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Review of the reflexes and neurological signs in the lower extremity

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A Review of the Reflexes and Neurological Signs in the Lower Extremity.

by

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Introduction and
Scope of the Work Attempted in this Paper.

A thesis dealing with neurological diagnosis must of necessity be limited in scope to only one or two phases of that subject. In choosing reflexes as the portion of that subject that will be discussed here, I am taking a portion considered by many writers to be one of the most important phases of diagnosis in neurological disorders. Their names and reasons for so thinking will be given later. Confining the paper to the clinical side of reflexes of the lower extremity, of necessity presupposes a general knowledge of physiology and anatomy of the nervous system on the part of the reader, and such subjects are therefore omitted except where clinical phenomena demand explanation on that particular basis. Likewise, much of the early research on reflexes in general was done by animal experimentation, and while of utmost importance, the results dealt strictly with physiology and not always paralleled clinical findings, therefore, they are discussed only when directly connected to the clinical phase of the subject. The variations found in very young children are not presented, and no attempt is made to give an encyclopedic list of the various diseases which may be indicated by each pathological reflex or sign; their occasional listing being used only as illustrations of certain general principles. Reflexes and signs requiring special equipment for their elicitation (e.g. electrical stimulation) are not considered, and the

effects of drugs on such signs are discussed only when such is a common modification found clinically.

To aid in the orientation of the reader as to the position of this subject matter in the field neurological diagnosis in general, I quote a complete plan of examination as recommended by Grinker (42):-

I Anamnesis

II Head and neck (inspection, percussion, auscultation).

III Cranial nerves

IV Upper Extremeties (motor power, tone, atrophies, coordination, sensation, reflexes and abnormal movements)

V Trunk (motor power, tone, atrophies, coordination, sensation, reflexes)

VI Lower Extremities (motor power, tone, atrophies, coordination, sensation, reflexes, abnormal movements.)

VII Cerebellar tests

VIII Speech, writing, and mental status

IX Gait

X Laboratory and Roentgenography as indicated.

Taking the heading number VI above, I have enlarged upon it to make the outline of material discussed in this paper:

I History -

A short chronological summary of neurological diagnosis in

general, and reflexes in particular.

II General Value and Use of Reflexes.

III Specific Reflexes and Signs----------Under each Reflex:

A. Tendon and Periosteal Reflexes

or so called Deep Reflexes

1. Patellar or Knee Jerk
2. Ankle Jerk
3. Adductor Responses
4. Flexion Reflex of the leg
5. Tendon Reflexes of the foot
6. Heel Tap Reflex
7. Rossolimo Reflex
8. Bechterew-Mendel Reflex
9. Adie Syndrome
10. Other Deep Reflexes

1. Theory of Production
2. Method of eliciting
3. Normal and Pathological Responses
4. Interpretation of Response
5. Special Values:
   (a) Thereputic use
   (b) Prognostic use
   (c) Examples of specific diseases showing the reflex or sign.

B. Cutaneous Reflexes or

Superficial Reflexes

1. Plantar Reflex, Babinski Sign and its modifications
2. Tibial Flexion of great toe
3. Chaddock's Sign
4. Gordon's Paradoxic Reflex
5. Oppenheim's Reflex
6. Other Cutaneous Reflexes of the leg and foot

C. Associated Movements and

Reflexes of Spinal Automatism

D. Miscellaneous

1. Diagnostic Signs such as:
   (a) Kernig
   (b) Lasegue
   (c) Patrick
   (d) Romberg
2. Reflexes in Transverse Lesions of Cord
3. Simulation and Malingering (detection by use of Reflexes).

IV Bibliography

Clinical examination of the nervous system in human beings differs markedly from the examination of other systems of the body. Although there are many gaps in our knowledge of the anatomy and physiology of that system, that which is known seems extremely definite and exact, and localization of disease processes can be made more accurately than in any other part of the body. The reasons for this are given by Grinker (42) as follows:

"In general medicine symptoms and signs of disease processes are for the most part positive in nature. That is, the disease manifests itself by some indication of its presence inherent in the new and pathological process. A neurological disease, however, manifests itself not by positive findings inherent in the disease, but by the effects of that disease on normal function. The pathological process produces destruction within the nervous system and the result of that lesion is loss of function, the loss corresponding to the area destroyed. Also, there may be abnormal activity because of release of lower structures which have been inhibited by the area destroyed. In general then we must seek to determine first, where is the lesion; second, what is the lesion, and third, what has caused the lesion."

It is toward this end that the clinical use of reflexes is commonly directed, and it is in this particular type of examination that they are of greatest value.

Newman (72) and Grinker (42) give a summary of the development of reflexes and reflex action in the evolution of life

forms, starting with the single celled organism. Here, they say, inherent properties in the protoplasm makes even a simple cell contain certain abilities to react and adjust to changes in environment. However, this is entirely a local response and as the scale of life ascends, and larger groups of cells compose the individual, rapid adjustments required a specialized protaplasm with high speed conduction. The first specialized nerve cell probably resulted from specialization of an epithelial cell. Processes developed on its free borders which were modified for conduction, and in that way the cell could transmit stimuli through its substance to the interior of the organism. This was essentially a sensory cell. From these cells, primary ganglion cells evolved by central migration. They received impulses from the above neurosensory cells and gave off processes which innervated effector organs. These cells had no polarization and only a diffuse connection with the periphery. Out of this simple structure of ganglion cells, groups of cells or ganglia condensed, and these always had to do with the correlation of motor and sensory functions of the one particular segment of the organism in which they had developed. The individual ganglions were connected with each other by long fibers. The most cephalad ganglia developed most rapidly and eventually controlled the segmental ganglia. Gradually there appeared more definite localization of activity as connections of neurones became more specific and their functions more localized, but never has the control of function as a whole dis-

appeared, since all structural units are still intimately connected.

A reflex (42) has been defined as:

"An invariable mechanically determined adaptive response to the stimulation of sense organs, involving a center of adjustment and the conductors necessary to connect this center with the appropriate effector apparatus."

However, under reflexes have been included the most simple and the most complicated motor and psychic reactions, and indeed, some neurologists believe all neuromuscular activity, in its broadest sense to be reflex.

The main characteristics of reflex activity, while not of direct clinical importance, are so fundamental to the understanding of complex reflex activity that they certainly bear repeating. Sherrington (87) has differentiated reflex function from ordinary nerve function and conduction by the following characteristic properties:

"1. Reflexes show a slower speed of conduction, as evidenced by the latency of response. 2. They show the phenomenon of after discharge when the stimulation has ceased. 3. They respond to a summation of several subliminal stimuli. 4. They show irreversibility to conduction from afferent to efferent neurone. 5. They show fatigue on repetition of stimuli. 6. They have a variable threshold value. 7. They are mutually related to allied and antagonistic reflexes and therefore may be exalted or inhibited.

8. They are greatly dependent on a constant blood and oxygen supply. 9. They show the less important characteristics of facilitation after repeated stimuli have traveled over the same pathway; and of occlusion and successive induction which promote smoothness in response."

Thus, have the main characteristics of reflex action been enumerated; the evolution of nervous integration been summarized and the portion of neurological diagnosis to be discussed in this paper been definitely outlined.

Chronological History

As Wechsler states (105):

"In a measure it is erroneous to trace the history of any specialty to the dawn of medicine in Egypt and Greece. While it may be true that the Egyptians carried specialization to such an extent that they had doctors for every disease, or part of the body, the fact is that they dealt largely in magic and not medicine and had neither doctors nor specialists. . . . Even the special clevage between medicine and surgery did not take place until the advent of the early Italian Universities. It is true that some physicians paid special attention to the nervous system, but they knew little of its functions and practically nothing of its clinical manifestations. Of neurology in general, one may therefore say that it is as old as medicine, but as a specialty it is as young as yesterday."

Wechsler (105) states that possibly the earliest neurological reference is the sign of meningitis in the Edwin Smith papyrus (2500 B.C.) where an Egyptian physician observed that his patient could not look down to his chest. He says also that in the Ebers papyrus (1580-1350 B.C.) there appears a drawing of an atrophied limb, probably from infantile paralysis; and that it gives definite evidence that they had words for brain, spine, vertebra and skull.

Garrison (31) in his review of the identity of all forms

of ancient and primitive medicine, gives no reference to neurology, reflexes, or neurological diagnosis, but in the realm of psychiatry he compares the folk lore of savages and their warding off of diseases by fantastic make-ups, dances and chants to our modern faith healers and to the psychotherapy used in some mental diseases.

Both of the above authors agree that Egyptian, Sumerian and early Oriental medicine contributed very little to neurological diagnosis. We then get to the period of Hippocrates where according to Wechsler (105) neurology as well as the other branches of medicine have their true beginning. Hippocrates described many neurological diseases but did his most extensive work in his treaties on wounds of the head. This takes us up to 1000 to 2000 years B.C. to the greatest physician after Hippocrates, Galen, who is thought of as the father of experimental physiology. (31). Galen never failed to explain all pathological phenomena on theoretical bases and did his greatest work as far as accuracy is concerned in neurology. He dissected ox brains and described many anatomical points -- the dura mater, pia mater, corpus collosum, the third and fourth ventricles and aqueduct, the fornix, corpora quadrigemina, vermiform process and the hypophysis. He also knew of seven pairs of cranial nerves and he mentioned the sympathetic ganglia as reinforcers of the nerves. While his anatomy was faulty, in the long run his experimental physiology was unexcelled. He made the first experimental section of the spinal cord and produced

hemiplegia. He caused aphonia by cutting the recurrent laryngeal nerve and gave the first logical explanation of respiration. Galen's myology was based on his dissection of apes but he knew much about muscles and their actions.

The Romans of this period added little to neurological knowledge.

After Galen, medical science didn't advance much for many centuries and the Byzantine, Mohammedan and Jewish periods up to 1096 A.D. also contributed very little. According to Wechsler (105) the middle ages did to neurology what they did to other branches of medicine and science in general, namely, put it to sleep. Except for an occasional spark of originality during the third and fourth centuries immediately following the death of Galen not a glimmer of light was seen on the horizon until the Renaissance was well under way.

Garrison says (31) that the Renaissance and revival of learning of the sixteenth century was important, but that really it was during the seventeenth century, during the period of individual endeavor that the greatest advances were made. The great names that stand out in this period are: William Harvey, Raymond Vieussens and Rene' Descartes. The latter in one of his treaties gives the first experiment in reflex action, the familiar one of making a person bat his eyes by aiming a mock blow at them. The name Johann Bohn (1640 to 1719) is important for as a German physiologist he experimented with the decapi-

tated frog in an entirely modern spirit. He declared reflex phenomena to be entirely material as against the current view of vital spirits in the nerve fluid and he showed that the nerves did not contain a nerve juice.

Then in the eighteenth century, Spallanzani (1768) founded the doctrine of regeneration in the spinal cord as based on his observation of its growth during regeneration of the tail in the lizard. He also showed that the sexual posture in the frog is maintained as a spinal reflex after decapitation.

William C. Cruikshank, (1745 to 1800) of Edinburgh was an assistant of William Hunter and he investigated the reunion and regeneration of divided nerves. Robert Whytt also of Edinburgh is memorable for his work on the physiology and pathology of the nervous system. Garrison says he demonstrated in 1750 for the first time that the integrity of the spinal cord as a whole is not necessary for reflex action and that the preservation of only a small fragment of it will suffice for this purpose. He was one of the first to describe spinal shock and his work was soon followed by the discovery of the cerebal spinal fluid in 1774 by Cotugno.

Garrison also describes electrophysiology as having its beginning in the epic making experiments on muscle-nerve preparations in 1792 by Galvani of Bologna. This was followed and improved on by Volta of Pavia.

Reaching the modern period (from 1800 on according to Garrison) we find the main work which proved that reflex phenomena

were not bound up with ideation and sensation. The Bell-Magendie experiment (1811 to 1822) and the discovery of the respiratory center by Legallois and Flourens were of great help to this end. Independent of these men, Marshall Hall (1790 to 1857) of Nottingham established the difference between volitional action and unconscious reflexes. It was Hall's work that gave reflex action a permanent place in physiology. However, he did not realize as Sherrington and others have since pointed out, (87) that volitional and reflex processes can pass from one to the other and that many nervous phenomena lie between the two extremes.

The masters of physiology according to Garrison (31) in the second half of the nineteenth century contributed the ground work of present day neurological knowledge; their contributions are too numerous to review but many are known by the prominent name of the discoverer. In the front rank are Helmholtz, Claude Bernard, and Carl Ludwig, and in the second rank come du Bois Reymond, Brucke, Goltz, Pfluger, and Brown-Séquard.

The modern concept of the reflex theory was an outgrowth of the cell theory with its important correllary the neuron theory, for it was through the labor of the histologist that the complex paths for transmitting impulses from nerve cell to nerve cell were traced out. The initial data were the Bell-Magendie law of the spinal nerve roots, the law of Wallerian degeneration, and Goltz's work on the effects of excision of parts of the central nervous system. The discovery of central inhibition of spinal reflexes

by Setchenoff (1863) plus his studies on localized reflexes, such as the knee jerk were of utmost importance. As the neuron theory and resulting pathways became more complex, it was soon perceived that the nervous system functions as a whole and it was this idea which was so remarkably handled by C. S. Sherrington (87) whose work on reflexes as shown by animal experimentation is unsurpassed. Other workers more interested in the clinical side were Erb and Westphal 1875, Jendressick 1885, Weir Mitchell and Lewis 1886, Lombard 1889 and Pavlov in 1900.

According to Viets (99) "The beginnings of neurology as a separate discipline of medicine are so clearly defined that one may say, neurology began with Romberg a hundred years ago.". However, Garrison (31) and Wechsler (105) think modern neurology is mainly of French extraction and derives from Duchenne of Boulogne, through Charcot and his pupils. They say Duchenne as the father of modern neurology was truly a lone wolf. He cared little for book knowledge and worked the entire problem of tabes dorsales out for himself and never knew of the work of Romberg until afterwards. He also pointed out that the paralysis of infants had to be a spinal cord lesion and not merely an atrophy from disuse.

