Certain Nervous Symptoms and their Origin

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Introduction

It is intended in the following pages to enter somewhat fully into a discussion of the central process concerned in the production of certain nervous symptoms.

The theory advanced differs from any that I have read of in some points. It most resembles that advocated by Dr. Edward Living under the name of Herxheimer.

The view about to be advanced is simply that, nerve impulse when it has reached the brain may overflow and involve, according to its intensity and the condition of the centres, a larger or smaller cerebral tract; and that overflow is most liable to occur in those centres which are in physiological relation to the one first involved.

Part I contains arguments in favor of the above stated hypothesis.

In Part II, applications of the principles, laid down in the first part, are considered.
Part I
Chapter I

It must be admitted by all, that any explanation of many of the functional neuroses that can be at present given, must be at best hypothetical. Indeed the very attribute (functional) applied to them is a passive acknowledgment of the fact. By functional we mean in medical language that the disorder to which the term is applied, is as transient as the changes which produce it. At the same time no one who has studied physiology can believe that a deviation from health can occur in a living being without a corresponding tissue change—possibly molecular and incapable of being demonstrated by our present means of detection—but still as less a change.

A careful consideration of the physiology of the nervous system would lead us to believe that in the human body there is always present a certain amount of energy capable of being converted into nerve impulses. Moreover it
It seems rational to conclude that part of this force is in a form ready for use, if we may be allowed the term, but that another portion is latent and can only be prepared for use by the action of the highest nerve centre, the brain. Probably in the typical living being nerve energy is pretty evenly distributed through the whole nervous system. Part of it is occupied in supporting processes necessary for life, e.g., the heart-beat, respiration, etc., and another portion is expended in the production of various reflex actions and the remainder we may consider to be stored up in the brain for the performance of acts which require volition, for the production of ideas or for the exercise of higher faculties generally. An exhaustion of nerve energy must end in death. The nervous system is undoubtedly the vital centre. The lungs and heart act as its servants, vital organs which it cannot fulfil its functions without.
but whose actions are regulated, ruled, and governed by it to the end.

Before going further it will be well to give a definition of the term "nerve impulse" as used in this paper.

By this term it is proposed to describe that force which is called into action in a nerve or nerve centre by external stimuli of any kind or by the action of the brain as an automatic centre. To illustrate this definition take a simple example of spinal reflex action. A stimulus is applied by tickling the sole of the foot; the result is violent movements of the limb. We have here the result of nerve impulses communicated in the first place to the spinal cord and then to the motor centre of the leg. Then again take the lifting of a weight as an example. Nerve force is generated in a portion of the brain and sent down through the spinal cord to the
Muscles, required and thus manifest itself as potential energy.
The simple phenomena of reflex action are now very generally understood. If a mild stimulus be applied to a decapitated animal, only one or two muscles are set in motion; but if the stimulus be stronger, a much larger group of muscles, or even the whole body, is thrown into action. The illustration may be carried further by a consideration of the effects of strychnine poisoning, where a very slight touch produces tonic spasm of every muscle in the body. Now let us analyze these facts rather more closely as to their significance. The doctrine of reflex action may be more pronounced. Irritation of a sensory nerve has a tendency to produce movement of a limited number of muscles; but if the stimulus be severe a much larger motor tract is involved. It would seem probable from these phenomena of reflex action that the nerve energy
which we have seen to exist throughout the nervous system generally, is, by the application of an external stimulus, changed into a nerve impulse which, starting at the peripheral termination of the irritated sensory nerve tracts to the spinal cord. If the stimulus be slight, the impulse is diffused from the central termination of the sensory nerve only to a limited number of motor fibers, but if the stimulus be severe the impulse generated is so powerful that instead of confining itself to a small motor tract it spreads over a large spinal area and produces spasmodic movements throughout a correspondingly large area of the body. We may say then that the normal effect of a stimulus applied to a sensory nerve is to cause a reflex action, definitely in character and more or less purposeful in action—in other words, the impulse has passed from the termination of the sensory fibers irritated to the motor centers of parts, the movements?
which are a natural sequence of such a
stimulus and have in their object a more or less
definite purpose (e.g. withdrawing the limb
from the stimulus, rubbing off irritating
substances as seen in the well-known experi-
ment of placing a drop of acid on the thigh of
a decapitated frog. At first the animal tries
to rub off the stimulating fluid with one leg,
but if this be held it uses the other).

