hypertonic bladder is due to an increase in motor impulses to the detrusor and a reciprocal decrease in impulses to the internal sphincter thus giving rise to a contracted bladder wall and a relaxed sphincter. This type of bladder causes true incontinence. A hypotonic bladder is just the opposite so that the bladder is relaxed and therefore enlarged, while the sphincter is spastic. This type of a bladder causes a simulated incontinence, the dribbling or frequent urination being the result of retension of urine

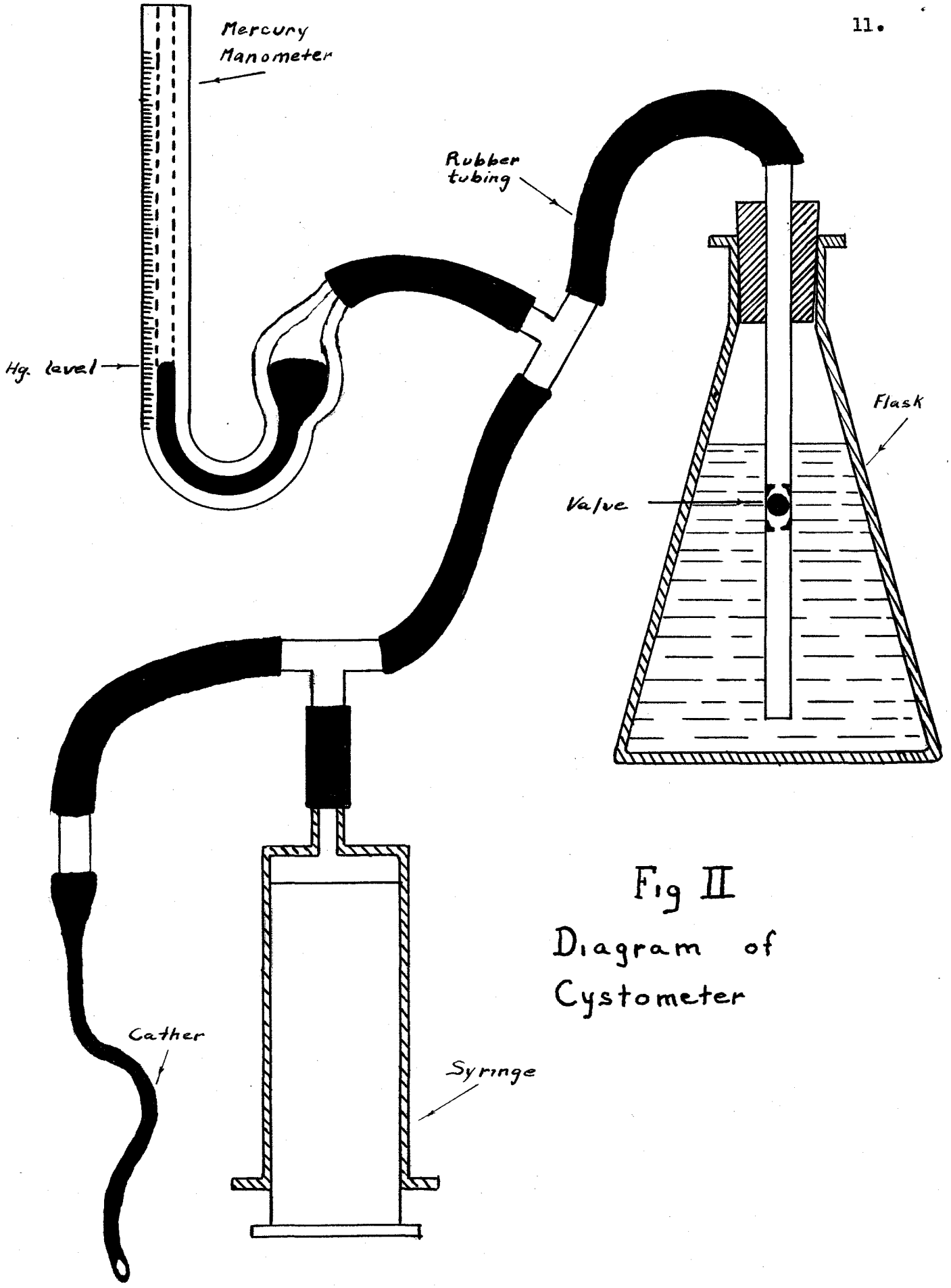


Fig II

Diagram of
Cystometer

with overflow. (18).

In making a cystometric study, the patient is instructed to state when he feels the very first desire to void. The volume of fluid injected is taken at this point. When the patient feels that his bladder is filled to its capacity, and when it becomes painful from the pressure of injected water, he is instructed to bear down as though he were going to void. The pressure in mercury at the peak of this voluntary effort is also recorded. Thus we obtain three factors:

(1) The volume of fluid at the first desire to void.

(2) The pressure curve during filling.

(3) The maximal voluntary pressure.

In the normal bladder, the pressure curve will rise slowly until about 500 cc have been injected into the bladder, after which the pressure rises more rapidly. 600 cc is considered a normal bladder capacity. The first desire to void occurs between 150 and 250 cc, and the maximal voluntary pressure is about 50 mm of mercury. (Fig. 3)

In the hypotonic bladder, the pressure curve is much different. It remains low even after large quantities of fluid have been injected; sometimes even after injection of 1,000 cc the curve will still be low. The first desire to void occurs much later in the filling, between 350 cc and 800 cc, and the maximal voluntary pressure is low, usually being less than 40 mm of mercury. (Fig. 4.)

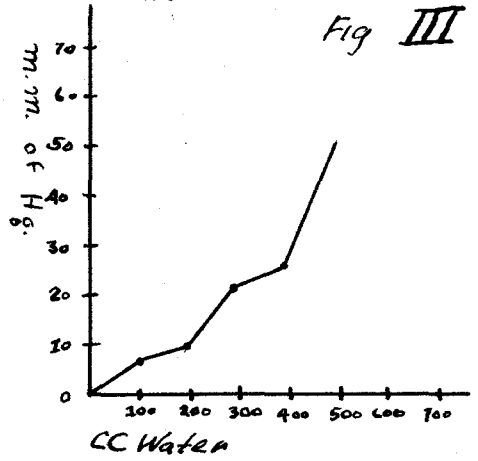
The hypertonic bladder is just the opposite. Here the pressure mounts rapidly after filling begins and the first desire to void occurs early, between 50 cc and 100 cc. Because of the increased tonus, the maximal voluntary pressure is always above 60 mm. (18), (Fig. 5.)

The detrusor - sphincter balance in various forms of incontinence is well illustrated in Figures 6-7-8-9-10-11. The ⁄ sign