Viets (99) says that Charcot as a contemporary of Duchenne worked in wider fields and with more accuracy than his better known collegue.

He created the greatest neurological clinic of modern

times and was followed by enthusiastic students from all parts of the world. Pierre Marie of Paris, Charcott's ablest pupil, made at least four original deliniations of new forms of disease. They are: his description of acromegaly, pointing out the pituatary lesion; hypertrophic pulmonary osteoarthropathy; hereditary cerebellar ataxia; and a special type of spinal arthritis deformans. His latest work has been on periphlral nerves.

The ablest German neurologist after Romberg is William Erb, already referred to in connection with electro diagnosis. Simultaneously with Westphal, Erb discovered the significance of the knee jerk in tabes and helped establish a statistical causal relation between tabes and syphilis. Other prominent Germans of the period were Friedreich, Westphal, Quincke, Strumpel, Oppenheim and Lewandowsky.

Viets (99) and Garrison (31) say that the English Hughlings Jackson and Gowers brought to completion the foundation of modern neurology as we know it today. They, however, were both building on the sound framework of the older German and French schools. Jackson's greatest work was in originating the doctrine of "levels" of the nervous system; Gowers work was especially on spinal cord diseases. Two other prominent English neurologists of the period were Bastian on paralysis and aphasia, and Horsely in the realm of neurosurgery and endocrinology.

In America, Weir Mitchell, trained by Claude Bernard in Paris, and Dana, with his textbook of 1892 added new knowledge

to neurology and pointed the way to the advances that were to be made in the twentieth century. Clinic lectures given by Weir Mitchell (64) in 1888 show that in the field of tendon jerks the knowledge of the time differed little from our present clinical conceptions. Thus at the turn of the century, most of the spinal cord and intrinsic nerve diseases were known, the larger tracts had been identified, the motor cortex had been partly demonstrated in animals and gross anatomy and pathology of the nervous system were quite well understood. Expert in diagnosis, (99) the neurologist of 1900 was equipped with very few methods of treatment, and clinical neurology was nearly at a standstill until the advent of three sources from which it was to derive new stimulation. These were: 1. the discovery of a specific test for syphilis, and the identification of tabes and dementia paralytica as syphilitic diseases; 2. Diagnosis by examination of the cerebrospinal fluid; 3. The discovery of an astounding pharmaceutical in arsephenamine. No less a source of stimulation was the work of Sherrington and his publication of "The Integrative Action of the Nervous System" in 1906. "To his laboratory", says Viets (99)," came a pupil from America who ultimately was to establish securely a new branch of medicine, neurosurgery. That pupil, Cushing, had so far advanced the field of neurosurgery that by 1914 surgical treatment was being used in over 10% of all neurological patients." From that impetus and from the problems arising out of the Great War, the growth of

neurology has been steady, and the changes have been a part of the every day life of many prominent neurologists living today. For example, the expert advice of the roentgenologist has increased the diagnostic acumen by demonstration of the bony structures surrounding the nervous system, and their pathological changes. Ventriculography, and the more useful encephalography have now become almost routine diagnostic procedures, and the newest product of the laboratory, electroencephalography is gradually entering the clinics with the possibility of becoming of greater value than any of the above.

So closes the outline of the growth of neurology in general and of neurological diagnosis and the reflexes in particular. Naturally, many names, many problems, many blind alleys of investigation, all almost as important as the ones listed above have been omitted, and no one has adequately expressed an opinion as to the future in this field, but all agree that neurology, especially in the light of biochemistry and biophysics of the nervous system, has much ahead of it that so far has barely been perceived.

The General Use and Value of Reflexes

There have been many definitions, or attempted definitions of reflexes, some have been stated previously and all are quite inadequate when considered from every angle. But, as Gordon (39) says, the reflex act is a fundamental manifestation of the nervous system, and he continues with a description of its components. In its simplest form it consists of a peripheral excitation, which is transmitted through the sensory or centripital pathway (afferent) to the nervous centers where it is transformed to motor action by passing to muscles or effector organs via the efferent or centrifugal pathways. Whether such simplicity and lack of associated neurons is ever found actually, is of little importance as far as the fundamental procedure is concerned. The chief characteristic of a reflex, he says, lies in its automatic execution irrespective of the individual's will.

Gordon (39) divides the common reflexes in man into only two groups: 1st, those associated with the cerebrospinal tracts, i.e. tendon, cutaneous and deglutition reflexes, and 2nd, those associated with the sympathetic system, e.g.(flushes, salivation, intestinal contractions, etc.).

Monrad-Krohn (67) distinguishes five types, for the purposes of clinical investigation: 1. Tendon and periosteal, or the deep reflexes, 2. Cutaneous reflexes, 3. Reflexes of spinal auto-

matism, 4. Postural reflexes, 5. Organic reflexes.

Purves-Stewart (77) classifies reflexes only as, 1. Superficial or skin, 2. Deep or tendon and 3. Organic or visceral; while Wechsler (105) classes all abnormal responses in one group, as the Pathological reflexes.

From the work of all those men mentioned above I concluded that the reflexes usually studied in diseases of the nervous system are limited in number, and are members of two main groups. These are the groups of greatest diagnostic importance and they form the basis of most reflex studies from the clinical standpoint. They are the Cutaneous and the Tendon or periosteal reflexes, and by them the cerebrospinal system is examined. Those reflexes in which the sympathetic system is involved are more complex, and very little is known about them.

Monrad-Krohn says, "In neurology more than in any other branch of medicine, the diagnosis is built up of a number of details found by clinical examination. The whole neurological examination should be completed without speculation into the diagnosis, then after tabulation of the findings, a focal diagnosis should be made based on anatomy and physiology. Only after this, comes the consideration of the nature of the lesion as based on general pathology. In this manner, a case doesn't have to be typical to be recognized and each case is individualized as it should be . . . . Testing the reflexes is one of the

most important parts of the examination - probably the most important. Its great advantage is its objectivity. It is not so dependent on the attention and intelligence of the patient as is the motor and sensory examination. It can be carried out in unconscious patients and in children, and attempts at simulating reflex changes is a rare occurrence and as a rule is very easy to discover."

Russell (84) picks reflexes to the exclusion of other methods of clinical examination, as the best means of arriving at a diagnosis of an affection of the nervous system.

Elliott (22) says that without doubt the study of reflexes is one of the most important phases of neurological examination, because: "1. They can be tested on the unconscious person, the child, the stupid and uncooperative patients of all kinds. 2. Their results can't be simulated, and it tells therefore if a thing is functional or organic, and if the symptoms are real or feigned. 3. It helps to localize the lesion, and then from a knowledge of pathology and from clinical experience, the nature of the disease can often be determined (e.g. if posterior columns are involved, tabes would be thought of because of the frequency of such pathology in that area)." However, he warns, that while the information given by reflexes is objective, it requires proper evaluation if the maximum or even correct worth is to be obtained. Always is it necessary to correlate other symptoms and findings, for an abnormal reflex alone is often of little im-

portance. He concludes with the statement that "In the co-operative patient of average intelligence, the history is still the all-important single feature".

Wechsler (105) feels that the main value of reflexes consists of the objective evidence which they furnish about the muscles, peripheral nerves, and the central segmental nervous system. The fact that they are usually easy to elicit is also a point, he thinks. Some practical points when examining reflexes are: to secure relaxation; to get distraction of the attention from the tests; and in deep reflexes to strike the tendon and not the muscle, since myotatic irritability is not being tested. He too, as did Elliott (22), feels that the mere presence or absence of a reflex is not in itself sufficient to indicate the nature or even the definite presence of organic disease, unless properly correlated with other findings. Normally, all the deep and superficial reflexes can be elicited and are equal on both sides. If the reflexes are naturally sluggish or naturally lively, the deep and superficial are equally sluggish or lively. Both the deep and superficial reflexes become significant of pathological disturbance if they are unequal on the two sides, or if there is a discrepancy between the deep and superficial. If the so-called pathological reflexes can be demonstrated in addition, then a definite lesion may be inferred, but <u>not</u> the nature of it.

Purves-Stewart (77) claims that many of the cutaneous and deep reflexes can be inhibited by voluntary effort, and that there-

fore cooperation of the patient is necessary.

Grinker (42) too says that it is very difficult to know the normal in tendon jerks for they all vary so much with the state of excitability of the nervous system at the time of the examination. He warns that reflexes must always be elicited with the clothes removed, for not only is the movement of the limb to be observed, but also must we watch for any muscular contraction that may be too slight to cause a real movement of the limb.

Russell (84) cites specific examples of cases sent to him with diagnoses of neurathenia, hysteria and functional affections of the nervous system where by careful examination he has found at least one pathological response of reflexes and so had picked up (as shown by the subsequent course of the cases) actual organic disturbances in the early stages. Likewise, cases referred as peripheral nerve diseases were found to have positive pyramidal tract signs and so were proven to be central in origin. From these cases he then states that reflexes are of real value in determining, 1. real or feigned symptoms, 2. functional or organic disease, 3. central or peripheral origin of symptoms; and as a result of these three, they are of value in, 4. medico-legal cases where the above things need to be disproven or substantiated.

Inman (54) in his "Notes on Pathological Reflexes", states that it is sometimes difficult to say with certainty

whether a disturbance of function in the nervous system depends on actual structural changes in the system, or whether it might be due to so-called functional "disease". He says this point should be settled, if possible, by neurological examination, before conjecturing as to the probable nature of the disorder, and it is in this determination that the pathological reflexes are important. Properly elicited, the presence of these reflexes denotes in the majority of cases, abnormal action of some part of the cerebrospinal axis, usually the cortico-spinal tracts. But he adds that it must be kept in mind that things other than central lesions may cause these signs or cause abnormal reflex responses, (and these of course will be taken up with the individual reflex and its interpretation).

Because the patient rarely knows whether or not a reflex should be obtainable, there is little or no chance of imposing a subjective syndrome by the examination in a case of questionable organic disease, and this is a distinct advantage of reflexes over subjective examination. Along this line, Babinski (7) states that while much of value in neurology can be learned from history and subjective examination, there is always the danger of imposing a subjective syndrome by suggestion, and he cites as an example the fact that by careful avoidance of any suggestion in his questions in taking histories in cases of hysteria, he had not encountered a single case of hemianesthesia in eighteen years.

Another point to be considered in the use of reflexes gen-

erally is discussed by Church and Peterson (17). They stress that the normal activity of reflexes requires not only the integrity of the reflex center in the cord, and both peripheral afferent and efferent limbs of the arc, but also a proper association with the higher cerebral levels. Thus, the mental state and general irritability of the central nervous system may modify the local segmental reflex response.

Also, the well known general rule that interference between the spinal center and the cerebral levels causes increased reflex manifestations of the cord, doesn't hold true in complete division of the cord in man (as will be discussed later). These men also contradict the statement made earlier by Elliott, Russell and Monrod-Krohn, that reflexes can't be inhibited voluntarily, and they go even further and say that almost all require absolute passiveness on the part of the patient. As a final statement on this topic of general use of reflexes, these authors point out that a local sensitiveness in a part (e.g. rheumatism in the lower extremity) may be associated with an increased reflex irritability of the part.

This then has shown what some neurologists think of reflexes, what their chief values are and some of the general points about the use of reflexes. I have tried to show where reflexes stand in the field of neurological examination and of what special advantage this type of examination is as compared with the other methods of obtaining information.

The Deep Reflexes

or

Tendon and Periosteal Reflexes

The Knee Jerk, or Patellar Tendon Reflex

The study of tendon reflexes in general has been so closely connected to the study of the knee jerk that this reflex will be taken up first in the deep reflex group. This is the most familiar and the most convenient of the tendon reflexes.

According to Pritchard (76), it was during the year 1875, that there appeared in the Archives fur Psychiatrie and Nerven krankungen, two articles on the knee jerk, one by Erb and one by Westphal. These appear to be the first recorded studies of a phenomenon which had been referred to in medical literature since 1780. (See chronological history). From the very first, the concept of these two early writers differed as to what the phenomenon actually was. Erb held that it was a true reflex involving neuron paths, while Westphal said it was merely a local muscular response.

This argument stimulated no less than 300 articles in the next eighteen years (as shown by the bibliography of a monograph by Sternberg (76)) on an almost purely physiological concept, and this argument has persisted to some extent even to the present time.

Fulton (27) in his monograph tells how the fact of the knee jerk being a spinal reflex was proven by Jolly, Snyder, and Dodge, against the muscular theory. Their work on measuring the time for the reflex response, coupled with Piper's measurements on the rate of conduction of impulses in mammalian nerve fibers, proved that although the time of the reflex was extremely short, it was not

short enough to be used as an argument against its being a true reflex.

Snyder (89) and Hoffmann(48) each working on normal subjects, found that the electromyographically determined patellar tendon reflex time ranged from .008 to .024 second. Travis and Hunter(95) eighteen years later in 1928 report that they found in 87 normal adults an average time of .0197 second, and that under normal conditions the reflex time was relatively constant for each subject.

Purves-Stewart (77) in his textbook states that while he doesn't consider the patellar tendon reflex a true reflex, it serves the same purpose clinically because it tests the integrity of the reflex arc by means of the reflex tonus normally present, which in turn is maintained by an intact reflex arc.

This seems to express the opinion of most workers at the present time (Abrahams(1) , Howell(51) , Wechsler(105) , Pritchard(76), etc.) and is sufficient to establish the clinical value of the test.

McCouch and Alpers (5) raise the question of elements involved in eliciting the knee jerk being present other than the stretch factor. They claim a response can be obtained by a blow on the inferior part of the patella which does not shorten the quadriceps tendon, and they suggest that sensation in the subpatellar bursa would be associated with the production of the knee jerk. Using cats, they produced evidence of ipselateral contraction which may be elicited by stimulation of the subpatel-

lar bursa either mechanically or electrically, and they think this component may contribute to the clinical response of the knee jerk and that errors may be made by an impairment of this component. They do not explain this assumption any further, and their work has not been confirmed.