In the case of excessive stimuli applied
to sensory nerves, however, the case is different.
The impulse is so powerful that instead of
being expired to the medulla oblongata corre-
sponding to the sensory fibers stimulated, it over-
flows & in proportion to its intensity travels
into other sensory tracts and may eventually
produce general spasm.

Now it seems to me quite in accordance
with facts to imagine the whole body
embroiled into various sensory areas and
that corresponding to the stimulation of
Each sensory area, we have a certain definite motor tract under active and passive movement of one or more muscles. This I would explain on the hypothesis that each sensory nerve fiber has in connection with its central termination a motor area and efferent nerve, and that the normal activity of a sensory nerve manifests itself partly by subjective sensation and partly by nerve impulses being communicated to this motor area and efferent nerve. Now if the stimulus be excessive and only does the subjective sensation increase in pain but the nerve impulses is too powerful to be confined to its own proper motor tract and efferent process is established. This is the principle I propose to apply in an investigation of certain of the sensory nerves.

No one can deny the applicability of the idea of reflex action in the spinal cord. A consideration of the phenomena of strychnin
poisoning gives almost irrefutable proof of its accuracy. In an animal poisoned with this drug a slight touch causes general convulsions. The impulse communicated to the sensory nerve and from it to the spinal cord is no longer in health confined to its own cortical motor area, but is distributed over the whole motor area of the cord.

Now so far every argument that has been asserted has in its favor physiological facts freely recognized by authorities as undeniable.

We are also told that reflex action takes place in the brain. Some physiologists are inclined to regard every act or thought as a reflex phenomenon depending solely on external conditions. Others, notably Carpenter, look upon the brain as an organ having the power of generating nerve impulses which is reproduced as an idea, an action, or an emotion.

Be this as it may, it is not an extravagant thing to advance that stimuli to any portion of the brain (through afferent fibers)
have traits of least resistance along which they tend to be propagated, or, in other words, have a tendency to spread in one direction rather than another. All those higher qualities of intellect which depend upon associated intellectual efforts may be explained on this hypothesis. A child when it first begins to show intelligence usually does so by recognising its most constant attendant—nurse or mother. Gradually it begins to associate various ideas centering in or rather arising from this intellectual unit—such as obtaining food, etc. The sight of the nurse sends an impulse to that portion of cerebral substance which usually responds to the retinal image and then the form (impulse) passes on into other channels, which every day become more numerous. By and by other ideational centres are produced which again end off branches. It is perfectly clear that if a nerve impulse keeps to its own proper channel provided it is not excessive no morbid phenomena can arise, thus thought may follow thought in
quick succession to do harm comes if provided
the sequence be natural; but, should from any
cause the nerve impulse, generating the first
idea, be directed into a wrong channel, thought
would follow thought but—a morbid state is
produced— in short insanity.
It would naturally follow that in the brain of
a highly educated man and in we have— if
we may be allowed the simile—an infinite
number of stations, in which nerve impulse can
be conveyed and in which it may possibly be
originated, and moreover that every station is
connected with others by a number of channels of
exit and entrance, that corresponding to each
entrance there is an exit and that impulse
arriving by one entrance has a tendency to
leave by the corresponding channel of exit. A
typical nerve cell in all probability would elude
correspond to our simile a ganglion certainly
does.
Now independent of gross lesion an abnormal
process in such an apparatus as we have described might be produced in one of two ways. Either the nerve impulse, if its not too great intensity, can find sufficient means of escape through its natural channel, or a stimulus of slighter severity may, by mis-direction of the central mechanism, be diverted into abnormal channels. In either case the result must be functional neuritis.
Chapt. II

The theory which I propose is applied to a consideration of certain functional neuroses. It is a very considerable resemblance to Dr. Living's theory of explosive force and reversions. Although the points of difference between the process believed in by Dr. Living and that described in this paper are in the main not small, still I think there are reservations and qualifications very similar conclusion which has been arrived at by a different line of argument.