CYSTOMETROGRAMS

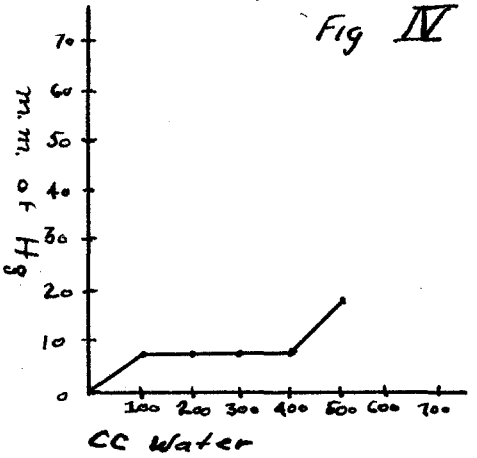
NORMAL

Fig III



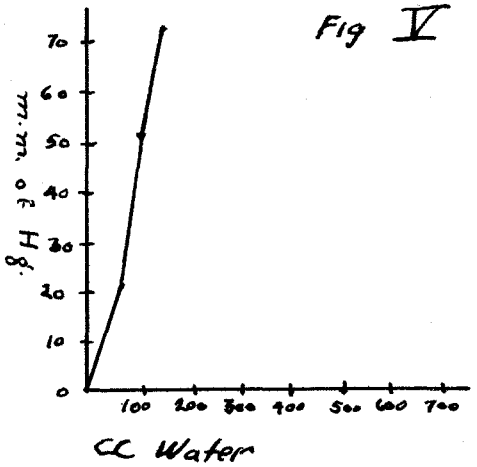
HYPOTONY

Fig IV



HYPERTONY

Fig V



GRAPHIC ILLUSTRATION OF VARIOUS FORMS OF INCONTINENCE

FIG VI

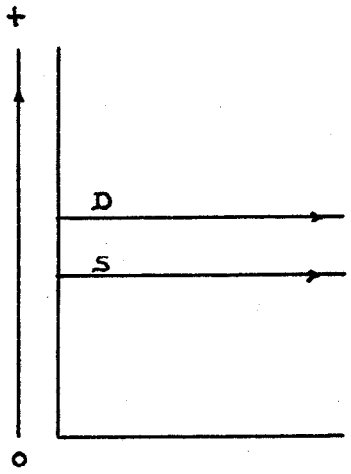


FIG VII

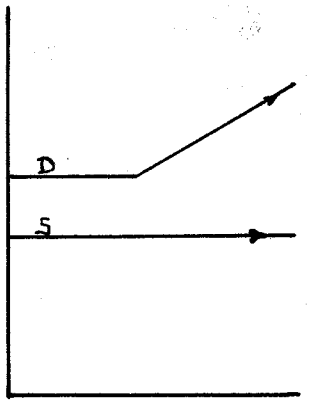


FIG VIII

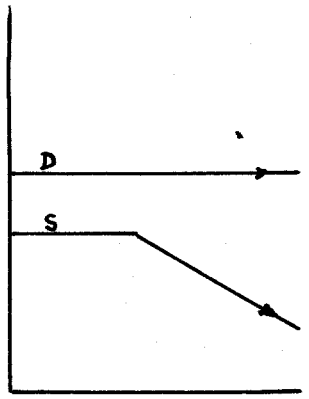


FIG IX

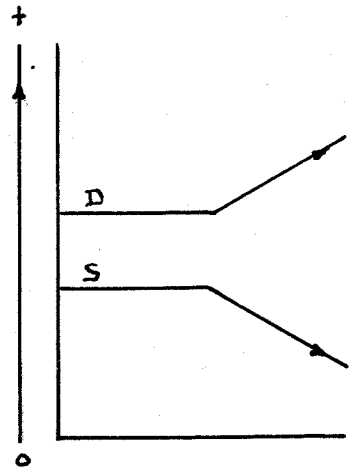


FIG X

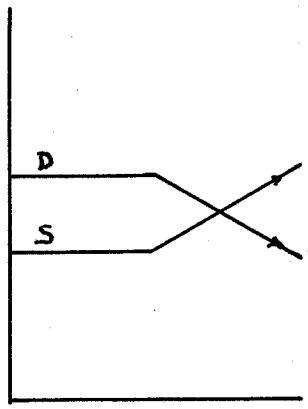
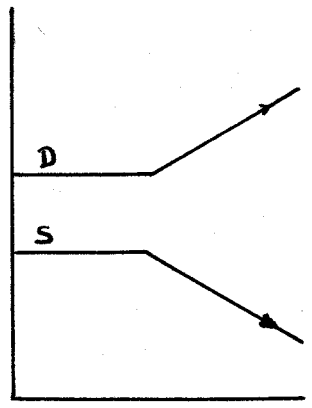


FIG XI



D = Detrusor

S = Sphincter

indicates increased tonus and the "o" sign decreased tonus. Detrusor and sphincter are abbreviated to D and S respectively. Figure 6 is a normal bladder showing an equal relationship in tonus between the detrusor and the sphincter. Figure 7 is a hypertonic detrusor. Figure 8 shows a bladder of the type seen after perineal operations causing a partial or complete incontinence. Here there is decreased tone of the internal sphincter. Figure 9 is a hypertonic bladder with increased detrusor tone and decreased sphincter tone. Figure 10 is a hypotonic bladder showing the opposite type. Figure 11 shows a form following a superpubic prostatectomy in which there is modified hypertonic bladder. It will be seen from the graphs that only Figures 9 and 10 represent true neurogenic bladders showing the reciprocal relationship between the sphincter and the bladder wall. (20).

BLADDER ABNORMALITIES RESULTING FROM SECTION OF A SPINAL CORD.
(See T 12)

A common cause of a neurogenic bladder is a transverse lesion of the spinal cord cutting off impulses from the brain below the site of the lesion. This type of lesion is frequently due to a fracture of the vertebrae with a resulting compression of the cord. The condition may, however, be produced by tumors, infections, and especially during war time, gun shot wounds.

If the lesion is above the 12th Thoracic spinal segment, and does not result in the death of the individual, the bladder becomes markedly hypotonic. Because of the damage to the cord, the patient goes at once into a state of "spinal shock." This condition is manifested by a marked state of depression of reflexes. The bladder is, of course, dependent upon reflexes in the lumbo-sacral region of the cord for its normal functioning. It is the depression of these reflexes that causes

the bladder to lose its normal tone. Cystometric study made on the bladder during this two to three week period of "spinal shock" will show the absence of a first desire to void, and an extremely flat pressure curve. The patient is usually unable to raise the pressure by a voluntary maximal effort. This type of bladder may hold quantities of fluid exceeding 1,000 cc with no sensation of distension to the patient and no attempt on the part of the bladder to expel its contents.

As the state of shock gradually wears off, a reflex emptying of the bladder is set up with emptying occurring usually six to eight hours apart. Micturation is completely involuntary and soon becomes powerful and quite complete; the initiation of flow evidentally being due to bladder wall distension. The sphincter bears the same reciprocal relation to the detrusor in a reflex bladder as it does in a normal one, relaxation of the sphincter occurring as the detrusor contracts.

If a cystometric analysis is made of a reflex bladder, the picture is seen to be entirely changed, for instead of being hypotonic, the bladder is found to be hypertonic. There will be no first desire to void or indeed any sensation as the result of the interruption of the sensory tracts in the cord, but the pressure rises rapidly as soon as filling begins, and the spontaneous expulsion of urine occurs at 200 - 500 cc. The total volume of urine held becomes somewhat smaller than usual, about 300 cc, and the resting pressure is extremely high. (4),(5).

Munro considers that after the state of spinal shock has worn off, and before the bladder has become hypertonic, that it goes thru an "autonomous" state. Here, the detrusor muscle and the internal sphincter begin to show signs of activity, as the result of the intramural nerve plexus activity. However, in this autonomous state, the bladder contractions are not powerful enough to cause complete

emptying, and so a more or less constant dribbling occurs, with retension of urine, as the muscle tonus increases, this state passes into the hypertonic state described previously.

If the treatment of the bladder has been adequate, the hypertonic state gradually passes into a state not unlike a normal bladder. In this case, re-education of control of micturation, and stretching of the contracted detrusor muscle will, in favorable cases, result in a more normal bladder capacity, a tonus, while still high, more nearly approaching the normal, and ability to inhibit, to some degree, bladder contractions. Thus, a cystometrogram made in this final stage will show (1) a residual urine up to 16% of the fill, (2) initial increased tonicity, (3) emptying contractions occurring usually throughout filling but always after 400 cc have been put in, (4) inability to retain bladder contents after 400-500 cc have been instilled. (34),(35).