Pritchard (76) presents an interesting phase when he states that all the changes in form of the knee jerk and its actual occurrence has been explained only in terms of physiological function (as given above) but that function is always related to purpose, and that as yet, no adequate explanation of the tendon reflexes has been given on this basis. It is difficult to explain tendon jerks in such a manner. He says that earlier writers thought they were purposeless peripheral phenomena, but that Sternberg considered them to be protective actions for the joints. But, even holding to the more modern viewpoint of their being examples of myotatic responses, only their mechanism and not their purpose is explained.

Briefly reviewing the anatomical connections of this well known reflex as presented by Ranson (79), Pritchard (76), Purves-Stewart (77), Grinker (42), and Gordon (39); there is first the receptor organ in the tendon, the Golgi tendon organ. This is thought to receive the impulse started by the stimulation of the stretch, since cocainization of this area will abolish the reflex, yet leave the voluntary motor power unaffected. Then the impulse travels up the anterior crural nerve in the afferent fibers

and enters the third lumbar segment of the spinal cord, through the posterior roots. Since an adductor muscle response is so frequently elicited too, the fourth lumbar segment may be said to be also involved in the response. At first, it was thought that no intercalated neurons were to be found in this reflex arc, but most of the above workers believe that at least some of the phenomena show that there is more than one synapse, probably two or three. Then starting from the anterior roots of these segments, the arc is completed by the efferent fibers in the same peripheral nerve, going to the quadriceps and adductor muscles of the thigh.

The first thing to be considered in the clinical aspect of the knee jerk, is the method of eliciting the reflex. This, as has been stated before, is one of the most convenient and the easiest to procure of the tendon reflexes. Gordon (39) summarizes the various unimportant methods and concludes that the patient should sit either with the knees crossed, or with the feet hanging down and not touching the floor; or, if confined to bed, the examiner lifts patient's knees by placing one hand under lower portion of thighs, then after relaxation, or abandonment of the limbs has been obtained in any one of these positions, the patellar tendon should be outlined between the patella and the tibia, and a short, sharp blow delivered to the tendon. A percussion hammer is preferred, but the fingers, side of hand, or some other object can be used. While such a detailed procedure seems unnecessary, it will be of value in obese patients, and one should try to get a standard

method to use alike on all cases and thereby be better able to judge the response. Wechsler (105) mentions the so-called supra-patellar response obtained with the leg extended, but relaxed so that the patella is moveable, then the examiner crooks his index finger above the patella, pushes down a little and strikes finger with hammer, the resultant kick-back of the patella has the same significance as the knee jerk.

The normal response to the stimulation of the knee jerk is an immediate movement of extension of the leg. It is the variation in this response that is of such great clinical importance. The changes depend on the anatomical and functional condition of the nervous system at that moment. Frequently, the examiner forgets the functional factor in interpreting the response. Howell (51) says, "The condition of the spinal cord and the general state of the central nervous system influences the general response of any tendon reflex. The effect of various normal conditions upon the knee jerk has been studied by numerous investigators, particularly Lombard (59). The results are most interesting in that they indicate very clearly that the irritability of the spinal cord varies with almost every marked change in mental activity. During sleep, the knee jerk disappears, and in mental states of a restful nature, its extent is relatively small. In conditions of mental excitement, or irritation, the jerk becomes increased. He also plotted his own nervous irritability as measured by the extent of the knee jerk and found a daily rhythm, which he charted. There-

fore, the knee jerk may be increased or diminished by whatever increases or diminishes the activity of the central nervous system, and so the mere degree of response has many factors in its production and can't be relied upon to give a true indication of organic states."

Even in considering the anatomical changes that cause variations in the response of the knee jerk it must be remembered that the anatomical connections as outlined previously for this reflex, represent only a part of the structures involved functionally, for this lower reflex are is connected, in man, to the cerebrum, and the responses obtained are modified by the state of these connections with the higher centers, (77) and (67).

The changes in response, most frequently encountered are absence or diminution, and exaggeration, plus certain other qualitative changes.

The presence of the knee jerk in adults who show no other abnormality is so consistent that it is usually considered to be constantly present. Pritchard (76) quotes the following figures on normal persons from other workers:

"Sternberg, 6000 adults, with only one failure.

Jendrassik, 1000 adults, with but one failure.

Zenner, 1000 adults, with two failures.

Black, an average of .72% failures in 5000 trials.

Berger, an average of 1.56% failures.

Eulenberg, 4.8% failures, the last two giving no figures on

the number of patients tested."

Gordon (39) says the knee jerk is present in 99% of all normal adults.

As is well known, reinforcement is sometimes necessary in order to bring out a sluggish but present knee jerk. As Purves-Stewart says, (77) "Reinforcement usually consists in having the patient do something with his hands, or by some other method, divert his attention from the examination of the knee jerk. Sometimes cases of actual hypertonus show "absent" knee jerk because the muscle spasm is so severe that movement of the limb is prevented, but once relaxation is obtained (e.g. by diverting patient's attention) the jerks will be found to be hyperactive. Purves-Stewart (77) prefers Laufenauer's method of reinforcement, for here the patient grasps the examiner's arm and squeezes it tightly when told to do so, and thus the examiner can tell if the patient is cooperating in the reinforcement. Also, since only one hand is used by the patient, the upper extremity reflexes can be reinforced considerably. The more common method of Jendrassik, consists of having patient interlock the fingers of the two hands, and then to pull hard just before the tendon is struck. Abrahams (1) mentions methods for reinforcement described by Dragenesco, Kronig, and Rosenbach, where attention is distracted by mental problems, and various physical maneuvers, but he considers them all inferior to the two described above.

Hughes and Strecker (92) say that the phenomena of reinforcement can be explained in two ways: first, that it works because of

"abolishment of cortical inhibition", or second, "by summation of excitation, due to addition of motor response by stimulation of more anterior horn cells than by the one reflex alone".

We can then see, that if the knee jerk is absent, even with reinforcement, that in the vast majority of cases it means some disease process in that individual. Loss of the knee jerk is known as Westphal's sign. What are the possibilities in such a case, and how should one interpret that finding in the light of information given previously?

Pritchard (76) says that one should first distinguish between cases where the knee jerk is lost along with all the other tendon jerks, and those cases where only the knee jerk is lost while others remain present. The former (i.e. when all are lost) can be said to occur in profound asphyxia and narcosis; after epileptic convulsions; in some cases of encephalitis; in extreme debilitation and starvation; in some aged people; in a general amyatonia (a myopathy); a general arthritis with fixation of joints; in increased intracranial pressure, especially in tumors of the posterior fossa (discussed later); and in association with the non-luetic Argyll-Robertson pupils as described by Adie (3) and discussed later in this paper. Such a general loss then is of no localizing value except in the last two cases, and both of these are described later. The others, can generally be diagnosed from the general findings and from the history of the illness itself.

However, when the knee jerk is lost, while other body ten-

don reflexes remain normal, or when there is a difference between the two knee jerks in one person, the picture is entirely different.

Gordon (39) says "A lesion of any portion of the simple reflex arc at this level (L.3 & 4) will abolish the reflex. If the knee jerk is lost and yet the patient can voluntarily use all muscles and has no myopathy, it means organic disease of the sensory or afferent side of the arc (if the loss is persistent)". Rarely could this be in the peripheral afferent fibers, because they are in the same nerve trunk as the efferent fibers and so a lesion here would almost always show some voluntary motor involvement. But it does mean that the lesion could be in the posterior roots, or posterior columns at this level, and of course the one disease most frequently giving such a lesion is tabes dorsalis. Syringomyelia, and multiple sclerosis (according to Abrahams (1)) could give such a picture, but rarely would they be confined to this area, and their other points of attack are much more characteristic.

Then going to the efferent side of the arc, the knee jerk would be abolished by lesions at any point along this side of the arc, but this is often accompanied by paralysis of the muscles of the thigh too. Such lesions may be, 1. in the anterior horn cells of the spinal cord (e.g. chronic or acute poliomyelitis) 2. in the anterior roots by infections, new growths or by trauma, 3. in the peripheral nerves by a neuritis of toxic, or traumatic origin, or by compression or destruction of the nerve at that point.(39)(67).

All of these last are usually accompanied by an atrophy of the muscles of the lower extremity to which these nerves go.

4. The muscles themselves may be the seat of the lost reflex, for in myopathies when the muscle fibers are gone, no response to the knee jerk will occur. Other things causing loss of knee jerk are as follows:

Some men think that at the onset of some acute infections, especially pneumonia, the knee jerk may be lost, but Abrahams (1) in a large series of such cases, reports 24.7% absent; 12.4% diminished; 44.4% normal and 18.5% exaggerated knee jerks. Likewise Gordon (39) thinks that the occasional variation of the knee jerk in acute febrile diseases is without diagnostic value.

Gould, (40) points out that in cases of neuritis, all the symptoms may have cleared up before the knee jerk returns, and indeed, the jerk may disappear before the onset of any other clinical signs.

Gordon (39) also states that there may be loss of the knee jerk in extensive brain lesions producing a suspension of cerebral functions.

Monrad-Krohn (67) reminds us that in any severe deformity of the lower limb, especially a flexion contracture, the tendon jerk of the knee may not be obtainable.

Some workers quoted by Monrad-Krohn (67) believe that when there is complete sensory loss to the part, that the tendon jerks are always abolished, but he has seen numerous exceptions. Monrad-

Krohn also found in 1919 (65) that the knee jerk may be abolished after severe body exertion, when he examined 49 men before and after a 50 kilometer ski race.

From the above facts, it can easily be seen that "simple" absence of knee jerks means a good deal more than just possible tabes, and that the correct interpretation depends on recognition of all the possibilities and especially on correlating that single finding with the general condition of the patient, the history and the rest of the neurological examination.

Now as to the interpretation of another common variation in the patellar reflex response, namely, exaggeration of the knee jerk. As was necessary in the loss of knee jerk, many of the points considered here under knee jerk also apply to tendon jerks in general, and will not be repeated later on when other tendon reflexes of the lower extremity are discussed.

Exaggerated jerks may be either functional or organic in origin, and often it is extremely difficult to tell the two apart. As was stated above, the knee jerk may change from day to day in the same individual, depending upon the state of excitability of the nervous system, and therefore it is often difficult to say definitely that the response is hyperactive, and always must a comparison be made between the knee jerk and other tendon jerks, and if there is a discrepancy, then one should be prepared to explain it. Pritchard (76) and Purves-Stewart (77) maintain that the knee jerk is often exaggerated in neurasthenia and occasionally

in other psychoneuroses, but this is usually along with other tendon reflexes. Exaggerated jerks due to so-called functional causes rarely are exaggerated to the point of clonus, and in fact Hoffman as quoted by Pritchard (48) says that a maintained patellar clonus is never found outside of organic nervous involvement. Lewandowsky, quoted by the same author, considers the addition of an adductor muscle response to the knee jerk indicative of organic disease, but this is disputed by Monrad-Krohn (67), Gordon (39) and Wechsler (105) who say that the adductor response is a frequent accompaniment of the knee jerk in normal individuals. Certainly, as Abrahams says (1), "the functional exaggerations are never accompanied by the Babinski sign, or by loss of the abdominal reflexes", but this shifts the proof of interpretation to another reflex and doesn't aid in evaluating the knee jerk itself.

Considering then the organic basis for exaggeration of the knee jerk. First, we find that it may be increased by overdoses of certain drugs, just as it was abolished by deep narcosis. Strychnine especially is mentioned by Abrahams (1) and Purves-Stewart (77) in this connection, as is also benzedrine sulphate by Wilber, Mac Lean and Allen (4). Then the conditions of tetany, either the toxic or the calcium deficiency types are mentioned by the former group of authors as causing increased knee jerks. All of these things usually cause a generalized increase in response in all tendon reflexes and not just the knee jerks alone.

The increase in knee jerks due to irritation in the early

stages of a degenerative or toxic affection of the nervous system is mentioned by Purves-Stewart (77) and he gives the early stages of peripheral neuritis and subacute combined sclerosis as examples.

Next, we take up the most common cause for exaggerated knee jerks, namely, lesions of the pyramidal tracts above the third and fourth lumbar segments. The mechanism for this phenomenon is disputed, but the majority consider it as due to a release of the lower reflex centers in the cord from the inhibitory action of the cerebral cortex. Gordon (39) dissents from this view and claims it is due to an irritation of motor fibers which are undergoing degenerating changes.

Usually such exaggeration is accompanied by spasticity of the lower limb. However, as Wechsler says (105), increased knee jerks even when proven to be due to pyramidal tract lesions, are of no definite localizing significance, for the lesion may be anywhere from the cortical Betz cells down to the level where the pyramidal fibers go to the anterior horn cells of the third and fourth lumbar segments. However, he adds, that if the exaggerated tendon responses and pyramidal signs were lacking in the upper extremity, and the knee jerks were exaggerated, then the lesion could be localized in the dorsal (or thoracic) region of the cord. As mentioned by Gordon (39) and illustrated by Ranson (79) a unilateral exaggerated jerk or spastic limb on one side may be of greater localizing value - for if the lesion is above the decussation of

the cortico-spinal tracts in the medulla, it will be on the opposite side from the exaggerated knee jerk; while if the lesion disrupted the tract below the decussation it would be on the same side as the jerk. Thus, if besides the hyperactive knee jerk as evidence of pyramidal tract involvement, the patient had cerebral symptoms (e.g. coma followed by hemiplegia) it would be almost certain that the lesion, whatever its nature, would be on the side opposite the exaggerated knee jerk. This only shows how isolated facts give only partial information, and must be correlated with the general picture to be of greatest value. Since lesions anywhere in the cortico-spinal tracts, from the cortex down to the anterior horn cells can cause exaggerated knee jerks, the different types of lesions that might be associated with this finding are numerous. Common examples are: vascular accidents in the brain, multiple sclerosis, new growths, infections, and trauma in the spinal cord. As a general rule atrophy and paralysis are lacking in this upper motor neuron type of lesion, but voluntary motion may be greatly impaired. The only exception to the rule of lesions of the pyramidal tracts causing exaggerated tendon jerks below the point of disruption, is in complete transverse lesions of the spinal cord, and these are discussed separately later on.