The idea of overloading nerve impulses is physiological. The relation of certain secured from first uttering the phenomena of auditory vertigo, which are universally acknowledged to be due to irritation (a paralysia?) of the semi-circular canal.

The problem I desired to solve was what relation have nausea, vomiting, and syncope (Meniere's symptoms) to the auditory nerve, and on what hypothesis can they be explained.
as results of lesions of the semicircular canals? Speaking of the auditory nerve I allude only to that portion of it which supplies the ampullae of the membranous semicircular canals, and as in this paper it is not intended to touch on any subject anatomically—but only physiologically—I may be excused if therefore I use the term ampullar nerve. It is not intended to give an account of what has been said or done concerning the physiology of the canals, but only to make use of well authenticated and acknowledged facts.

It can be proved by anyone who has patience for the necessary operative procedures and who is fortunate enough to obtain the permission of the Home Secretary that the same train of symptoms can be produced in the lower animals by rapid rotation in the horizontal plane, as by declin or irritation of one or more of the membranous canals. Yet although the results vary slightly in different animals, still the broad facts
remain that injury to the semicircular canals produces (1) Vertigo, or phenomena (circus movements falling off). (2) To the movements of the head (3) Emietity, or paresis. (4)眩目 may have seemed the most striking effect only as it will serve our purpose.

The phenomena of auditory vertigo are well known and comprise vertigo (possibly, a symptom in a mild attack), and a tendency to presbyopia or even complete loss of consciousness. Clearly, the same sequence of events can be produced by irritation in sensitive persons.

From what has been just said it appears that certain of the symptoms observed in animals are not often produced in man.眩目, however, been found in more than one occasion, to be dependent upon the existence of disease or injury of the middle ear, and forced movements of the head have been observed by George of Amsterdam in connection with aural disease. It must also remember that in the human subject
The same amount of irritation is never applied to the semicircular canals, whether by disease or design, as has been used by physiologists in their experiments on the lower animals. From the facts before us we are entitled to conclude that the ampullar nerves terminate in a central brain area, which when severely stimulated gives rise to the sensation of vertigo if we prefer, an equilibrating centre whose excessive stimulation produces the symptom in question.

Let us look once more at the phenomena accompanying the giddiness in a severe case of auditory vertigo or Meniere's disease. Dizziness, vomiting, and oppression are a tendency to it. How is this association of symptoms to be explained? The only rational explanation that suggests itself is reflex action. But this is a somewhat vague term, when applied to cerebral processes, and one which is often used without presenting...
The mind any definite idea. The process in this instance can, I think be explained on one hypothesis and only. When an unusually strong stimulus is applied to the ampullae of the utriculus it strikes the equilibrium center so as to produce the subjective sensation of vertigo. If a still more powerful wave of nerve impulse be communicated to the central organ, the equilibrium center can no longer contain it and it overflows to other centers—the vomiting and cardiac inhibitory thus producing sickness and syncope.

(3) Bynon has demonstrated that very interesting fact that in the section of each semi-circular canal produces a movement of the eyeball in a corresponding direction. He therefore very properly concluded that there is a physiological, if not an anatomical connection between the uculo-motor center and the central termination of the ampullae nerves. That this fact applies to man is probable from cases of associated ear disease and
Stagnamus when the latter depended upon the former. A medical friend who suffered from a certain degree of acquired strabismus informed me that he could correct it with but that in so doing he experienced nausea and vertigo. This proves that not only may nerve impulses travel from the equilibrating to the vomiting and eulo-motor centres, but that the converse is also the case.

Now let us analyze the meaning of this term, physiological relation. It means, in my opinion, that centres in physiological relation are connected with one another by channels for the propagation of nerve impulses. For instance, if an impulse strikes one area and is too powerful to confine itself to that centre it will affects that portion of the brain which is physiologically most intimately connected with the centre first stimulated. Now by a study of the phenomena of vertigo we find that in man the equilibrating, the
Vomiting, the colo-mot and cardiac inhibitory centers are all more or less physiologically related to one another, that probably a stimulus striking the equilibrium center has in the human subject a tendency to overflow in a certain definite physiological direction viz. just to the vomiting, next to the cardiac inhibitory center but rarely to the colo-mot center. In many of the lower animals the various centers for movement of the head must be included. That this applies also in some cases in man was proved by Gage.