BLADDER ABNORMALITIES RESULTING FROM PUDIC NERVE NEURITIS

A cystometrogram made on a patient with this type of lesion will show an essentially normal first desire to void, pressure curve, and volume. However, when the bladder is filled to about a normal limit, the pressure will rise suddenly and, at comparatively low pressure, the patient will void spontaneously. It must be remembered that a person with a normal bladder will be able to retain its contents even though the bladder is filled to capacity. In these cases the cystometrogram together with a history of enuresis, perineal tingling, and constant desire to void in the absence of definite neurological findings, will serve to establish a diagnosis of hyper-irritability of the nerve to initiate micturation, that is, the Pudic nerve. (21).

BLADDER ABNORMALITIES RESULTING FROM TUMORS OF THE BRAIN

Watts and Uhle, in a series of cases with brain tumor, showed

that many of them had, in addition to local brain pathology, neurogenic bladders. These were of both the hypotonic and hypertonic type, and were sufficiently abnormal so that they could not be explained on a basis of increased spinal fluid pressure, or mental changes.

The hypotonic types usually had no abnormal urinary history but cystometric studies showed low intracystic pressures, large volumes, shift of the first desire to void to the right, that is occurring later than normal in filling and a subnormal maximal voluntary pressure.

The hypertonic cases usually complained of urgency, and sometimes of involuntary micturation. Cystometrograms made on these cases showed characteristic hypertonicity findings such as high intracystic pressure, shift of first desire to the left occurring earlier in filling and a low volume with a high maximal pressure.

In cases of this kind, you must think of brain tumor in the absence of local pathology, spinal pathology, and negative blood and spinal Wassermanns.

No attempt was made to localize the micturation centers, or to tell from the cystometric studies what portion of the brain was involved.

Watts and Uhle feel that the production of these two opposed types of bladders is due to the fact that the brain is capable of sending out either inhibitory impulses, or exitatory impulses and that the bladder pathology would therefore depend on which center was involved. On the other hand, it may depend on whether the lesion was destructive or irritative in nature. (22).

Bechteren made a review of earlier experiments on this subject, which showed that stimulation of certain portions of the cerebral cortex lead to a vigorous contraction of the detrusor with complete emptying of the bladder. He was able to confirm these experiments, obtaining

his best results by applying stimulation to the medial portion of the sigmoid gyrus both anterior and posterior to the cruciate gyrus. (23).

Evidence of a double pathway from the cerebral cortex to the bladder, one pyramidal, the other extra pyramidal, has been presented by Hunsicker and Spregel. They found that cortical stimulation produced stimulating as well as inhibitory effects on the bladder, and that both reactions depended mainly upon the pelvic nerves: the reactions were not impaired more by severance of the pyramidal tracts than by the extrapyramidal tracts. (24).

Also, using the Horsley-Clark stereo-toxic instrument, Ranson and his co-workers showed that in cats, hypothalamic stimulation produced bladder contractions. (25).

It may be concluded, then, that both exitatory and inhibitory units are present in the cerebral cotrex, region of the hypothalamus, and even in the brain stem. "The abnormalities in bladder function, tone, and sensation in patients with brain tumors are probably the result of a disturbance of bladder representation in certain parts of the brain or of tracts descending from them." (22),(26).

BLADDER ABNORMALITIES RESULTING FROM TABES DORSALIS

Langworthy and his co-workers in a study of 278 patients with tabes dorsalis found that 140 of them had urological complaints. Of this urologic group, urinary incontinence occurred in 83, hesitancy in 71, and nocturnal incontinence in 22. 19 had at one time developed acute retention necessitating catheterization. (27).

Experiments with cats have shown these changes to be dependent upon bilateral section of sacral 2,3,4. They are not produced by section of the lumbar roots of the posterior spinal columns. (28).

D. K. Rose considers tabetic bladders to be divided into two classes.

The first group is that in which the luetic nerve changes produce a spasm of the internal sphincter. As a result of this blockage, the urine accumulates in the bladder causing a myogenic failure of the detrusor. Also, the continued pressure of the urine against the bladder wall causes an anaesthesia.

A cystometrogram will show a low pressure curve, shift of the desire to void to the right (anaesthetic effect), large bladder capacity, and lowered maximal voluntary pressure.

If after a period of retention catheterization, cystometric studies show the detrusor to be gaining strength rapidly, with sensations shifting to the left, it shows the failure due wholly to an irritation of the motor nerves to the sphincter. When the detrusor fails rapidly after the removal of the catheter, some damage has been done to the motor nerves of the bladder wall.

The second type is that in which the neurogenic failure of the detrusor is greater than that of the sphincter. This is a sympathetic over-balance syndrome. Here the cystometrogram may show no desire to void even after 1,000 cc of water have been injected. The pressure remains extremely low throughout the filling and maximal voluntary pressure will be only 10-30 mm of Hg. (21).

BLADDER FUNCTION IN SPINA BIFIDA

Here again, the records of bladder function can be separated into two groups, depending on whether the motor or sensory roots are predominately injured. Langworthy cites one example of each group. The cystometrogrom of one patient showed waves of vesical contraction occurring throughout filling. The contractions varied in their force,

but all were poorly sustained. Thus, with each contraction a small amount of urine escaped. After 250 cc had been introduced, the patient voided. These findings show the parasympathetic fibers to have been injured, so that the recurrent small contractions of the bladder are due to stimulation of the muscle by the intrinsic vesical plexus.

The second patient's bladder had an increased capacity (750 cc), shift of first desire to the right, and while the maximal voluntary pressure was 61 mm., he was unable to maintain the contraction. This study together with a neurologic finding of anaesthesia of the buttocks and back of thighs, was sufficient to diagnose the nerve injury as predominantly sensory, partly motor. (27).

BLADDER FUNCTION IN ENURESIS

Enuresis is the result of a physiologic imbalance of the bladder nerves with a parasympathetic predominance. This decreases the bladder capacity and lowers the threshold of sensation so that the first desire to void produces an impulse to stimulate a forcible contraction of the detrusor. Thus a cystometrogram will show a hypertonic bladder. It is of interest to note that in this condition, the cystometer is used not only for diagnosis, but also for treatment. This treatment consists in instilling water into the bladder and raising the pressure to ever increasing heights. This treatment increases the bladder capacity, in conjunction with a proper psychological regime, may result in a secondary raising of the sensory threshold. (29).

BLADDER ABNORMALITIES RESULTING FROM DAMAGE TO THE CORTICO-SPINAL TRACTS

Langworthy and Kolb found in experiments on cats that by removing one cerebral motor cortex, they could produce a marked decrease in bladder volume, and that by removing both cerebral motor cortices, an

even greater reduction in volume resulted. Later experiments, in which the motor cortex was stimulated showed that stimulation produced a fall in intravesical pressure. If the stimulation was prolonged, the preliminary fall was followed by a sudden rise in pressure, and emptying of the bladder. (7), (9).

Cystometric studies of patients with bilateral cortico-spinal injury showed the bladder capacity to be reduced to as little as 50 cc. The stretch reflex was hyperactive so that the introduction of small quantities of fluid produced vigorous waves of contraction. The pressure curve mounted rapidly and the final contraction to produce micturation occurred suddenly raising the pressure to higher than normal levels.

In persons suffering from a unilateral destruction of cortico-spinal pathways, the same type of a cystometrogram was obtained, except that the changes were less marked, However, those persons with an interruption of the impulses from the left hemisphere showed more impairment of function than those with a right lobe interruption, leading these investigators to believe that the left cerebral motor cortex is dominant in bladder control. (27).

BLADDER ABNORMALITIES ASSOCIATED WITH LESIONS OF THE CEREBELLUM AND ITS PATHWAYS

In 1926, Holman showed that midline cerebellar tumors produced abnormalities in micturation. Since there was, in no case, evidence of damage to the cortico-spinal tracts, Holman believed the bladder disturbance to be the result of pressure on the medulla interferring with the centers in the cortex which control micturation.