Many times when an exaggerated knee jerk is organic in origin, a patellar clonus can be obtained. Monrad-Krohn (67) says that any tendon reflex may exhibit clonus when the reflex arc is hypersensitive, and that therefore clonus is simply an exaggerated

tendon reflex elicited in a different way. Most of the authors already mentioned in connection with exaggerated knee jerks, consider true clonus (i.e. maintained as long as pressure is used) to be found only in organic disease, but this is still disputed. Patellar clonus can be elicited in two ways: 1. (Monrad-Krohn(67)) The leg is extended and supported , and the patella is seized and pulled briskly downward, it then starts oscillating up and down due to clonic contraction of the quadriceps. 2. (P. Marie by Gordon (35)) Leg is extended partially and not supported in the middle, then patella is pushed down toward foot and held there, and the entire leg undergoes clonic flexion and extension.

These tests especially may require reinforcement to get sufficient relaxation.

The interpretation of patellar clonus, is then, the same as the exaggerated knee jerk.

There are some additional variations in response of the knee jerk to stimulation, and these are classed by Pritchard (76) as qualitative changes. Normally, when the patellar tendon is tapped, the antagonistic biceps muscles are felt to relax first, and then to contract to limit the excursion of the limbs. This phenomenon has been described by Sherrington (87) and discussed clinically by Monrad-Krohn (67) and others. This relaxation of the biceps is used experimentally to measure latent periods, and to detect slight responses which are unable to be seen. Clinically, the relaxation of biceps can rarely be felt, but the antagonistic con-

traction of them can be seen and felt. This phenomenon is disturbed in some of the qualitative variations of the knee jerk, especially in the knee jerk in cerebellar diseases. Gordon Holmes (49) and Pritchard (76) state that in unilateral cerebellar lesions there is sometimes an ipselateral diminution of tendon reflexes, but more frequently, there is an ipselateral hypotonia of the musculature resulting in a lessening of the antagonists' contractions and therefore a greater excursion of the knee jerk. This response with greater excursion, and a to and fro oscillation of the limb is termed a "pendular" knee jerk.

Another variation quite similar to this one above is the "sustained" knee jerk frequently found in chorea. This is described by Grinker (42) and Abrahams (1), who say that in chorea the leg has a tendency to be "hung up" momentarily in extension, and then to drop back in a pendular manner.

Another qualitative change in the knee jerk response is just the opposite of the free and pendular movements already mentioned. This change is frequently seen in paralysis agitans and hysteria (Monrad-Krohn (67), Grinker (42) and Pritchard (76)), where there is an increased muscular tone and a failure of the antagonists to relax when the knee jerk is elicited, thus giving a very short excursion of the leg.

P. Marie as quoted by Gordon (38) has described under the name contralateral reflex, the phenomenon of extension of the opposite leg as well as the one on the side of patellar stimulation.

This is usually found where all the tendon reflexes are exaggerated, and has the same meaning as an exaggerated knee jerk, he says. This same author, and also Grinker (42) state that in spastic legs the knee jerk elicited from one side may cause a contralateral adductor contraction. They explain this on the basis that because of spasticity, when the knee jerk occurs, it so tilts the pelvis as to stretch the adductor muscles of the opposite side and so elicited a jerk from them.

Besides these common variations in the knee jerk, the literature contains many studies on changes in knee jerk response in various systemic conditions of body and mind. While most of these are interesting only from the physiological standpoint, some have clinical significance. For instance, Travis and Dorsey have written two articles (96) (97) dealing (a) with the patellar tendon reflex time in psychiatric and neurologic cases and (b) the effect of alcohol on the knee jerk. These two articles were based on experimental work conducted to determine in the central nervous system, the relation between the highest and the lowest order of development. Thus, they found that alcohol acted on the higher centers as a depressant, first and last, and that this allowed the lower centers to escape domination and therefore to enjoy freer expression. This resulted in a more rapid tendon reflex time. When the diseased nervous system was likewise tapped, they found similar effects, namely, that in cases of stupor, muteness, etc. which shows arrested function of the higher levels, the peripheral reflex arc

has striking freedom of expression and when a case shows hyperfunction of cerebral or cortical levels, the lower arc is definitely depressed. With improvement of either case, the reflex time likewise approached the normal. In this work they concluded that it would seem that the transcortical neurons or highest levels are a definite functional part of the peripheral arcs, or lower levels.

Strecker and Hughes (92) made studies on patellar reflexes in psychoses in 1936, and they came to some rather different conclusions as regards to the extent of response, and response to reinforcement. They found that in manic-depressive, depressed, and involutional patients, showing actual symptoms of depression, the response to knee jerk stimulation was large, and that as the mental symptoms subsided somewhat, the size of the response decreased. In hypomanic patients, they found that the responses were unpredictable, and in the agitated and depressed groups together that they were not able to reinforce a maximal patellar reflex response. This they said was probably due to the reflex characteristic of occlusion, (already referred to in this paper). In the schizophrenic patients, the responses were found to be similar to those in the control group of normal individuals.

A very unique article is that by N. D. Royle (83) on the knee jerk as an indication for sympathetic ramisection. He says that in a number of vasospastic diseases, the deep reflexes are increased in excitability, and have a prolonged relaxation time

with few or no oscillations when coming to rest after elicitation. This, he says, occurs in Raynaud's disease, Hirshsprung's disease and in some cases of Buerger's disease. In the first two, hypertonia has been present in every case examined by the author, and he says that the hypertonic knee jerk as described is an indication that operation on the sympathetic trunk will definitely aid the patient, and that after operations the hypertonicity of the knee jerk is reduced markedly. He gives no other information, quotes no data, nor gives any explanation for the phenomenon described.

If all of the points described, and all the cautions to be observed could be recalled at the time of elicitation and interpretation of the simple knee jerk, that test would be of far greater clinical value than it is in its usual mode of employment. It would then play a very definite and important role in the examination of the nervous system, and when properly correlated with other findings it would give almost as much information to the clinician as it has to the physiologist in his experimental work.

Other Reflexes Elicited Around the Knee Joint.

For the sake of completeness, one or two rather little known reflexes that come under this heading, will be mentioned just as found in the few articles and textbooks that discuss them. In general they are of slight clinical value either because they are difficult to elicit, or because their interpretation is too much in dispute for them to be of definite value.

Wechsler (105) mentions the flexion reflex of the leg as serving to test the integrity of the reflex arc at the level of the fourth and fifth lumbar and first and second sacral segments, by way of the sciatic nerve. To elicit this the leg is semiflexed at the knee, abandonment is obtained by partially supporting distal end of limb and the tendons of the semimembranosus and semitendinosus muscles are tapped with a percussion hammer. Normally, a flexion of the leg, or at least a contraction of those muscles, is the response to be expected. The only interpretation discussed deals with their presence, which is normal, or their absence which can be used as additional evidence for a lesion in the reflex arc at that level, when such a lesion has been suggested by other findings in the clinical picture or neurological examination.

In the discussion of the knee jerk there was mentioned one variation, the fact that occasionally a contraction of the adductor muscles of the same or opposite limb was noticeable. Adductor responses have been known for a long time, but the literature shows

that only a few men have studied its clinical aspects to any degree. Inman (54) says that the adductor responses were first described by Pierre Marie, and later exploited by Lewandowsky.

Myerson (71) in 1916 made quite an exhaustive study of the phenomenon. His method of study and elicitation of the reflex was as follows: He had the patient supine, with the legs extended and rotated slightly outward in a normal, comfortable position, with the adductors relaxed. Then using a reflex hammer and a force just below that of causing pain, he tapped these points; medial and lateral surfaces of the knee joint, internal and external maleolus, sole of the foot near the transverse arch, achilles tendon, and the anterior-superior iliac spine. Then he sat the patient up and stimulated patellar tendon in the usual manner. He observed three groups of responses; the first where the homolateral adductor muscles were more active than the contralateral; second, the contralateral response more active than the homolateral and third, where the predominence of response alternated, or was mixed. His interpretation and conclusion of this response was: "1. Adductor responses are present in health as a mild and occasional homolateral or contralateral response from any of the above sites. 2. Fatigue diminishes them to the point of abolition. 3. The appearance of a persistent contralateral adductor response, especially from the patellar tendon, external condyle, or anterior-superior spine, is a phenomenon of disease and this disease is

usually organic in nature. 4. The side responding seems to be closely associated with its knee jerk, but has no relation to the knee jerk response of the side stimulated. 5. There is no relation of adductor responses to the ankle jerk."

His explanation for the response was that it was an increased tonicity of the muscle group due to a central lesion releasing central inhibition and making the group hyperactive and so giving an adductor contraction.

Four years later, Inman (54), in a study on pathological reflexes says that the crossed adductor response serves to draw attention to beginning pyramidal tract disturbances. He claims that it occurs early in central affections, especially in beginning anteriosclerotic changes in the cord and brain; also, that it may be present as evidence of past inflammatory changes in the central nervous system or meninges. He said that the sign could be obtained only by percussion of the patellar tendon, or the subcutaneous bony structures around the knee joint.

Both Inman and Myerson point out that the true adductor muscle response is not to be confused with the mechanical agitation of the adductors as mentioned previously in the knee jerk discussion where Grinker (42) described a contralateral adductor response which was sometimes obtained while eliciting the simple knee jerk in certain spastic legs. This latter response, as Grinker says, is due to mechanical tilting of pelvis when any tendon jerk is elicited in a spastic lower limb.

The more recent texts just mention the adductor responses as sometimes occurring when the knee jerk is stimulated, but they attach no individual significance to the phenomenon, and this holds true also for the articles that take the time to even mention the response. From this I would conclude that this test has been, as have so many obscure tendon jerks, almost discarded clinically because of its lack of specific localizing significance, and because other signs give more definite information on the same type of lesion that it designated.

Deep Reflexes Elicited From the Region of the Foot and Ankle

This group is generally believed to be of less importance clinically than that of the upper leg, and with the exception of the ankle jerk, perhaps that is true, but, as in all reflexes, the additional information furnished by this group, may, when fitted into the general picture of the neurological examination, lead to a definite diagnosis, even though that information alone was quite without value.

The ankle jerk is quite similar to the knee jerk, especially as concerns the theory for the explanation of its production, and the physiological concepts involved in its elicitation and interpretation, but it is different in its gross anatomical connections and therefore in the clinical interpretation of its response. The reflex arc in the case of the ankle jerk has its center in the first and second sacral segments of the cord (77) (79), and it is this that gives it its few differences when compared with the knee jerk. Here, after the end organs receive the impulse, it is carried up the afferent portion of the arc in the sensory fibers of the tibial nerve, on up the thigh in the sciatic and the posterior nerve roots of the first and second sacral segments. The peripheral efferent arc begins in the anterior roots of that segment and go out the same nerve trunks to terminate in the gastrocnemius and soleus muscles. (41)(79). While both

of these muscles are usually said to be involved in the production of the ankle jerk, Purves-Stewart (77) quotes Weir Mitchell as saying that it is only the contraction of the soleus and not the gastrocnemius muscle, for the latter is relaxed completely in the usual position for eliciting the ankle jerk.

Since the physiological and basic neurological concepts involved in the ankle jerk are exactly the same as those in the knee jerk, I will not repeat the work done along that line but will proceed to the method of eliciting the reflex. The best position for the patient to be in, according to Gordon (39) and Monrad-Krohn (67) is on the knees on a padded chair, but if confined to bed, the feet may be externally rotated to outline the achilles tendon. Usually it is necessary for the examiner to slightly dorsiflex the foot and hold it lightly in that position while striking the tendo-achilles with a percussion hammer or some similar object. The same precautions as regards relaxation, and care in tapping only the tendon where it crosses the "hollow" are to be observed here as in the knee jerk. A short sharp blow to this area normally results in a contraction of the calf muscles, and a certain degree of plantar flexion of the foot.

The interpretation of the response to stimulation of the ankle jerk concerns only a few variations. These are, absent jerks, hyperactive jerks and ankle clonus. These variations from the normal are discussed by nearly all general articles and texts on tendon reflexes, but I quote especially from Goodhart (33), Gordon

(39) Pollock (75) Purves-Stewart (77) Monrad-Krohn (67) and Elliott (22).

Before saying definitely that the ankle jerk is absent, the methods of reinforcement must be tried just as outlined in the case of the knee jerk. Once it is decided that the ankle jerk is absent, what are the possible causes for its absence? In this area factors outside the nervous system often produce circumstances that abolish the ankle jerk. For example, severe edema, arthritic or post-traumatic deformities of foot and ankle, congenital deformities and the like may so alter the structures of foot and ankle as to abolish the normal ankle jerk on one or both sides. Naturally, such conditions offer no real diagnostic problem since the cause can be seen grossly.

The two ankle jerks may be abolished by more or less general body disturbances which may also affect other reflexes. Thus in polyneuritis from any toxic or infectious cause; in deep coma or narcosis from any cause; extreme debilitation; general amyotonia; increased intracranial pressure (especially in tumors of the posterior fossa); in complete transverse lesions of the cord; and in the so-called Adie syndrome of tonic pupils and absent tendon jerks (latter two to be discussed later), the ankle jerks may be lost. These conditions usually present such a striking clinical picture that the diagnosis is made on other signs and symptoms, and the loss of the ankle jerk is merely incidental and certainly not of definite diagnostic significance.