The next point to be considered is whether the evidence as to the physiological relation of centers disclosed by a study of vertigo and its accompaniments are borne out by other clinical facts.

A patient with a tendency to syncope or unexpectedly complains of nausea and giddiness and actual vomiting is not rare. These symptoms are perhaps best
marked in cases of faintness from pain, the centre involved being just the central termination of certain cranial nerve fibres, the cardiac inhibitory, the vomiting, and the equilibrating. It may perhaps be urged that the vasomotor centre should at least be included, but may not the vasomotor symptoms (paleness, etc.) be only the result of a diminished blood-supply furnished by the weakened heart? Also says "in individuals fainting from emotion or from severe pain is the result of reflex inhibition of the heart."

Death from shock is in all probability due to a similar cause, but this we shall refer to again.

The symptoms produced by acute lesions of the stomach furnish us with a further illustration. Vomiting, faintness and collapse are among the most common—whether goodness is also present in these cases, I have had no opportunity of ascertaining. The
affuncrt channel is the pneumogastric and the impulse is thus communicated first to the vomiting and then to the cardiac inhibitory centres.

I think that enough has been said in this chapter to justify the conclusion that there can be traced a distinct physiological connection or relationship between certain centres and that certain phenomena may be explained on this hypothesis.

(1) Urbanlochslach Lehrbuch der Chirurgi 1841.
(2) Über die meningiche Krankheit von Gaze.
(3) Cyon. Recherches Experimentales sur les Fonctions des Canaux Lenticulaires.
(4) Gaze op. cit.
Chapter III

It is now almost universally held that many of the so-called functional neuroses are very intimately related to one another. Thus most authorities in speaking of the etiology of epilepsy speak of heredity as a very important element but at the same time insist that the ancestral taint need not be epileptic but only produced by one of the allied affections.

Thus according to Nitsch, 1) "herm in the mother may produce itself as epilepsy in the child."

Russel Reynolds 2) ascribes epilepsy in a large number of cases to a family proclivity to nervous disease.

Hammond 3) writes, "In my own experience I have noted in regard to this point, in three hundred and ninety-six cases of these sixty four had epileptic fathers, mothers, grandparents, uncles, aunts or brothers.
sisters and myself had relatives insane, malarial, cataleptic, affected with severe cephalalgia and of remarkably irritable nervous systems.

Dr. Living in his works on the germ has given a most careful and elaborate exposition of his own views and those of others tending to establish a very close relationship between the functional neuroses.

How it seems probable that a neurotic tendency (if we may be allowed the term) either de rigueur or congenital, must exist in the patient prior to the development of any of the so-called explosive neuroses. In many cases unstable equilibrium of the grey matter of the brain is the condition regarded as the precursor of disease. Let us analyze this term "unstable equilibrium" a little more closely. It means according to the theory advanced in these pages that the nerve impulse when
it arrives at an unstable portion gey matter does not only produce that effect or it is for a intellectual – which under normal circumstances it ought to cause, but additional purposes and even dangerous phenomena. These may be motor as in convulsions, vital as in syncope or intellectual as in insanity. These symptoms may all be accounted for by the hypothesis that the nerve impulse which originated there has a tendency to go out of its own proper channels into them and to produce antidid action. It is the custom of many writers to speak of certain functional centers of the nervous centers as due to vaso-motor changes, produced by reflex action. But this, if it mean anything, surely means that a stimulus has been applied to a nerve of general a special sense and has spread from the termination of the nerve (tissue proper center) so as to involve the vaso-motor center; but if the vaso motor center why not?
other centres? Have we physiological data to prove, or even to warrant the belief that the vaso motor centre is in such intimate relation with all other parts of the central nervous system that it is alone exposed to excessive stimuli?

Besides in sleep we have a cerebral anaemia without any abnormal symptoms. Of course in this case it is probable that the anaemia is the effect rather than the cause, but that does not affect the value of the argument.