Langworthy and his associates are of the opinion that disturbances in the cerebellum itself are capable of causing bladder symptoms. They show two cases, one with a cerebellar tumor, the other with

disseminated sclerosis. Both patients gave a history of hesitancy and retention. Their cystometric studies show hypotonic bladders very similar to those found commonly in tabetic patients. These investigators believe that tone in the bladder musculature, like that of striated muscle, is reduced when the cerebellar pathways are injured. (27).

BLADDER ABNORMALITIES ASSOCIATED WITH PREGNANCY

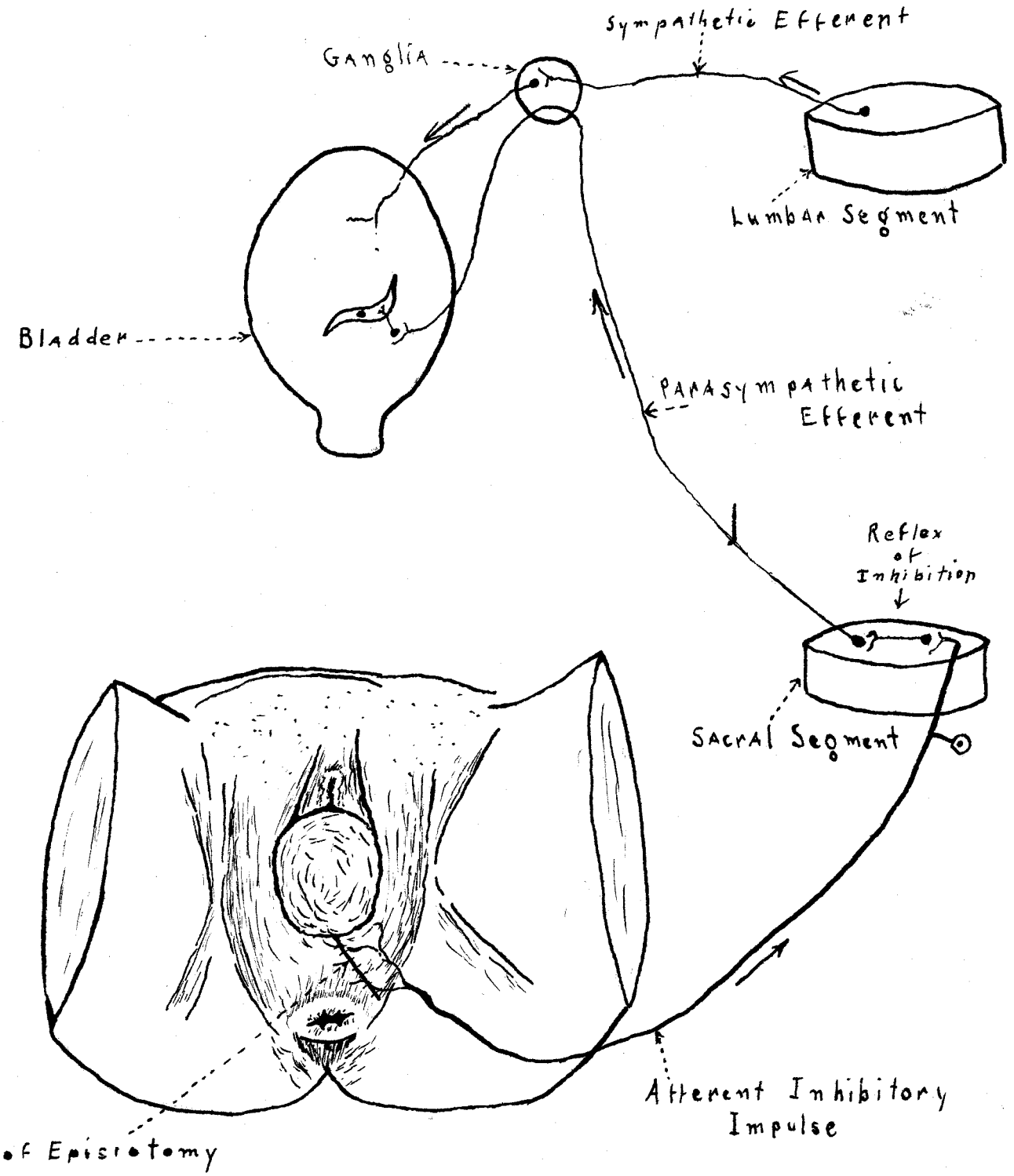
As a result of increase pressure on the nervus pudendus in pregnancy and at delivery, a physiologic nerve block may result. This takes place either at the inner surface of the ramus of the ischium or on the inferior border of symphasis pubis. Since it is this nerve which normally conveys the sensation of a first desire to void, the sensory threshold is elevated.

Thus a cystometric study will show a shift of the first desire to the right, and a low curve until the sensory nerves (sympathetic) of distention are stimulated, after which the motor reflex will cause contraction of the detrusor and elevate the intracystic pressure. Total volume is increased, but maximal pressure will be within normal limits. Micturation, once initiated, will continue only long enough to reduce the fluid volume to a level at which the sensory nerves are no longer stimulated, thus leaving a residual volume of urine.

Post partum retention may in some instances, be influenced by episiotomy. This may be explained by noting that the incised area is innervated by the same spinal cord segments as those entering into the motor portion of the autonomic reflex mechanism, so that a referred or reflex type of sympathetic over balance may ensue from the reflex inhibition of the parasympathetic nerves. A cystometric study in a case of this kind will show a characteristic hypotonic bladder.

Fig. XII

Diagram of possible reflex inhibition of Bladder by Episiotomy



It follows that both of these neurologic disturbances may be present at the same time, in varying degrees. (12).

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PART III

A GROUP OF BRIEF CASE HISTORIES WITH CYSTOMETROGRAMS

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PART III --- A GROUP OF BRIEF CASE HISTORIES WITH CYSTOMETROGRAMS

Dr. Payson Adams has also done considerable work in the field of cystometry. The following cases are those in which he has used the cystometer to establish a diagnosis of neurogenic bladder, and in which I have had the good fortune to assist him. The case histories, for brevity's sake, include only the salient points in symptoms and findings.

TABES DORSALIS

O. D., #23443, Douglas County Hospital. Aged 58 (Fig. 13). Admitted with history of infrequency for the past two months, and root pains and inability to walk for the past four years. Spinal Wassermann positive. Neurological examination showed an alternating strabismus, taste and smell subnormal, leg muscles atrophic and spastic on attempted movement, and tender reflexes hyperactive. Babinski and Oppenheim negative. Cystoscopic examination was not made. Diagnosis of syphilitic myelitis. A cystometrogram showed a markedly hypotonic bladder, of the tabetic type. Patient died in spite of vigorous anti-syphilitic treatment.

G. S., #53721, University Hospital. Aged 57, (Fig. XIV - XV). Admitted with a history of dribbling, nocturia, and nocturnal incontinence. The prostate was not hypertrophied. Neurological examination revealed irregular pupils of Argyl-Robertson type, knee and ankle jerks absent, and negative Babinski. Blood and spinal Wassermanns negative. Cystoscopic showed a 100 cc residual, injection and trabeculation of the mucosa, and no "V" at the anterior vesical neck. The posterior urethra could not be seen. A neurological diagnosis of tabes dorsalis was made. The cystometrogrm showed the first desire to be normal, but the pressure curve rose slowly and the capacity was increased, therefore,