When, however, the ankle jerk on one or both sides is found absent with no such general explanation, the interpretation is of much greater significance. Purves-Stewart (77) and Gordon (39) consider the ankle jerk to be equal to the knee jerk in diagnostic significance. They say that abolition of the jerk is a precocious sign of tabes and that often it is lost before the knee jerk is, depending on what level the degeneration of the posterior columns occurs at first. Compression or destruction of the nerves of the cauda equina will cause abolition of the ankle jerk too. Injuries to any portion of the reflex arc, from any cause, will abolish the jerk. Thus division or injury of the peripheral nerves to the calf muscles (see anatomical discussion), a neuritis, especially sciatic neuritis, as well as pathological processes at the level of the first and second sacral segments of the cord will cause loss of the ankle jerk. Therefore, in acute and chronic poliomyelitis, new growths and in trauma to this level, the loss of ankle jerks will be an important localizing sign. Monrad-Krohn (67) states that the loss of the ankle jerk in sciatica comes on a few days after the onset, and may persist long after the pain has gone away, or even permanently. According to Goodhart (33), loss of the ankle jerk in sciatica means a true neuritis and not a neuralgia. In addition to these causes for absent ankle jerks, the same things as given under the knee jerk may abolish the ankle jerk if the pathology happens to

involve the level of its reflex center.

Exaggeration of the ankle jerk is quite common both in organic and functional conditions of the nervous system. The causes and the interpretation of exaggerated ankle jerks are exactly the same as in the exaggerated knee jerk, and so will not be repeated here. However, severe exaggeration resulting in ankle clonus is even more common than patellar clonus, and so a few additional points will be mentioned about it and about clonus in general. As has been stated before, clonus is nothing but an exaggerated tendon jerk elicited in a different way. Any of the deep reflexes may become clonic. Especially in cases of suspected organic lesions of the pyramidal tracts must we look for clonus. "Clonus (77) is a rhythmic series of muscular contractions produced by a sudden passive stretching of a tendon, the clonus continuing as long as the tension on the tendon is maintained." Gordon's method of bringing out clonus in the ankle is to have the patient lying down. Then patient puts relaxed leg on palm of examiner's left hand, and the examiner uses right hand to grasp the ball of the foot and bring in first down (plantar flexion of foot) and then abruptly upwards (dorsiflex foot) and hold it there with moderate pressure. The result in cases showing clonus is a frequently repeated to and fro jerking of the foot from dorsiflexion to plantar flexion and back. This may be limited to a few vibrations or may continue as long as pressure is maintained. According to Gordon (39) and Russell (84) this reflex

usually accompanies an exaggerated patellar tendon reflex, and like the latter, it is the clinical expression of an irritation or a lesion of the pyramidal tracts, either in its spinal (ipselateral) or cerebral (contralateral) portions. Many, they say, feel that it occurs only in organic disease of the nervous system, but to be so interpreted, the clonus must be marked and prolonged, for a brief clonus may occur in hysteria. Monrad-Krohn (67) specifically states that there is a way to differentiate the two types. He says that true clonus due to a pyramidal tract lesion is inhibited by plantar flexion of the big toe, such being induced by pinching the skin, or by some other noxious stimulus; while a functional clonus is very likely to continue even if great toe is plantar flexed.

The mechanism of production of ankle clonus, like the patellar clonus is disputed; some saying it is due to irritative lesions of pyramidal tracts; others claiming that it is due to degeneration occurring in those tracts and still others say that it occurs only when pyramidal tracts are destroyed at some level, thus releasing the lower segments from cortical inhibition.

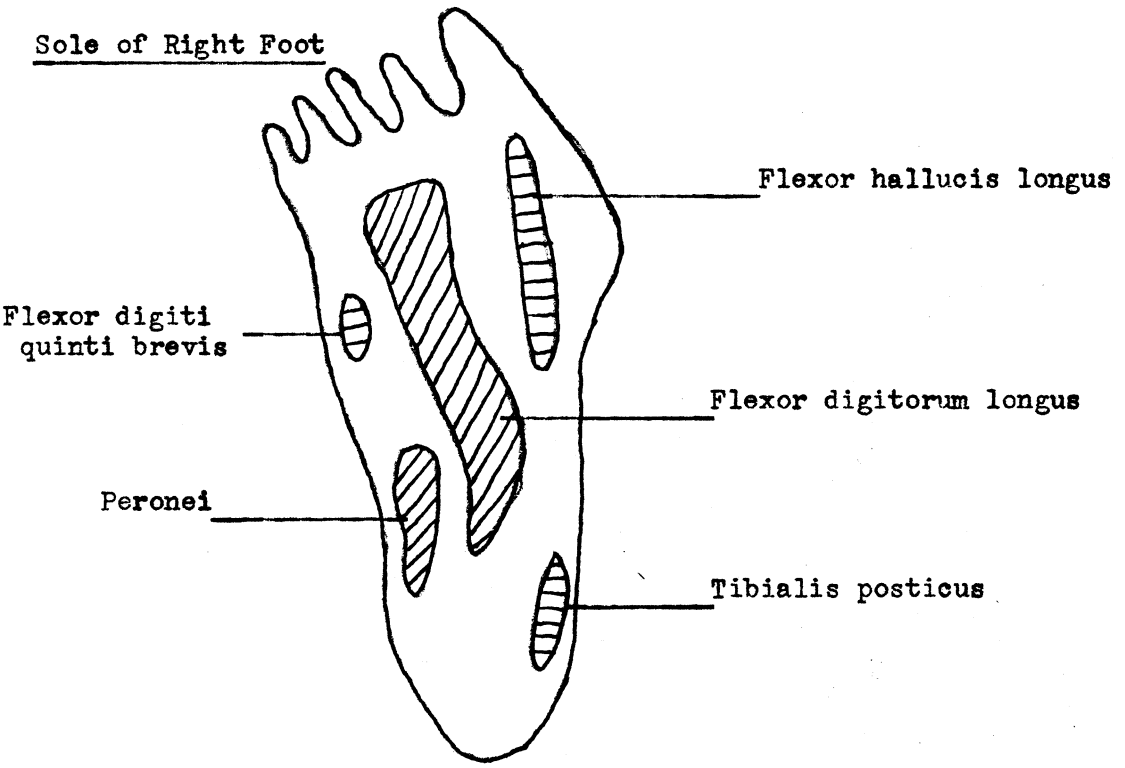
While clonus will be produced by any lesion that destroys the pyramidal tracts, it is especially prominent in advanced multiple sclerosis, and when clonus is found, the nature of the lesion should be classified if possible, and here again we can see how the isolated neurological finding, must be interpreted in the light of the entire examination and history, to derive its full value and

meaning.

As a rather isolated bit of work on the ankle jerk, I present a phenomenon discussed by Boveri (10) in 1919. He describes a sign as follows: "With patient prone, the leg is flexed so that it is at right angles to the thigh, and feet are in the air. Then the achilles tendon is struck as in eliciting the ankle jerk. In positive cases, the ankle jerk is absent, and there results a flexion of the second phalanx of the great toe". The interpretation of this sign he says is not entirely known, and I found no other work on it. It was seen especially in war injuries associated with the sciatic and external popliteal nerves, or in injuries to the spinal cord roots making up those nerves. It doesn't always occur in absent ankle jerks, but it never occurs when the jerks are present, nor in normal persons. In general, he thinks, it shows a partial lesion of the sciatic nerve and indicates a good prognosis in sciatic injuries. Likewise, in cord root involvement, its appearance during course of convalescence he regarded as a good sign.

Leaving the ankle jerk, and proceeding to the foot, we find that tendon jerks have been elicited in nearly all the muscles of that region, and that while the diagnostic value of these tendon jerks is not generally accepted, it may in some cases aid materially in localizing certain lesions. Essentially, all this work has been done, or at least reported by one man, Weingrow (107)(109) (108). In 1932 (107) in his article on plantar tendon reflexes he presents five new tendon reflexes of that area, with the intro-

ductory statement that there is only one way in which a clear physiological interpretation of reflexes in this area can be accomplished, and that is by tapping single tendons as they cross hollows, and thus evoke a myotatic response in the muscle. In all attempts, there must be a distinct differentiation between plantar skin and plantar tendon reflexes. His new reflexes and the points from which they are elicited are best shown in the diagram that he gives and which is copied below:



He says that normally these tendon responses can be obtained except in cases of obvious deformity of the feet. The responses normally are: 1. Flexor hallucis longus tendon gives plantar flexion of great toe and the segmental center is L.5, S.1 and 2.

2. Flexor digitorum longus gives plantar flexion of outer four toes and foot. (L.5, S.1 and 2). 3. Peronei, gives plantar flexion of foot (L.4 and 5, S.1 and 2). 4. Tibialis posticus gives plantar flexion of foot (S.1 and 2). 5. Flexor digiti quinti brevis gives plantar flexion of small toe (S.1 and 2). Since the reflex center for some of these is Lumbar 4 and 5 and the ankle jerk is Sacral 1 and 2, he says that these will aid in more definite focal diagnosis of lesions that abolish the normal response. He says, that they are abolished most frequently in sciatica and in central nervous system syphilis.

In 1935 (109) he adds to those already given, four new tendon reflexes elicited from the side of the foot. These are produced by tapping the tendons of the following muscles: abductor hallucis, abductor digiti quinti, extensor digitorum longus, and the extensor hallucis longus. The last two have their centers in the cord at the level of Lumbar 4 and 5, and are therefore two segments above the ankle jerk center. He says that all of these can be obtained with the foot in any position, and the response normally obtained is the same as that indicated by the name of the muscle. These reflexes are said to be diminished or absent in sciatic neuritis, also in central nervous system syphilis in many cases (cases presented by the author in his article). Especially, early in this latter disease, are these reflexes likely to become unequal on the two sides.

Another reflex, elicited from the foot region is known as

the Bechterew-Mendel reflex. This sign, according to Inman (54) was described independently by Von Bechterew in 1902 and by Kurt Mendel in 1904. The sign, according to Inman (54), Monrad-Krohn (67), Weingrow (108) and others, is obtained by tapping the dorsum of the foot at the junction of the cuboid and outer metatarsal bones (just in front of the external meleolus). This deep reflex is supposed to be periosteal in origin and normally there results either a dorsiflexion of the small toes, or no movement at all. In cases of disease of the nervous system the response to percussion over this area is a plantar flexion of the toes (except where the flexor muscles are weak or paralyzed). These authors interpret this response as evidence of an organic lesion of the pyramidal tracts at any level of their existence. However, Weingrow in his series of cases in 1935 (109) reports that not once in pyramidal disease did tapping the dorsum of the foot cause plantar flexion of the toes, and consequently he thinks the sign is inconstant if not entirely lacking in such cases.

A reflex elicited from the foot and that is more generally accepted as valuable is the Rossolimo Sign. Goldflam (32) as quoted by Weingrow (108) considers this phenomenon to be "a pathological periosteal tendon joint reflex". Wechsler (105) also considers this to be not a skin but a tendon reflex. He says it is a pathological reflex and consists of plantar flexion of the toes on tapping the ball of the foot.

Weingrow (108) says there are four types of response eli-

cited pathologically by this maneuver: "1. Plantar flexion of the outer four toes alone, 2. Abduction of the big toe alone, 3. Fanning of the outer four toes alone, 4. Combinations of the above three types."

In general, the presence of this sign is said to be indicative of pyramidal tract disease, and Monrad-Krohn (67) claims that it certainly isn't proof of such a lesion. More specifically, Wechsler (105) says that extirpation experiments have shown that the reflex occurs in lesions of the pre-motor area, in close association with the fanning and grasp reflexes, and that the sign is especially useful when there is, by chance, a paralysis of the extensors of the great toe, thus rendering the Babinski equivocal.

Weingrow (108) cites one strenuous objection to the Rossolimo reflex. He says that one is using several different stimuli at one time, i.e. tapping skin, tapping or pinching tendons, stimulating the periosteum and indirectly stretching the achilles tendon by movement of the foot. Because of this, he says, parts of the pathological response can often be elicited in normal individuals. As a more satisfactory substitute, he offers in an article in 1933, the Heel Tap, which he says is a new sign of pyramidal tract disease consisting of fanning or plantar flexion of the toes, or any other movement of the toes following tapping of the heel. He says this occurs only in definitely advanced involvement of the pyramidal tracts. He does not say why his objections to the Rossolimo would not apply here also.

In concluding this section on the deep reflexes, I wish to mention one syndrome that may give absent tendon reflexes in the lower extremity. This is the so-called Adie syndrome of tonic pupils and absent tendon jerks. Specifically this is a benign, non-syphilitic Argyll-Robertson pupillary reaction, coupled with an absence of one or more tendon reflexes in the lower extremity. The tonic pupil was described in 1902 by Saenger (85) also by Strasburger (91), and although their records (according to Adie (3)) show some cases of areflexia they made no mention of this. The cases were usually described (3) as "probable congenital tabes", or "incipient tabes of congenital origin".

In 1921, Behr (9) described eight cases of pupilotonia and states that this abnormality alone did not justify the diagnosis of syphilis. Then in 1924 Moore (68) published his first paper on the "Non-Luetic Argyll-Robertson Pupil". Other papers in 1927 and 1931 by Morgan and Symonds (70)(3) associated the changes in the pupils with the absent reflexes and said that they were of unknown origin, but certainly were not luetic. Then in 1931-1932 articles by Moore (69), Gordon Holmes (50), and Adie (2) established the two as evidences of the same thing.

There are many forms which the syndrome may take, but most of them are based on ophthalmological details, however, it is well to keep in mind that one form shows the areflexia without the pupilotonia.

Adie says (3) that 80 - 85% of the cases showing both signs

are in females. It is entirely asymptomatic, picked up usually in a routine examination, is entirely benign and never becomes otherwise as far as is known. He says that most frequently one or both ankle jerks are lost, and that at least one ankle jerk is gone in any case that shows any reflex changes at all.

Adie gives no definite explanation for the loss of reflexes in his syndrome, but says it is not hereditary, and he cautiously suggests a possible endocrine origin for the syndrome.

The Superficial, or Cutaneous

Reflexes

The Plantar Reflex and the Babinski Sign.