It seems not unreasonable to suppose that the nerve cells of the cerebral hemispheres have special functions connected with the higher faculties. These cells are innumerable but no are the thoughts and actions that a well trained man is capable of forming and performing.

I will consider a nerve cell as representing an intellectual unit — in fact the smallest
Conceivable action gains idea just as a nerve expanse in the smallest conceivable portion of a nerve, we can early picture to ourselves the renewal tendency and its nature.

Take a group of cells, each representing a particular idea (the simplest idea is really a complex process); and suppose that each cell becomes linked to its fellow by a branch capable of conducting nerve impulse and that a sensory nerve tunnel enters one of the cells. We have an apparatus which is capable of producing the idea. Let another of the cells be connected with a motor nerve fiber and we have the idea turned into action.

We can conceive moreover any number of such systems and, the more we have in an individual brain the greater will be its intellectual capacity.

I do not advance this as a thing but merely as an illustration, but let us pursue it no further. I show the sensory plenitude of one of the imaginary
systems receive to etray an impression - a wave qv a new impulse it powerful for it to contain it must overlap its bounds and flow into an adjoining system. If this be repeated again, links will be formed out of cells of adjoining systems and beneath even an ordinary stimulus will tend to travel in an abnormal path and produce a neurosis.

As been stated I do not wish to be understood by saying that this actually occurs, but in the present state of our knowledge some such process must be assumed to exist until more light is thrown on the subject of psychology. How certain drugs, notably bromide of potassium, diminish the action and at the same time exercise a beneficial and sometimes even curative effect on some functional disorders of the nervous system, especially epilepsy.

In this disease the prognosis is probably
since its proportion to its duration is according
dojen to its proportion to the number of
eclipses which have occurred. In other words
the more perfectly formed the abnormal channels
in the propagation of nerve impulses, the less likely
is care to result. From what we know of the action
of bromide of potassium it is not unfair to
assume that it has more or less the power of carrying
an impulse to its own proper channel of prevent overflow.
From a consideration of the etiology of epileptic
from attacks and convulsions, which may be
taken as types of functional seizures, one is
led irresistibly to the conclusion that they may,
with regard to their causation, be divided into
two classes according as they are produced by:
(1) Excessive stimulus applied to a comparably healthy nervous system—that is ex-
cessive either through its violence or long continu-
ation, e.g. intense emotion, fear, continued
irritation of peripheral branches of sensory nerves.
(2) The normal stimuli incident to existence, acting in a nervous system in it for some cause the resistance to overflow of nerve impulses is weak e.g. atrointestinal constitutional (especially hereditary kind)

(1) Simson's Cyclopaedia of Med. Vol XIV p. 201
(2) System of Med. Vol II p. 294
(3) Hammond: Diseases of the Nervous System p. 676
(4) E. Living: On Migraine & Sick Headache chap. IV
(5) Deuel Reynolds: System of Med Vol II Epilepsy
(6) Simson's Cyclopaedia Vol XIV. Nott and Nagel on Epilepsy.
Part II
Chap I
Epilepsy

Although various lesions have been found post mortem in the bodies of epileptics it can hardly be denied that in the great majority of cases this is a functional disease. On one hand, one can say that if we have a certain gross central lesion epilepsy will result, and, on the other hand, in many cases there is a definite absence of lesion, either macro- or microscopic. As has been said before, the disease is generally due to an hereditary or nutritive condition, but may also be caused by peripheral irritation when long continued and emotional disturbance such as fear. How it would be useless and quite superfluous to begin a systematic account of the well-known symptoms of epilepsy. I shall therefore confine my attention to those points upon which the study advanced in these pages can be brought to bear directly or indirectly.
It is well known that the aura, when present, may be sensory, motor, or mental. Most writers agree that this should be looked upon as part of the epileptic paroxysm. It is further urged by Richter (1) that the aura is central in its origin and that in those cases where collapse of a limb cuts short the attack, the result is due to reflex inhibition. The doctrine has found widespread acceptance that the primary epileptic change occurs in the medulla oblongata and affects this part by vascular spasm originating in the medullary vascular centre. (4) Now the question arises does this view correspond with the various forms of aura met with? If we recognize the latter as part of the attack, we must of necessity attribute it to the epileptic change and again it is all probability to the case that it is central in its origin. Must we assume that the first change has occurred in that portion of the
brain, in which the nerve channels involved in the aura terminate—the cerebral centre of the region affected?