CYSTOMETROGRAMS

FIG XIII O.D. #23443

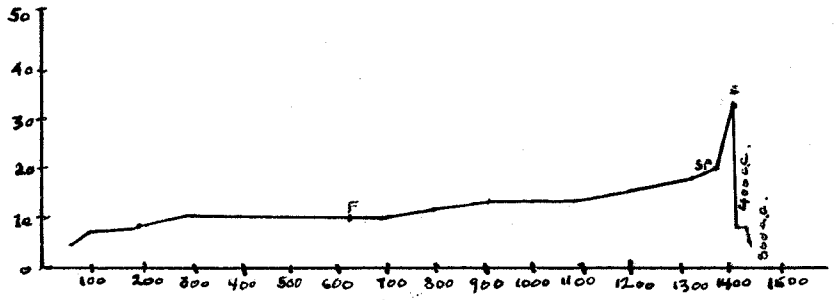


FIG XIV G.S. #53721

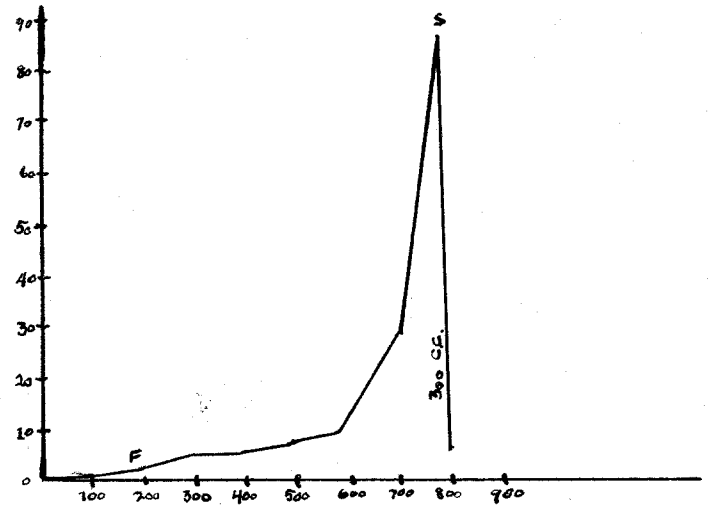
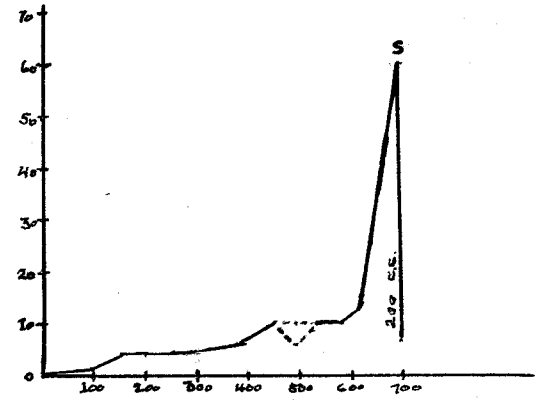


FIG XV GS 53721



an early cord bladder of the hypotonic type. Patient was put on antiluetic treatment.

C. M., #60227, University Dispensary. Aged 50, (Fig. XVI - XVII). Complained of urgency of micturation amounting to incontinence at times, but did not dribble continuously as an over-flow or sphincter incontinence would. Has had enuresis at times and hematuria for the past five days. Neurological showed irregular pupils of Argyl-Robertson type, some nystagmus, ataxia, positive Rhomberg, and increased tendon and muscle sense. Blood Wassermann positive. Cystoscopic found a trabiculated bladder wall, with many small hemorrhagic areas. Diagnosis of Tabes-Dorsalis and Neuro-Syphilis. A cystometrogram revealed a normal bladder, another taken five weeks later confirmed the findings of the first. It is quite likely that the bladder symptoms of this patient were due to the local irritation in the bladder.

REFLEX BLADDERS

P. M., #19535, University Dispensary. Aged 27, (Fig. XVIII - XIX). Loss of bladder and rectal control following fracture of cervical vertebrae. Complains of dribbling and incontinence. Neurological showed spastic paralysis of muscles below level of injury. Babinski was positive. A cystoscopic examination was not made. A diagnosis of traumatic paraplegia -- upper motor neuron involvement was made. The first cystometric study showed generalized increase in tonus, shift of the first desire to the left, but an increased bladder capacity. A cystometrogram made at a later date revealed a markedly hypertonic bladder with small capacity demonstrating the third stage in the development of a reflex bladder.

A. D., #55246, University Hospital. Aged 19, (Fig. XX). Complaint of loss of bladder control following a fracture of T 9-10. Neurological

CYSTOMETROGRAMS

FIG XVI C.M. #60227

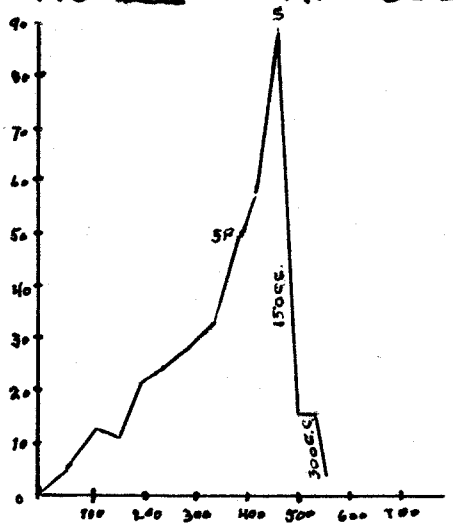


FIG XVII C.M. #60227

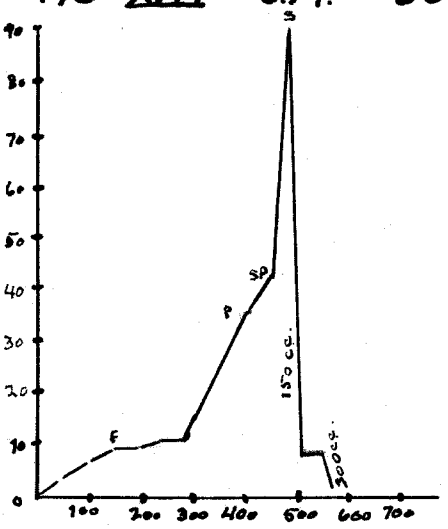
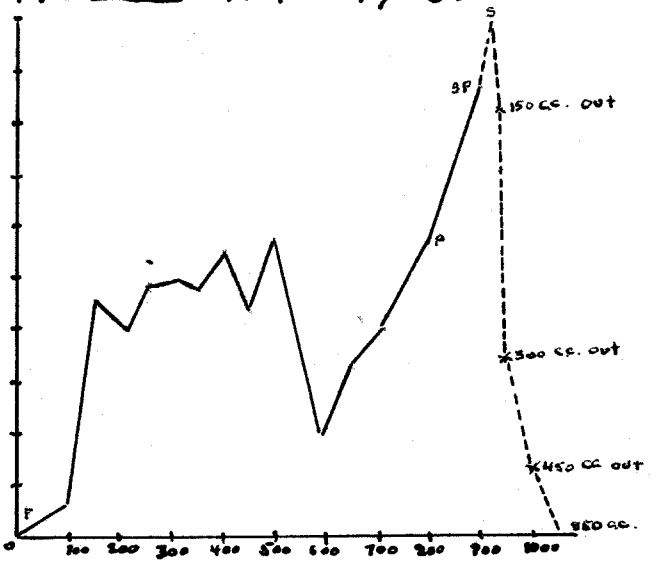


FIG XVIII A.M. #19535



CYSTOMETROGRAMS

FIG ~~XIX~~ A.M. # 19535

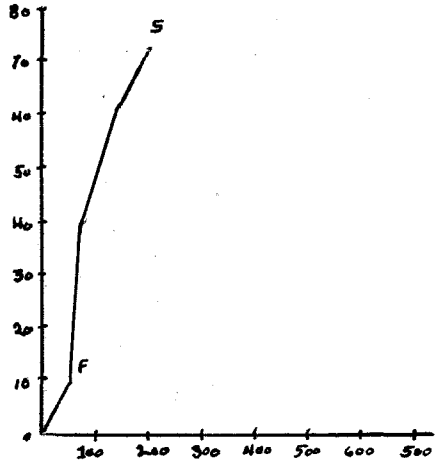


FIG ~~XX~~ A.D. # 55276

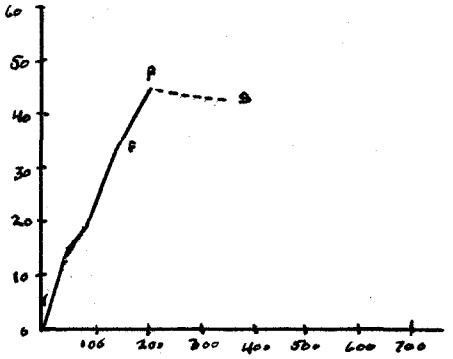
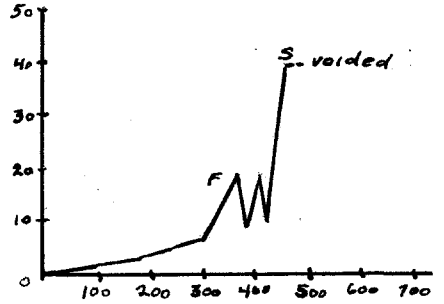