This group of reflexes is considered by many authors to be of more clinical significance than are the deep or tendon reflexes. These too, however, must necessarily be correlated with the rest of the neurological examination, the general physical examination and the history, in order to assign them their correct value.

As an introduction to the cutaneous reflexes in general, I quote Wechsler (105) who says:

"Normally, all the deep and superficial reflexes can be elicited and are equal on both sides. If the reflexes are naturally sluggish or lively, the deep and superficial reflexes are equally sluggish or lively. Both the deep and superficial reflexes become significant of some pathologic disturbance if they are unequal on the two sides, or if there is a discrepancy between the deep and the superficials. Then, if some of the so-called pathological reflexes can be demonstrated in addition, a definite lesion may be inferred, but not the nature of it."

As has been stated before, Russell (84) considers the reflexes as the most valuable single method of neurological examination, and now we may add that he picks the plantar reflex as the most valuable single reflex. He says that other reflexes are of very limited application when compared to the large number of cases where alteration in the plantar response occurs, and so

because of the applicability of this reflex to such a wide variety of affections he gives it the title of the most valuable single reflex. He recognizes the fact, however, that certain affections can be diagnosed by signs that are of greater value in that one particular disease.

Although the plantar response to stimulation had certainly been noticed before the time of Babinski, it was with the advent of his discovery that most of the clinical studies of the reflex in normal and pathological cases were begun. Therefore, in beginning the study of plantar responses it is pertinent to refer to Babinski's original article in 1896. This article as translated by Hall (43) was entitled "On the Cutaneous Plantar Reflex in Certain Organic Affections of the Central Nervous System". The entire sign, and much about its method of elicitation were presented in a few sentences by Babinski: (43)

"In a certain number of cases of hemiplegia or crural monoplegia associated with an organic affection of the central nervous system I have observed a perturbation of the cutaneous plantar reflex of which the following is a brief description: On the healthy side, pricking the plantar surface of the foot provokes, as is usual in the normal state, flexion of the thigh on the pelvis, of the leg on the thigh, of the foot on the leg and of the toes on the metatarsus. On the paralyzed side a similar excitation gives rise also to a flexion of the thigh on the pelvis, the leg on the thigh and of the foot on the leg, but the toes, instead of flexing, ex-

ecute a movement of extension on the metatarsus. I have observed this disturbance in cases of recent hemiplegia of only a few days duration as well as in cases of spastic hemiplegia that had existed for several months; I have demonstrated it in patients who were incapable of voluntary movement of the toes, and also in subjects who could still execute voluntary movement of the toes; but, I must add that this disturbance is not constant. I have also observed in a number of cases of crural paraplegia due to an organic lesion of the cord a movement of extension of the toes following pricking the sole of the foot."

Since the time of the above article, a great deal of work on the plantar reflex and the Babinski sign has been done, on every phase of the subject.

We will begin by discussing the various theories advanced for explaining the presence of an extensor response in diseases of the pyramidal tract in man, and the normal flexion response in man.

Solomon (90) presents an early article in which he believes the phenomenon is explained on a phylogenetic background. He quotes Astawazaturof (6) and Edinger (21) in the building of the following basis for that theory: First, the Babinski sign is normally positive in infants; second, lower animals show extended position of the toes; third, a lesion in the pyramidal tracts is especially suitable for the appearance of any possible rudimentary phylogenetic sign because it is a relatively new part of the ner-

vous system. In the nervous system many changes in form and function have occurred in the course of evolution from the segmental to the type where cortical connections give that organ a controlling, directing and inhibiting influence over the lower spinal reflex centers, and by virtue of the pyramidal tracts the purely reflex activity of the cord has been lessened. In considering the evolution of the great toe from the grasping to the static function found in man we pass through various stages, two of which are most important. 1. The stage where only the four lesser toes were used for grasping, and the great toe extended. 2. The stage of baring the claws on the lesser four toes. Then in putting this all together, Solomon says that a pyramidal lesion releases the lower segments from cortical inhibition and a reversion to metameric activity occurs as evidenced by the extension of the big toe and flexion of the others (grasping) or of fanning of the toes (baring the claws).

This theory is also supported by other workers, especially by Rabiner and Keschner (78), who reached the following conclusions:

"1. The plantar reflex depends on the integrity of the final common motor pathway; an intact sensory component of the reflex arc; and the influences on these by the pyramidal and extrapyramidal systems.

2. Dorsiflexion of the big toe represents a reversion to the normal posture of the foot in all primates except man.

3. The adjustment to erect posture and to the change from aquatic and arboreal existence to a terrestrial one must be associated with a neural apparatus that also changes and this change is evidenced by the evolution of the pyramidal and extra-pyramidal systems.

4. This above statement is borne out by the preponderance of the pyramidal over the extrapyramidal tracts as we ascend the animal scale. (Diagrams from Sherrington (87) are given in the article to show the relative sizes of the pyramidal tracts in man, monkey and dog).

5. Removal of pyramidal influences in man, by any cause, will result in reversion to a dominant extrapyramidal system and gives dorsiflexion of the great toe.

Friedman (26) also expresses a favorable opinion towards the phylogenetic theory, and adds his belief that the infant has a positive Babinski because the pyramidal fibers are unmyelinated and that it becomes flexor in type as the child learns to walk and the tracts are myelinated.

Davidson's (19) is the most recent work on the theory of production of the plantar reflex and he thinks that none of the theories are adequate to explain the phenomenon. He quotes Rudolph (19) as being against the myelin theory since his own (Rudolph's) work has shown that subhuman primates with an intact myelinated pyramidal tract still have normally an extensor plantar response.

He then points out that at birth the infant has a flexor response, then during infancy it becomes extensor and remains so until the child walks, and this he says is ontongeny recapitulating phylogeny since phylogenetically the reflex goes from flexion in the mongoose to extension in the monkey to flexion in adult man.

Davidson also says (19) that French physiologists think the plantar response and its variations are caused by a difference in chronaxie between extensor and flexor muscles.

He concludes with his statement that all the theories are inadequate and says that to be of value any new hypotheses should explain:

1. Why the normal response in man is flexor, while in primates below man it is extensor.

2. Why during infancy the positive Babinski is often seen.

3. Why extension occurs in adults in disease of the pyramidal tracts, even when the extrapyramidal tracts are cut (McCouch 19).

Leaving the possible explanation of the normal and pathological plantar reflexes, we take up next the method of eliciting the plantar response.

Dosuzkov's article (20) discusses the plantar reflex in all its phases. Concerning the method of eliciting the reflex he points out that it must be examined when the patient is in the supine or dorsal reclining position, with the legs extended and the feet resting with a solid base under the heels. He says that

if the legs are flexed, no Babinski can be elicited. He says that a blunt needle or pin is the instrument of preference and definitely states that no object with a wide end (such as the handle of a percussion hammer) should be used because such an object necessitates heavy pressure and this disturbs tendons, periosteum and pulls or twists the skin covering a large area. This excessive stimulation often elicits a normal plantar flexion even when a neurological lesion is present. Conversely, a false positive result may be obtained if the skin over the metatarsal-phalangeal joint is stimulated excessively. The correct stimulus then, he says, is the light pressure of a pin being pulled slowly along the internal or external borders of the sole, from the heel to the elevations at the metatarsal-phalangeal joint.

Purves-Stewart (77) and Solomon (90)stress the fact that the feet should be well warmed before attempting to elicit the plantar response. Also, they claim the best position is with the knee and hip slightly flexed and the limb externally rotated so that the foot rests on its outer border. They say that this reflex too, often needs distraction in order to bring out definitely a difficult or equivocal response.

Weingrow (107), Davidson (19) and McKendree (107) say that the best site for stimulation is the outer border of the plantar region, and that the stimulus should be a painless scratching from heel to ball of foot.

Walshe (102) in his article on variations in form of re-

flex movements says: "Different degrees of spasticity as invoked by variation in posture, etc., cause different types or degrees of reflex response. Thus, certain reflexes may be brought out more clearly by knowledge of this factor, and lack of it may result in clinical "absence" of a reflex that is present but not marked." Applying this more specifically to the plantar response and the Babinski sign he says that, "The Babinski type of plantar response may be diminished or even abolished by rotation of the head to the paralyzed side in a hemiplegia. How often this occurs cannot be definitely said, but the fact that it ever occurs should make it worth while to rotate the subjects head to the opposite side whenever the Babinski is equivocal in form, or difficult of elicitation."

So much then for the method of obtaining the plantar reflex; now, what constitutes a normal plantar response and what are the abnormal variations of this response?

Dosuzkov (20) says that in general the plantar reflex can be divided into three parts; (a) the movement of the large joints (hip, knee, talo-crural); (b) the movement or contraction of the tensor fascia lata muscle and (c) the most important, the movement of the toes.

The first two can be described briefly because of their relative lack of importance as compared with the third. He says that the usual response of the large joints to plantar stimulation consists of flexion of the femur, and the tibia and fibula,

plus a dorsal flexion and internal rotation of the foot or the ankle. Such a response occurs constantly in only 35% of normal individuals and naturally requires a stronger stimulus than for the more common reaction of the toes. When such a response occurs it tends to obscure the more important movements of the toes. The reflex also occurs in functional and organic nervous diseases, but is not even constant there.

The reaction of the muscle (tensor fascia lata) of the thigh, Dosuzkov describes as a visible shortening of the muscle with increased tension of the fascia of the thigh. This stimulus needs to be a little stronger than the stimulus for the toe reaction and was found to be present in over 50% of healthy individuals. Absence of this reaction is often observed in hysteria and when combined with absent plantar flexion of the toes, is called Crocqs' combined plantar phenomenon. (observed in 60% of his cases of hysteria). Absent alone, it is occasionally found in pyramidal lesions, and in chronic epidemic encephalitis. An increase in the fascia lata response, he says, occurs in over 60% of central hemiplegias, in the hyperasthetic stage of poliomyelitis, and also less constantly in dementia praecox and hysteria.

Now as to the all important response of the toes in plantar stimulation.

Wechsler (105), Church and Peterson (17) and Jelliffe and White (19) say that flexion of all the toes is the normal response.

Purves-Stewart (77), McKendree (107) and Monrad-Krohn (66) say that flexion of the great toe alone is the normal response.

Davidson (19) et al, did their work in 1931 on 161 normal people under standard conditions of degree of stimulation, mode of stimulation, position of patient, etc., and came to the following conclusions:

"1. There is no one normal type of response in adults. In most cases, all the toes flex, but extension, even of the big toe is compatible with a normal motor system.

2. Isolated extension of the big toe, plus fanning of the other toes does not occur in normal persons. (Other forms of extension of the big toe with different movements of the other toes did occur in normal persons).

3. Position of the head, knee, or the use of distraction are not necessary or important.

4. Withdrawal of the whole foot means only that too strong a stimulation was used.

5. A pin stuck in ball of foot, is a noxious stimulus and was followed by flexion of all the toes, and so the Babinski is not a defense reflex."

They also found that most of their falsely positive responses of extension were encountered when the skin over the ball of the foot was stimulated.

Friedman (26) says that even extension of the great toe

is observed in health, when accompanied by dorsiflexion of the other toes.

Collier (18) doubts if the plantar response is ever completely absent during health, but this is disputed in many more recent texts.

Grossman as quoted by Dosuzkov (20) says that the plantar response is present in 90 - 98% of all healthy persons, and that the normal response is flexion and adduction of all the toes.

Since the normal plantar response is so much in dispute, it would naturally follow that there is much discussion as to what constitutes the most important abnormal response, i.e. the Babinski sign.

Certainly, in the translation of the original description by Babinski (as already given) he says that the sign is extension of the toes, and nothing more.

Davidson (19) says that it is isolated dorsiflexion of the big toe, with fanning of the other toes, but that it is really the fanning that is the Babinski sign.

Lomstadse (60) believes dorsiflexion of the big toe can be accompanied by plantar flexion of the other toes, and still be a positive Babinski.

McKendree (107) says that it is only the first movement following stimulation that is of any importance.

Wechsler (105) states that the response indicating the Babinski sign is fanning and plantar flexion of the lesser toes, with

extension of the great toe.

Purves-Stewart (77) says that it is only the first movement of the big toe that is significant, and that besides the extension of the great toe, and fanning of the lesser toes, one also notices that the response is quite slow, and is accompanied by a contraction of the hamstring muscles.

Dosuzkov (20) gives several forms of response that indicate a positive Babinski: 1. Dorsiflexion of all the toes. 2. Dorsiflexion of great toe and plantar flexion of the others (he says this is the classical type). 3. Extension when the external side of foot is stimulated, with a normal response on the inner side. 4. Inconstant types of extension and fanning.

Solomon (90) says that the usual Babinski consists of slow extension of the big toe and simultaneous flexion of the other four toes, but he says the response may be complete or incomplete. In the complete type, there is extension of all toes, plus the fanning phenomenon. The incomplete shows extension of the big toe with the other toes flexing or remaining stationary. He says that even a third type is occasionally indicative of a positive Babinski, i.e., where there is flexion of the four lesser toes and no movement of the great toe.

Elliott (22) considers the true sign to be a slow extension of the great toe, with fanning of the other toes.

Monrad-Krohn (66) points out that the plantar response is prolonged and all the movements last longer in the Babinski

sign than in the normal reflex plantar response.

From the above statements we can see that the actual response indicating a true Babinski sign varies considerably according to different authorities, and this must mean that more than one type of response is indicative of the upper motor neuron lesion for which Babinski described his sign, but in general, the variations are minor and probably all would be found in an examination of a large number of patients with organic disease of the pyramidal tracts.

We have now discussed the theory for the production of the Babinski sign, the methods of eliciting it, and the various responses that constitute a positive Babinski; now, if that sign is present, what does it mean to the examiner? In general,(Purves-Stewart (77)) the normal plantar reflex occurs only when the reflex arc is intact, and when in addition, the lower arc is in connection with the cerebral cortex through the pyramidal tract. If the pyramidal tract is interrupted at any point, and the lower arc is not broken, the response to plantar stimulation is the Babinski sign. Such a response alone, however, has no localizing value since the pyramidal lesion may be anywhere from the cortical Betz cells to the level of the first and second sacral segments where the pyramidal fibers go to the anterior horn cells. (Wechsler (105)). If, along with this, the upper motor neuron tests of the upper extremity are normal, the lesion could be said to be below the first

thoracic segment of the cord. Dosuzkov (20) says too that a positive Babinski almost always means a lesion of the pyramidal tracts and lists many specific entities that may give it. Among these are: compressions, inflammations of the spinal cord, syringomyelia, multiple sclerosis, amyatrophic lateral sclerosis, hemiplegias of all types, etc. Also, the sign may be present, he says, in meningitis, cerebral tumors and chronic epidemic encephalitis. He says it has been found positive in 15% of his cases of delirium tremens, and dementia praecox and in 24% of his series of genuine epilepsy; all of these latter groups showing no other evidence of pyramidal involvement. He says the Babinski is found as a temporary occurrence in deep normal sleep, chloroform and scopalamine narcosis, and during or after epileptic paroxysms. In none of these, however, can temporary inactivity of the pyramidal tracts be ruled out and so the sign still retains its general interpretation.

Dosuzkov then lists some cases where the Babinski occurred almost certainly in non-pyramidal and extrapyramidal entities: 1. The psudo-Babinski of athetosis. 2. In a rare case of a bilateral lesion of the substantia nigra. 3. And in any case where the flexors of the toes are paralyzed or weaker than the extensors so that any movement has to be extensor in type (e.g. myopathies, neuritides, poliomyelitis, radiculitis and peripheral nerve lesions, all of which may cause paralysis or weakness of flexors of the toes).

He then concludes his discussion of the interpretation of

a positive Babinski sign with a summary of the four types which may occur even in the presence of a normal pyramidal system:

"1. The physiological Babinski which is encountered in children, even up to three or four years of age. 2. The peripheral Babinski, resulting from local atrophy of the flexors, nerve lesions, or anterior horn cell lesions. 3. The Pseudo-Babinski of chorea and athetosis. 4. pseudo-pathological Babinski which is the response occurring when too strong a stimulus is used and there is a resulting extension response plus movements in the large joints of the limb.

Elliott (22) says that a positive Babinski obtained after infancy indicates depression in function of the pyramidal tracts. He adds:

"It is a valuable aid to the neurologists armanentarium, for experience has shown that it is never produced by hysteria or neurasthenia and therefore indicates that something more than a so-called functional disorder is present. Before this, it sometimes took weeks to prove that a paralysis was due to hysteria. It is especially useful in the early diagnosis of multiple sclerosis, where the transitory symptoms suggest a hysteria."

Collier, (18) in an early article confirming the investigations of Babinski, says that the sign occurs in almost all lesions of the pyramidal tract but that it may occur in sleep.

Monrad-Krohn (67) says too that the usual meaning is

a lesion of the pyramidal tracts but adds that in evaluating this sign, the motor power of the big toe must always be tested, and paralysis of the flexors or anesthesia of the sole must be searched for. He says too, that the absence of a Babinski doesn't prove that the pyramidal tracts are free from disease, for he gives a case (66) of amyatrophic lateral sclerosis where no Babinski was obtainable, yet autopsy proved pyramidal lesions.

Besides these more common interpretations of the Babinski sign, there has been much written about the presence or absence of the sign in many conditions of the body. Some may have real significance, but many are in dispute, or are not constant enough to be of any diagnostic value. The presence of the Babinski in many comatose states has been alluded to previously. Purves-Stewart (77) says that a bilateral Babinski may occur temporarily in post-epileptic, and in uremic coma, also in the narcosis of acute morphine poisoning. Newman (73) reports a case of a positive Babinski in hypoglycemia. Here, the patient, a diabetic, was given an overdose of insulin. Hypoglycemia was proven by blood sugar and urine examinations. The plantar response was normal before and after the attack, but was <u>extensor</u> in type during the period of hypoglycemia. Similar cases are reported by Hart and Bond (44).

Hawthorne (45)(73) studied the plantar response in all types of coma and while he recognizes the Babinski as an indication of pyramidal disease, his studies forced him to name <u>coma</u> as another

very common cause of a positive Babinski. He says that in coma where the Babinski is negative, the trouble is that not the correct degree of unconsciousness is present, just as not all types of sleep will produce a positive Babinski.

Tournay (94) reports on the incidence of a positive Babinski in Jacksonian epilepsy. He finds that it is very variable, and believes it occurs only when the convulsion involves that part of the motor cortex controlling the legs, and that the mechanism is by exhaustion of the involved motor cortex.

Cases have been reported of a positive Babinski without pyramidal tract lesions or coma, but in two reported by Rouquier and Couretas (82)(82) they were due to atrophy of the flexors of the toes (a condition already discussed as a pseudo-Babinski). However, Fox (25) reports a case of a British marine officer who would have a positive Babinski one day and negative the next, or would have a negative response in the morning and a positive at night when fatigued. He was under observation and was never known to have any organic nervous disease, but neither was it proven that he did not have a disseminated sclerosis.

Kennard and Fulton (56) tried to determine a definite localizing value for the Babinski in its various forms, when associated with other neurological signs. They used extirpation experiments on animals. One of their conclusions was:

"That spasticity, forced grasping, the Rossolimo reflex, and

fanning of the toes points to lesions involving the premotor area or its projection system from the cortex, while the simple extensor Babinski without fanning plus flaccidity and depression of tendon reflexes suggests a lesion restricted to the pyramidal tracts."

Gould (40) in studying fifty cases of tumors of the posterior cranial fossa, says that 74% had more marked signs of pyramidal tract involvement on one side of the body, but that this was usually a hyperreflexia and only rarely a positive Babinski.

This concludes the discussion of the most important abnormal variation in the plantar response, namely, the Babinski sign.

The other abnormal responses to plantar stimulation are of less clinical importance, but if observed, they may be of diagnostic value in some cases.

The first of these lesser known abnormal responses to plantar stimulation is simple diminution in the amount of response. This is said to occur according to Dosuzkov (20) when the reaction doesn't occur in all the toes, especially when the great toe doesn't react. This diminution occurred in 12% of apparently normal subjects in his series, it also occurred as a rule in hysteria, in lesions of the reflex arc at that level (neuritis and radiculitis), often in epilepsy, and in long standing hemiplegias and paraplegias.

Collier (18) says that the plantar reflex was entirely

absent in 20% of his cases of tabes dorsalis.

Dosuzkov then lists another quantitative change in the plantar response, an increased normal response. This is said to occur when the first phalanx of the toes takes the chief part in the movement and thus gives a clawed aspect. He has seen such a reaction in high pyramidal lesions, in hemiplegias in children, in frontal lobe brain tumors, previous to epileptic paraxysms, and in some schizophrenia cases. Other changes, classed as qualitative changes by Dosuzkov are given as follows:

1. Limitations of the normal plantar response to one toe only (especially the fifth or first). This was first described by Bastian in 1890 in a transverse lesion of the cord. 2. Isolated abduction of the fifth toe as described by Puusepp, is said to occur in a great variety of neurological disorders and is therefore of no diagnostic significance. 3. The contralateral reflex, as caused by plantar stimulation. This may be either a flexion or an extension response. Contralateral plantar flexion of the toes produced by stroking the opposite plantar skin occurs in 50% of all cases of central hemiplegia, and occasionally in transverse lesions of the cord. Contralateral extension is rarely noticed, but may occur in hemiplegia, syringomyelia,multiple sclerosis and progressive muscular paralysis. It appears on the ipselateral side of the body by stimulation of the sole of the foot on the healthy side.

The contralateral plantar reflex is discussed by Gordon

(38) too. He studied 40 hemiplegias from various causes and concluded that it was of most value in the intermittent hemiplegias where a direct Babinski can't be obtained, but that the organic nature of the thing could be demonstrated by contralateral stimulation with resulting extension on the involved side.

These are the most of the clinical variations encountered in stimulation of the plantar skin, and it can be seen that the introductory statement proclaiming it the most valuable reflex on the basis of its wide applicability is certainly justified.

Other Cutaneous Reflexes

Before discussing the work done by various men on other cutaneous reflexes in the lower extremity, it may be wise to insert a statement by Grinker (42) which may somewhat modulate the enthusiastic claims that support the majority of these reflexes. He says:

"Since the discovery of the plantar reflex by Babinski, many neurologists have tried to attain fame by having their names attached to a certain reflexogenetic point. There are now only a few spots left open for future investigators. . . . . The plantar reflexes and their variations, with multitudinous names are all reflexes of defense which may be obtained anywhere on the skin as high as the segmental innervation corresponding to the lesion. These reflexes are important only in that they tell us of the presence of an abnormal release of lower cord centers from higher control, and also, they help some in localization."

Because other authorities tend to agree in general with the above statement, these reflexes will be but briefly discussed, and in most cases will merely be described. No elaboration on detail, such as were given on the Knee Jerk and Babinski Sign will be attempted here.

Hermann Oppenheim described his sign in 1902. His original description of the sign is translated by Hall (43) as follows:

"If one draws the handle (or shaft) of a percussion hammer over the inner surface of the leg from the upper margin of the tibia downward, one sees in healthy persons either no movement at all in the foot and toes or else a plantar flexion of the toes. If the irritation is made strong enough, distinct plantar flexion of the toes is the rule, but sometimes it is necessary to divert the attention of the patient to obviate voluntary movements. Whereas in persons with the symptom complex of spastic hemiparesis, this manipulation causes a reflex movement of the muscles that extends the great toe and adducts or abducts the foot."

This sign indicates organic pyramidal tract disease, and has occasionally been reported when the Babinski is negative. Most authors consider it only as a modification of the Babinski sign, and when present it has the same significance as that better known sign.

In May, 1911, Chaddock read a paper before the St. Louis Neurological Society, concerning a new diagnostic sign. This was published later (15) and was supported by case reports of the same author later in the year(16). Chaddock says that the sign consists in irritating the outer side of the foot below the external moleolus. The degree of irritation needed is variable, in some cases the merest touch being sufficient, while in others a rather severe scratching is required. The response, when positive, consists of extension of one or more, or all of the toes with or

without fanning. He says it occurs in disease of the spinocortical reflex paths. He has found it alone, without a positive Babinski sign in general paralysis, skull fracture, transitory unilateral brain lesions, and old hemiplegias. He says it never occurs in peripheral lesions or in tabes.

Ingram (53) reviews cases studied in the neurological department of the Cincinnati Hospital, and corroborated the work of Chaddock. He agrees with the originator, that while the Chaddock sign doesn't replace the Babinski, it is equal to it, is more delicate, and therefore appears earlier and lasts longer than the Babinski. He claims it appears without the Babinski in mild pyramidal disease, and that the Babinski sign never occurred without the Chaddock.

Alfred Gordon (34) described in 1904 what is classified by him as a"pathological cutaneous reflex", better known as Gordon's Paradoxic Reflex. This sign is obtained by having patient either lying on his back, or sitting up with his feet resting on a stool in front of him. The feet are rotated externally to relax the muscles, and the examiner stands outside the leg and places his hand or hands so that the thenar and hypothenar eminences are on the inner surface of the tibia. Then his fingers make deep, steady and firm pressure on the middle or lower calf muscles to transmit pressure to the deep flexors of the leg. If the reflex is present, extension of the great

toe or all of the toes will be noticed. In this article and in all subsequent ones (37)(39) by Gordon on his reflex he states that the sign is valuable for the diagnosis at the beginning of an organic affection, and shows that the pyramidal tracts are being irritated. He says that while the Babinski is of extreme value in well established pyramidal lesions, the Gordon sign is indicative of a transient irritation, or of a slight or beginning lesion of the same tract. He says there is often an antagonistic action between his reflex and the Babinski sign, i.e. when one is present the other is absent or diminished. He found that his sign was always associated with exaggerated knee jerks. In 1911 (37) he presented three cases of proven anatomical irritative lesions, interfering with but not disrupting the pyramidal system, where the Gordon sign had been positive, and the Babinski absent or equivocal. His examination of 800 normal individuals, failed to show any with a positive Gordon sign.

The comment on this sign by other men, particularly Grinker (42), Wechsler (105), Hall (43) and Elliott (22)seems to be generally the same, namely, that while it adds another sign to the diagnostic armamentarium it certainly doesn't replace the Babinski sign even in the cases for which Gordon claimed it to be particularly valuable, and that really, it is only a modification of the Babinski phenomenon.

Lomtadse (60) in 1932 described a sign that he thinks

has definite localizing value. This sign when positive consists of flexion of the big toe, when pressure is made with the thumb on the outer surface of the tibial bone. He says other pyramidal signs have no localizing value, but that this sign means definitely a lesion in the cortico-capsular region and the middle cerebral artery, and that even if this is present without other symptoms or signs, it is well to warn patient to restrict activities of both a physical and a mental nature.

Purves-Stewart (77) describes an adductor foot reflex that was first reported by Hirshberg (47) in 1903 and later studied by Marie and Meige (62) in 1916. This reflex is elicited by stroking the inner border of the foot (not the sole) with a blunt object, from the base of the hallux toward the heel. The positive response consists of a contraction of the tibialis posticus muscle, which raises the inner border of the foot (adducts it) and slightly extends the ankle joint. They say this reflex can sometimes be elicited in superficial organic lesions of the motor cortex, in which the plantar reflex is irregular in form.

Weinberg (106) in 1937 reported a new way of eliciting dorsiflexion of the big toe without external stimulation. This was done by having the patient first plantar flex the foot, and then slowly dorsiflex the foot. In such a maneuver in cases of pyramidal disease, the big toe goes noticeably more dorsal than it does in normal cases. He says he has seen this occur in all

kinds of pyramidal disease, and he believes it appears earlier and lasts longer than any other modified Babinski tests.

Reflexes of Spinal Automatism, Associated Movements or Reflexes, and Some Neurological Signs that are not True Reflexes.