FIG ~~XXI~~ A.T. # 53160



shows a complete sensory and motor paralysis below the illac crests, no reflexes present, and loss of rectal sphincter tone. Diagnosis of permanent cord injury below the level of injury was made. A retension catheter was placed in the bladder and a cystometric study taken thru the catheter show the bladder to be entirely reflex in character, that is, a hypertonic bladder of low capacity with spontaneous emptying. Voluntary straining did not raise the pressure, rather it fell off.

A. T., #53160, University Hospital. Aged 28, (Fig. XXI). Complaint of bladder and rectal incontinence and paralysis since spinal injury five years previous to admittance. Neurological examination disclosed a loss of all sensation below D 5 with pyramidal tract signs, spastic paralysis, and sphincter incontinence. A cystoscopic examination was not made, but a cystometrogram showed shift of first desire to right, decreased capacity, and spontaneous emptying of bladder at a low pressure. Patient was unable to raise the pressure voluntarily. A diagnosis of post-traumatic transverse myelitis at T 6 was made. His condition did not improve while in the hospital.

Mr. G., Methodist Hospital. Aged 35 (Fig. XXII). History of bladder and rectal incontinence following an injury to the spine. Examination shows a complete sensory and motor loss below site of fracture, L 1. Reflexes below the injury were all absent. A cystoscopic examination was not made, but a cystometric study shows a shift of sensation, albert vogue, to the left, sharply rising curve, low capacity, and spontaneous emptying of the bladder at a low pressure. The patient was unable to voluntarily raise the pressure. The diagnosis was a transverse myelitis at the site of fracture.

SPINAL TUMORS

CYSTOMETROGRAMS

FIG XXII M.G. Methodist

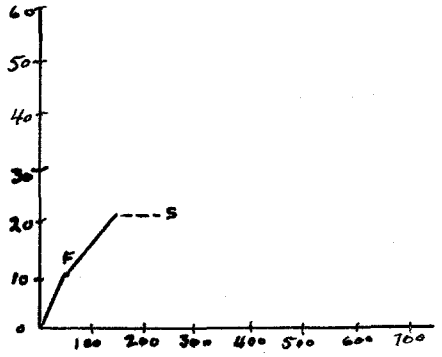


FIG XXIII F.W. # 53900

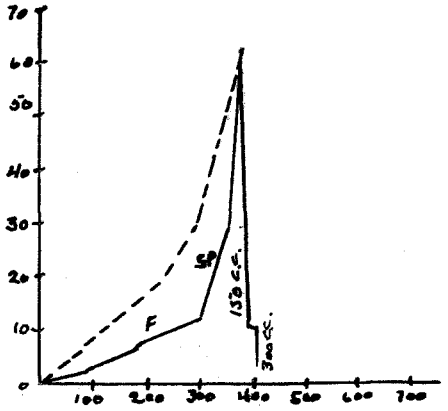
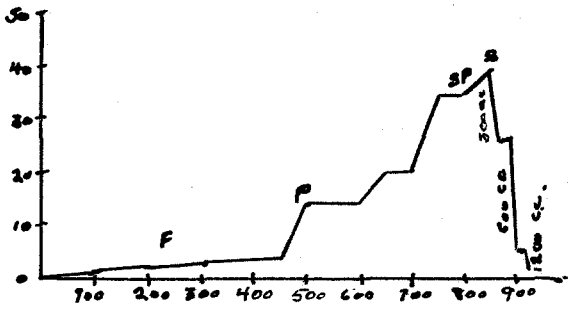


FIG XXIV H.W. #553375



F. N., #53900, University Hospital. Aged 56, (Fig. XXIII). History of retension. Retension catheter installed. Complained of pain in back, neurological examination disclosed complete anaesthesia below level of T 5, band of hyperalgesia above this level. Abdominal and cremasteric reflexes absent, anal reflex normal, deep reflexes hyperactive on lower extremities, more marked on the left, Astereognosis of both feet, and Babinski, Oppenheim and Eansalimo positive, bilaterally. Sapastic paralysis of right leg, no impairment of left leg. A cystoscopic examination was not made, but a cystometric study showed sensation to be normal, but a sharply rising curve, and low capacity, that is, a hypertonic type. An x-ray diagnosis was made of a lytic neoplasm of the third thoracic vertebra. The patient was dismissed with not treatment.

H. W., #53375, University Hospital. Aged 69, (Fig. XXIV). Complaint of loss of bladder control for past 1½ weeks, paralysis below waist, pain in lower back; neurological examination showed loss of sensation from mid thorax to hips. Tactile sense decreased from mid-thorax. The bladder was not cystoscoped, but palpation revealed the median lobe of the prostate to be hypertrophied. A diagnosis of a tumor at T 5 causing cord compression was made. A cystometrogram showed the first desire within normal limits, but there was increased bladder capacity and hypotonicity of the detrusor. The patient died not long after admission and an autopsy a tumor involving the third and fourth thoracic vertebrae and the fourth and fifth ribs, and causing cord compression was found. The enlarged prostate would not account for the decreased detrusor tonus.

BRAIN TUMOR

M. W., #53192, University Hospital. Aged 62 (Fig. XXV).

CYSTOMETROGRAMS

FIG XXV M.W. #53192

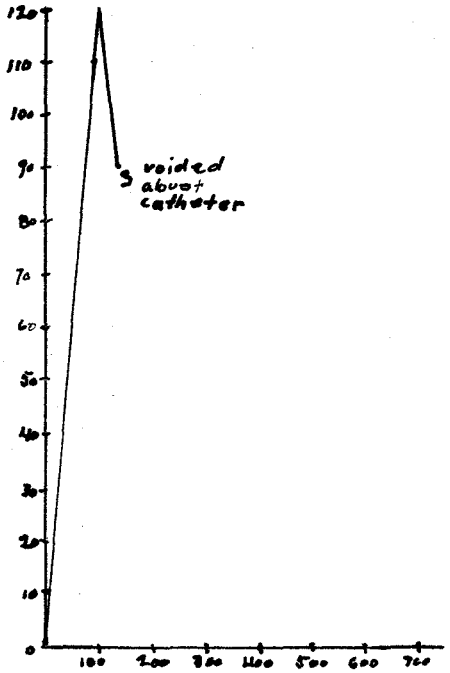


FIG XXVI J.K. #22464

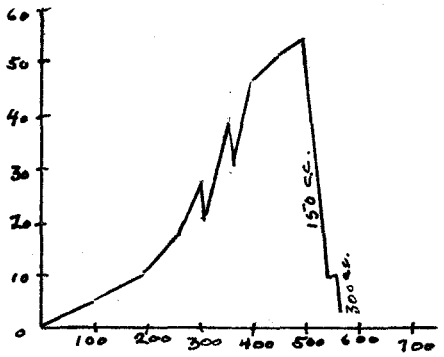
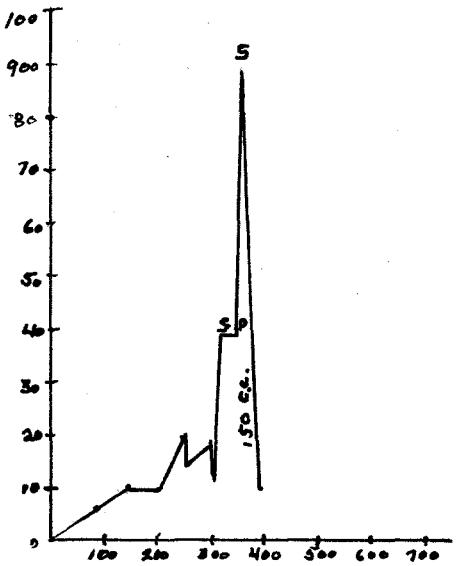


FIG XXVII C.Z. # 53860



Complaint of incontinence, headache, drowsiness, weakness, loss of memory. Examination showed a decrease in mentality; choked disc and blurring of vision; increased motor tonus on left side, no sensory changes; positive Babinski; benign hypertrophy of prostate. A diagnosis of a right lobe glioblastoma was made. Cystometric studies revealed a markedly hypertonic bladder, small capacity, and spontaneous micturation. The patient died, and an autopsy was performed and the ante-mortem diagnosis was confirmed.

MULTIPLE SCLEROSIS

J. K., #22464, Douglas County Hospital, aged 47 (Fig. XXVI). History of Pernicious Anemia. Present complaints of incontinence, hesitancy, nocturia, difficulty in walking, blurring of vision, paresthesias. Neurological examination showed proprioceptive sense fair in upper extremities but lost in the lower extremities. A diagnosis of multiple sclerosis secondary to pernicious anemia was made. A cystoscopic examination was not done but a cystometrogram showed a loss of sensation, but normal curve and capacity. However, the bladder emptied spontaneously at a pressure somewhat below normal.

C. Z., #53860, University Hospital, aged 15 (Fig. XXVII). Urinary complaint of incontinence for one week. Also had unsteady gait and diplopia with some spasm of the deep back muscles. A neurological examination showed all the reflexes to be hyperactive, bilateral ankle clonus, ataxia. There was no Rhomberg however. A diagnosis of multiple sclerosis was made. Cystometric studies showed a markedly hypertonic bladder, with low capacity.

MISCELLANEOUS

P. M., #70536, University Dispensary. Aged 23 (Fig. XXVIII). Complaint of infrequency, and some left sided paralysis. Neurological

CYSTOMETROGRAMS

FIG ~~XXVIII~~ P.M. #70536

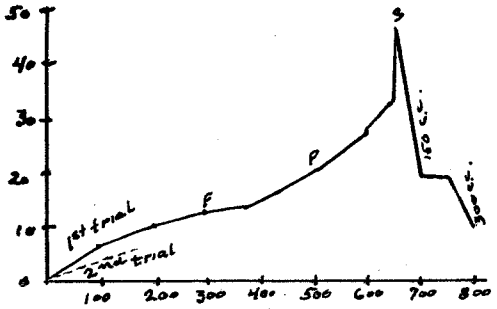
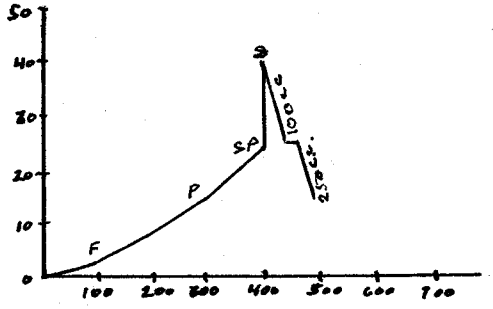


FIG ~~XXIX~~ T.M. #53399



showed spastic paralysis of the left extremities, lagging of left facial muscle, and the eyes were slow in accommodation. The reflexes were all normal. A tentative diagnosis of hysteria was made, but a cystometrogram showed a shift of the first desire to the right, a low curve, and a low maximal pressure; a true neurogenic bladder, of the hypotonic type.

F. M., #53399, University Hospital. Aged 47, (Fig. XXIX). Complaint of dysuria, polyuria, stoppage, difficulty in starting stream, and shooting pains in both legs. A neurological examination showed an atrophy of the lef muscles with resulting loss of function, and a stocking type of burning paresthesia over both lower extremities. The reflexes were all normal. The condition was diagnosed as functional. A cystoscopic examination was made, but the only positive findings were a tight meatus and spastic external sphincter. A cystometrogram showed a shift of first desire to the right, low bladder capacity, and low maximal voluntary pressure. The pressure curve was normal.

CONCLUSION

Cystometry has proven to be of definite value in the diagnosis not only at the presence of a neurogenic bladder, but also, in some degree, of the type of neurogenic involvement.

However, the localization of spinal lesions still presents many difficulties for, as Muschat has pointed out, the same type of curve is found in lesions of different spinal segments, and it is impossible to tell if one system of the autonomic mechanism is irritated, or the other destroyed. (33) - (Fig. XXX).

There are some pathological conditions in the bladder which will simulate a cord bladder. An acute hypertonic curve will be obtained in tumors of the bladder, stones in the bladder and acute and subacute