Reflexes of Spinal Automatism.

There have been several different names given to this group of reflexes by various authors; in addition to the one used as the title above, they have been termed defense reflexes, spinal reflexes, or merely pathological reflexes. Actually, some of the more common reflexes already studied could be put in this group, but strictly speaking, the group is limited to the reflexes that are given below.

Monrad-Krohn (67) describes these as reflexes which in the normal person are subjected to such inhibition that as a rule they cannot be elicited. In central motor lesions, and particularly in spinal lesions, however, they may be elicited with such ease, that they serve to diagnose some of those lesions. These reflexes are regarded by many as the clinical homologues of reflexes seen in decerebrate and spinal animals, as described by Sherrington (86). Since in man they are frequently seen in complete and incomplete cord lesions, this subject will be considered along with this group of reflexes.

The first of these reflexes is, the flexion reflex of the lower extremity, or the pathological shortening reflex. This is described by Walshe and Ross (103), Monrad-Krohn (67), Wechsler (105), Purves-Stewart (77) and others. A noxious stimulus applied to the distal part of the lower limb elicits a complex reflex movement consisting of flexion at hip and knee joints, dorsi-

flexion at the ankle joint. Normally, dorsiflexion at the ankle occurs only when the stimulus is applied to the sole of the foot, and when a stimulus applied elsewhere (especially on the dorsal aspect of the foot) elicits dorsiflexion at the ankle joint it is pathological and indicates a pyramidal lesion. Likewise, the response in normal persons, if present at all, is a quick one associated with flexion of the toes, while in central motor lesions it is slower,and is often accompanied by extension of the toes. This reflex takes two forms according to the degree of spinal injury. The uniphasic (flexion only) occurs in complete division of the cord and the biphasic (flexion followed by extension of the limb) occurring in incomplete lesions. In all, the upper limit of the reflexogenous zone often corresponds to the lower limit of the spinal lesion, and never extends above it; thus it aids in localization.

The second in this group of reflexes is described by the same men, the crossed extension reflex. This consists of homolateral flexion or shortening of the stimulated leg with contralateral extension of the other leg. This reflex is said to indicate an incomplete spinal lesion.

The third reflex is called the extensor thrust, and consists of active extension of the limb when the distal portion is pushed upward, the limb having been passively flexed first. This reflex,according to Riddoch (80) is never obtained in complete division of the cord.

The fourth reflex in this group as described by these same men is the well known mass reflex. This is a response to any noxious stimulus applied to the lower limbs and consists of (a) the flexion reflex as described previously (b) evacuation of the bladder (c) sweating from the cutaneous segments below the lesion. This is the typical reflex associated with spinal lesion and according to Oldberg (74) occurs after the period of shock, and before the onset of sepsis and destruction of irritability of the cord.

Robinson (81) says that the mass reflex is of definite thereputic value in gross lesions of the spinal cord where loss of voluntary control of the urinary bladder occurs. Its use prevents over distension and aids in the establishment of an automatic bladder.

Oldberg (74) gives a very complete discussion and review of transverse lesions of the cord in his recent article. He says that Bastian (8) in 1890 made the first contribution that refuted the theoretical idea that transection of the cord would result in exaggeration of reflexes in the lower limb. He stated that such a lesion would result in flaccidity of the limbs and absent tendon reflexes. This statement was supported by the work of Burns (12) in 1893 so that there was founded the Bastian-Burns law: "If there is a complete transverse lesion of the spinal cord cephalad to the lumbar enlargement, the tendon reflexes of the lower extremity are abolished". In 1902 Warrington (104) modified this law in that "When the disease is of a slowly progressive nature, the reflex

function of the cord may be retained".

All this work was enlarged on by Sherrington and Riddoch (80), and it is now recognised that a reasonably rapid production of a transverse lesion of the spinal cord in man will result in: 1. A stage of muscle flaccidity and complete loss of tendon and plantar reflexes. This stage lasts from one to several weeks. 2. A stage of reflex activity of a variable duration, depending on the occurrence of any toxic febrile state. 3. The final stage of inanition and sepsis.

In the matter of prognosis, Oldberg (74) states that the reflexes are of definite value in transverse lesions. His conclusion was:

"1. Paraplegia from any source (traumatic, neoplastic, or infectious) when associated with complete flaccidity and total absence of tendon reflexes, existing for more than a day or two in the traumatic type, and for more than a few hours in the other types, is an almost hopeless prognostic sign despite removal of the cause."

Walshe and Ross (103) say that in minor cord injuries, the reflexes of the lower extremity are almost all increased in activity.

Collier (18) states that he has found the positive Babinski sign to be the only reflex present in the lower limbs, after a total transverse lesion of the cord. But, Monrad-Krohn (67) says the plantar response is flexion in complete transverse lesions of the cord, and is a slower response than the normal.

The picture of cord injuries then, presents an important variation in reflex responses, as shown above, and must be kept in mind in the evaluation of responses, or lack of responses in both the tendon and cutaneous reflexes. The presence of those reflexes of spinal automatism presented above, will aid in the diagnosis of such a neurological disorder.

Associated Movements or Reflexes

This group of reflexes is quite different than the types discussed previously. The group contains many isolated phenomena described long ago by clinical neurologists in detecting simulation and malingering, and it is only by more recent workers that they have been classed as associated movements or reflexes.

The entire group has been studied especially by Brain(11) and also by Grinker (42), Monrad-Krohn (67) and Wechsler(105), and it is from W. R. Brain that most of this material has been obtained.

In general these movements are explained by the above writers as follows: certain voluntary movements have a tendency to be accompanied by other, involuntary movements called associated or synkinetic movements. Some that are normally present are, the pendular swinging of the arms in walking, and the facial contractions accompanying violent muscular exertion. In pathologic conditions, such associated movements may be lost or exaggerated, or new ones may appear.

(42) In extrapyramidal motor disease (paralysis agitans type) there is usually a marked diminution or loss of all associated movements. In pyramidal lesions we find many new associated movements occurring, and these may so disturb voluntary motion that there is serious motor impediment.

There are usually considered to be three classes or types

of these reflexes: 1. The generalized movements of ordinary pyramidal hemiplegia. These tend to produce a typical attitude of predilection, i.e. flexion and adduction in the upper limb, and extension in the lower limb. These are brought out by any kind of muscular effort, (e.g. squeezing observer's hand with the non-paralyzed hand). 2. Symmetrical associated movements. These are involuntary irritative movements in paretic limbs accompanying voluntary movements of the healthy limb. These are especially noticeable when the patient carries out quick movements. 3. Coordinated associated movements. These are involuntary movements of synergic muscle groups that accompany voluntary efforts in the paretic limb, (e.g. the Strumpell sign as listed below).

Brain (11) has described what he calls a quadrupedal extensor reflex, which consists of involuntary extension of flexed, paretic upper limbs when the patient bends far forward or begins to assume the position of our "ancestral" quadrupeds.

Peterson (17) says that most of these are observed in central pyramidal lesions where there is increased tonus or rigidity. He says they are less often elicited than other pathological reflexes, but occasionally are present when the others are not.

The opinions of most of these writers as to the value of this group is expressed by Brain (11) when he says that besides helping to diagnose the disease itself, it helps differentiate organic and non-organic paresis.

This use in detecting simulation and malingering is confined

especially to certain signs: 1. The trunk-thigh sign of Babinski. Here the patient lies on his back with the arms folded across the chest and he then is asked to sit up. A true paretic limb is involuntarily lifted up in such an effort, either alone, or to a greater height than the normal one. 2. Hoover's sign. Normally, with a person on his back, when one leg is lifted voluntarily, there is an involuntary counter-pressure downward of the other leg (noticed by examiner placing his hand under the patient's heel). This is accentuated in attempts to lift a really paretic limb; abolished in hysteria, and would be absent in the paralyzed limb itself if the healthy one were being elevated. 3. Strumpell's sign. This occurs in pyramidal hemiplegia. On voluntary flexion at the knee joint, there is an involuntary dorsiflexion and supination of the foot. 4. Souque's leg sign. Patient sitting in a chair is suddenly tipped backward; normally an involuntary extension of both legs occurs, but in paralysis agitans or in actual paralysis such an extension would not occur.

Some other ways of recognizing simulation and malingering as given by the above authors, are: 1. To bring in complicated instruments to confuse the patient. In organic lesions this does not matter, in the neurotic it makes them more enthusiastic and in malingerers it confuses them. 2. Using a dynometer for testing the motor power, instruct patient to press as hard as possible every ten seconds for thirty trials. Normally, the compression becomes weaker and weaker; if it doesn't become weaker, it shows

the patient was not using all his voluntary power to begin with. 3. If patient has a questionable paralysis, pick up the "paralyzed" leg then let it drop, telling him to let it down slowly to keep from injuring it. This often tricks a malingerer into exposing some voluntary power. 4. If the patient complains of inability to do certain things or make certain movements, turn him on his stomach on a narrow couch, ask him quickly in different terms to do the things he says he can't. This reversed position often confuses a malingerer. 5. Hurry the patient as he dresses or undresses, and observe carefully for slips in movement. 6. In feigned contractures, heavy counterweights quickly fatigue the malingerer and straighten the limb, while real contractures take hours to straighten.

Grinker (42) says that in neurology, the problem frequently confronts the examiner as to the genuineness of the patients complaints and symptoms. Especially in time of war, in compensation cases, or in psychogenic cases where illness serves to gain attention, or is a compensatory reaction to a guilty conscience or an inadequacy. In such cases, he says, careful study of a group of pathological reflexes called associated movements, will often aid in the correct diagnosis.

Neurological Signs That Are Not True Reflexes.

These signs are commonly used and certainly need to be included in any review of neurological signs and reflexes of the lower extremity. While most of these are not true reflexes as far as being similar to the knee jerk and plantar reflexes are concerned, they are reflexes if considered in the light of an earlier statement made that many neurologists consider all neuromuscular activity to be basically reflex in origin.

Of these, the Kernig Sign is probably the best known, and is used most frequently clinically. This sign, according to Hall (43) was described by Kernig in 1884 (57), in an article on "A Little Known Symptom of Meningitis". In it he said: (translated by Hall)

"In the majority of cases of meningitis, contractures are not present in the extremities while the patient is lying down, whereas if one tries to extend the knee while the patient remains sitting, one succeeds only to an angle of about 135 degrees. In cases in which the phenomenon is pronounced, a right angle is maintained. The phenomenon is so striking, and the difference between the entire absence of this contracture in the reclining position and its presence in the sitting position is so readily seen that it is worth while to pay particular attention to this symptom and to look for it in every case."

He continues the article by saying he had never found the

sign described previously in the literature, and also he describes the method of eliciting the sign with the patient recumbent, as is more commonly done at the present time.

The sign he considered not to be a reflex, but a phenomenon elicited by passive manipulation of the leg. The sign is said to be positive when the angle between the calf and thigh cannot be opened to one and one-half right angles (i.e. 135 degrees), when the other leg is kept down and fully extended. The sign points to meningeal irritation, however, in young children (67), the sign may be absent in meningitis, or may be caused by other types of infection outside the nervous system.

Wechsler (105) says the sign consists of pain and resistance on passive extension of the lower limb, and that it is due to involvement of the lumbosacral roots and meninges.

Another sign obtained in the lower extremity is that described by Lasegue, and published first by one of his pupils (Forst) in 1881. Hall (43) translates the original article which gives the method of eliciting and the interpretation of the sign. The patient is in the dorsal reclining position, and the examiner places hand under patient's heel and another on the knee; thus keeping limb extended he flexes thigh on the pelvis. Normally, only a sensation of pulling in the gluted region is experienced, while if the sign is positive, the movement can be carried only a short distance before there is severe pain in the sciatic notch. This sign, according to

Hall (43) is truly pathognomic of sciatic nerve disease, and he says that it is caused by pressure of the muscles of that region on the nerve at the point where it emerges from the sciatic notch.

Wechsler and Monrad-Krohn consider the pain due to stretching of the nerve. They say it is obtained in the same manner as the Kernig, and that the pain is along the course of the sciatic nerve. They consider the sign important in the diagnosis of pain in the leg, for in sciatic pain this movement is limited because of increased pain with movement, while in the pain due to tabes, there is an associated tabetic hypotonia that allows increased range of passive motion without increased pain.

In differentiating between sciatic pain, and hip joint disease and pain, a useful sign is that described by Patrick (43) (105). This is elicited with the patient supine in bed, the thigh is flexed and the ankle of that side is placed on the opposite extended leg as high up as possible (preferably above the patella). The flexed knee is now pressed downward and outward, thus causing abduction and external rotation at the hip joint. In hip disease, pain is elicited before the knee reaches the level of the bed. Wechsler (105) considers the sign useful also in detecting sacroiliac disease.

The last sign in this group is really not limited to structures in the lower extremity, but is closely associated with the signs already described and is of some value in diagnosis. This

is the sign, described by Romberg in 1840 (22) and given his name. It was described as one of the cardinal signs of tabes, along with absent knee jerks and fixed pupils. Monrad-Krohn (67) says it can be tested for in any patient that can stand unsupported. The patient is asked to stand, and put his feet close together, get steadied and then close his eyes. If a swaying occurs which is quite marked as compared with when the eyes were opened, the sign is positive. The degree of unsteadiness should be noticed, and any attempts to move feet a little to regain balance also noticed.

While Elliott (22) says the sign is indicative of spinal cord disease, usually associated with the posterior column degeneration of tabes, it may occur in other conditions, both within and outside the nervous system. But, if definitely positive and no mechanico-anatomical disturbance is present, one can almost say definitely that it is due to failure of the posterior columns to conduct impulses of deep sensibility upward to the brain.

When, according to Elliott (22), Gordon (39) and Monrad-Krohn (67), the sign appears to be positive in questionable neurotic individuals, having them do some other test at the same time (e.g. the finger-nose test) will usually render the Romberg negative in functional cases, while a true tabetic betrays his unsteadiness all the more.

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