FIGURE XXX

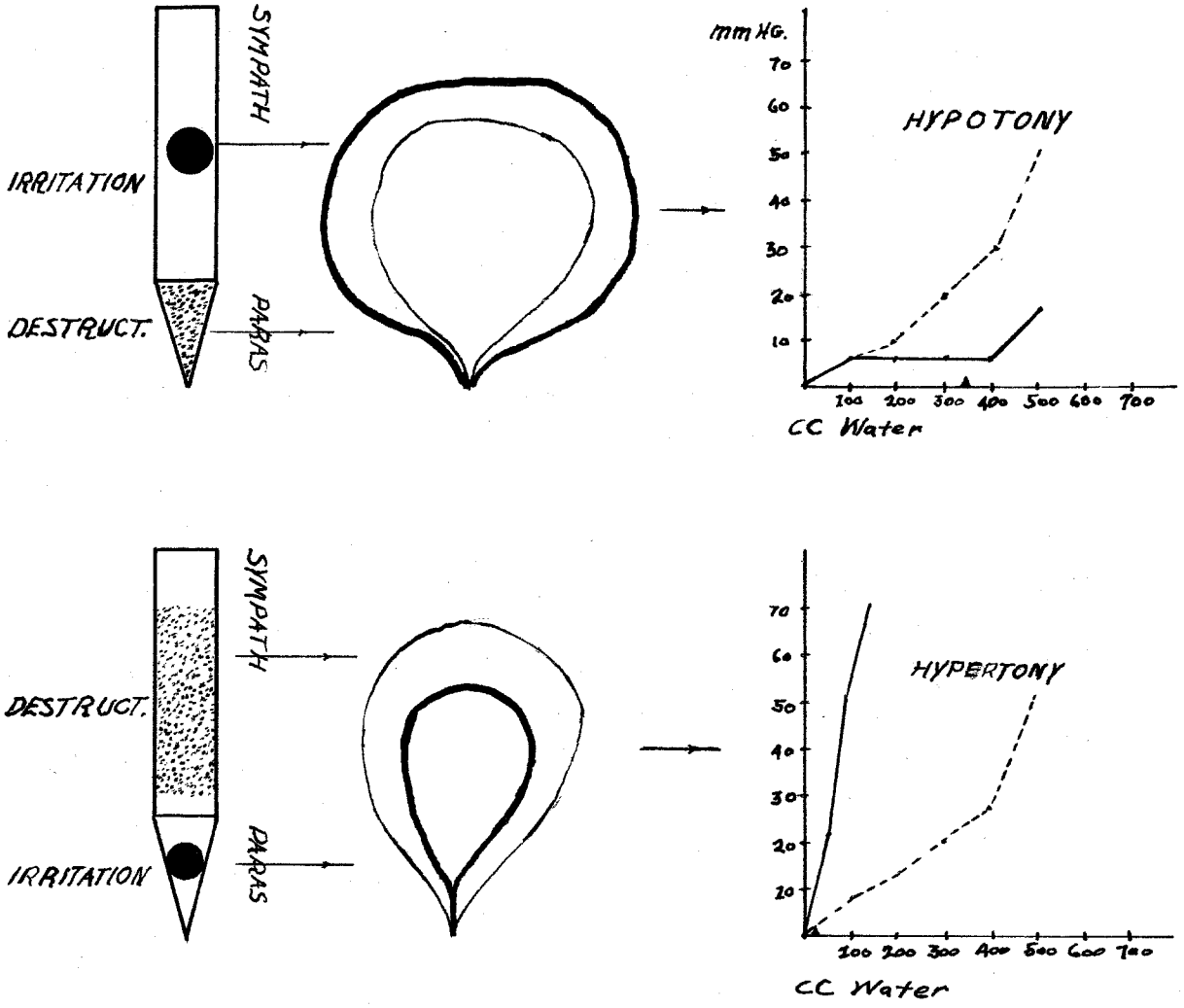


Diagram showing the type of bladder and type of cystometrogram one obtains in destructive and irritative lesions of the lumbar and sacral segments of the spinal cord. The same curve is obtained in an irritative lesion of the lumbar sympathetics as in a destructive lesion of the sacral-parasympathetics, thus making diagnostic localization very difficult.

cystitis. A hypotonic curve will be obtained in a cystocele, diverticulum, renal reflux, and post-operative or post-partum dysfunctions. A complete urological examination, which should always accompany a cystometric study will differentiate these conditions, from true neurogenic disfunctions. (18).

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BIBLIOGRAPHY

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BIBLIOGRAPHY

1. SACHS, E., D.K. ROSE, and A. KAPLAN

"Tumor of Filum Terminale with Cystometric Studies."
Archives of Neurology and Psychiatry, 24:1133. 1930.

2. LEARMOUTH, J. R.

"Contribution to Neurophysiology of Urinary Bladder in Man."
Brain 54:147. 1931.

3. MULLER, L. H.

"Innervation of the Bladder."
Die Lebensnerven, 4:301. 1924.

4. DENNY-BROWN, D.

"Nervous Disturbances of the Vesical Sphincter."
New England Journal of Medicine, 215:647. 1936.

5. THOMSON-WALKER, J. W.

"The Bladder in Gunshot and Other Injuries of Spinal Cord."
Lancet, 1:173. 1917.

6. ROSE, D. K.

"Urinary Incontinence."
Journal of the Missouri Medical Association, 32:363. 1935.

7. LANGWORTHY, O. R., and L. C. KOLB

"The Encephalitic Control of Tonus in the Urinary Bladder."
Brain: 56:371. 1933.

8. BARRINGTON, F. G. F.

"The Localization of the Paths Subserving Micturation in the Spinal Cord of the Cat."
Brain, 56:126. 1933.

9. LANGWORTHY, O. R., and L. C. KOLB

"Demonstration of Encephalitic Control of Micturation by Electrical Stimulation."
Bulletin of the Johns Hopkins Hospital, 56:37. 1935.

10. ELLIOTT, T. R.

"The Innervation of the Bladder and Urethra."
Journal of Physiology, 35:367. 1907.

BIBLIOGRAPHY

11. ROSE, D. K., and R. DEAKIN

"Cystometric Sutdy of Pharmacology of Bladder, with Additional Data Regarding Physiology of Urination."
Surgery, Gynecology and Obstetrics, 46:221. 1928.

12. ROSE, D. K.

"Postpartum Pyelitis of Pregnancy - Explanation Based on Physiology of Bladder."
American Journal of Surgery, 25:394. 1934.

13. DENNY-BROWN, D., and F. G. ROBERTSON

"On the Physiology of Micturation."
Brain, 36:149. 1933

14. WESSON, M. B.

"Anatomical, Embryological, and Physiological Studies of Trigone and Neck of Bladder."
Journal of Urology, 4:279. 1920.

15. YOUNG, H. H., and D. I. MACHT

"Physiology and Pharmacology of Trigonum Vesical."
Journal of Pharmacology and Experimental Therapeutics, 22:329. 1923.

16. MUNRO, D.

"The Activity of the Urinary Bladder as Measured By a New and Inexpensive Cystometer."
New England Journal of Medicine, 214:617. 1936.

17. EVANS, J. P.

"Observations on the Nerves of Supply to the Bladder and Urethra of the Cat; With a Study of Their Action Potentials."
Journal of Physiology, 86:399. 1936.

18. MUSCHAT, M.

"Diagnosis of a Neurogenic Bladder by Means of the Cystometer."
Pennsylvania Medical Journal, 39:493. 1936.

19. ROSE, D. K.

"Determination of Bladder Pressure with Cystometer, New Principle in Diagnosis."
Journal of the American Medical Association, 88:151. 1927.

BIBLIOGRAPHY

20. MUSCHAT, M.

"Graphic Illustration of Various Forms of Incontinence." American Journal of Surgery, 31:104. 1936.

21. ROSE, D. K.

"Various Cystometrograms and Their Interpretation." Journal of Urology, 27:207. 1932.

22. WATTS, J. W. and C. A.W. UHLE

"Bladder Dysfunction in Cases of Brain Tumor." Journal of Urology, 34:10. 1935.

23. BECHTEREN, W.

"Die Functionen Der Nervencentra." (Third Edition, Volume 1.) Jena, Gustav Fischer. 1911.

24. HUNSICKER, W. C., Jr., and E. A. SPIEGEL

"Conduction of Cortical Impulses to Autonomic System." Society for Experimental Biology and Medicine. 31:874. 1934.

25. KABAT, H., H. W. MAGOUN, and S. W. RANSON

"Electrical Stimulation of Hypothalamus." Society for Experimental Biology and Medicine. 31:541. 1934.

26. WATTS, J. M., and C. A. W. UHLE

"The Significance of Hypotonic Bladder Associated with Megacolon: Evidence for Central Origin." Urologic and Cutaneous Review, 40:244. 1936.

27. LANGWORTHY, O. R., L. G. LEWIS, J. E. DEES, and F. H. HESSER

" A Clinical Study of the Control of the Bladder by the Central Nervous System." Bulletin of the Johns Hopkins Hospital, 58:89. 1936.

28. DEES, J. E. and O. R. LANGWORTHY

"An Experimental Study of Bladder Disturbances Analogous to Those of Tabes Dorsalis." Journal of Urology, 34:359. 1935.

29. ROSE, D. K., and R. DEAKIN

"Cystometric Diagnosis of Central Nervous System Syphilis: New Appreciation of the Term 'neurogenic bladder.'" American Journal of Syphilis, 13:371. 1929.

BIBLIOGRAPHY

30. HOLMAN, E.

"Difficult Urination Associated with Intracranial Tumors of the Posterior Fossa."
Archives of Neurology and Psychiatry, 15:371. 1926.

31. MACDONALD, A. D., and McCREA, E. D.

"Observations on Control of Bladder, Effects of Nerve Stimulation and Drugs."
Quarterly Journal of Experimental Physiology, 20:379. 1930.

32. EVANS, C. L.

"Recent Advances in Physiology" (Fifthe Edition)

Philadelphia, Blakeston's Son and Co., Inc. 1936.

33. MUSCHAT, M.

"The Value of Cystometry."
Journal of Urology, 33:366. 1935.

34. MUNRO, D.

"The Cord Bladder."
Journal of Urology, 36:710. 1936.

35. MUNRO, D., and J. HAHN

"Tidal Drainage of the Urinary Bladder."
New England Journal of Medicine, 212:229. 1935.