If we had the sensory and motor auras to deal with, the right possibility still be explained on the hypothesis of the medulla being primarily involved, and then producing reflex phenomena. But can we account in this way for hallucinations when they are the immediate and constant precursor of a fit?

In the production of any form of idea we must look to the gray matter of the hemispheres and here too must be sought the primary epileptic charge in three cases where the focus is mental.

When we consider the various and almost infinite number of forms the aura may take, we surely cannot hardly believe that the medulla is the seat of the first change in all cases, unless we refuse to acknowledge that it is caused by the same
abnormal central state that produces the remainder of the paroxysm. It seems more easy to believe that the epileptic change may originate in any portion of the central organ and frequently attacks one or many of the medullary centers.

Before entering upon a discussion of the symptoms it will be well to refer to their etiology. It has been attempted to show, when speaking of the epileptic aura that the primary change may probably begin in any part of the brain. Now, this being the case if it is not possible and even probable that the epileptic fit is caused by a wave of nerve impulse striking an unstable nerve center by its overflow involving various centers the vaso-motor among them.

It may be asked where this impulse originated.

The answer is to be found in the fact that
Stimuli, either physical or mental, are ever present while life remains; and the phenomena of strychnine poisoning show that under certain conditions of the nervous system a very small stimulus will produce great results.

It is probably in cases of haut mal and epileptic coma the vasomotor centre plays a part, but it seems likely that in addition we may have the same wave of nerve impulses which originates the vasomotor spasm passing over various other centres. Syncope and sleep are produced accompanied by cerebral anaemia but cause no convulsions, although it is not meant for a moment to dispute the fact that very severe anaemia of the brain will produce epileptiform phenomena.

Most of the phenomena of haut mal may, I think, be explained on the theory of overflow of nerve impulses to the various centres...
involved, but if we turn to some of the less typical although no less real forms of epilepsy our hypothesis finds more decided support. Ordinary epileptic vertigo we have paller which is usually and very probably rightly attributed to vascular spasm, but if in these cases we assume the same condition of the whole encephalon to exist how is the absence of convulsions to be accounted for? May should cerebral anaemia in one case produce the latter and in another merely a momentary loss of memory? Russell Reynolds describes an abortive form of epilepsy which has been observed also by Ritchard and by Schröder, van der Kroft and others. It is characterized by local spasms and unconsciousness often absent or very slight. If we consider the process in these cases to be similar to that in true epilepsy we can most easily account for the symptoms by assuming that a wave
If nerve impulse has overflowed and involved certain motor tracts, without reaching the center for consciousness.

The ordinary petrel that the impulse seems to flow always towards the intellectual center avoiding those concerned in motion. It is in this form of disease that epileptic insanity is most common. This is perfectly consistent with my theory, for in such cases we must assume the failure caused to the condition of instability. Abnormal channels of communication are opened and thoughts may follow in wrong succession.

The epileptic maniac sees a loving son. Instead of the retinal image calling forth feelings of affection it produces a homicidal inclination. The nerve current is misdirected and produces thoughts which lead to acts out of affection but of murder. The theory of overflow of nerve impulse in
channels of least resistance seems to account
most easily for many phenomena which
are not equally explicable to any theory of vaso
dilation and vaso constrictor nerves. Indeed we
know very little of either the one or the other, so
little, certainly, to make use of the facts and
explain nervous phenomena. On the other hand
the theory advanced is fonned, as it were, by
the laws of reflex action, by the phenomena
of oblique preference and vertigo.
Virchow and Hellel assert that the epileptic
fit is often followed by the presence of sugar
in the urine. This result has not however,
been found to be frequent, which seems to show
that the true diabetic centre is only occasion-
ally stimulated by the wave of nerve impulse
carrying the symptoms of the fit.
Hippel has found that Raoul's attacks
are usually followed by a temporary albumini-
uria. Now, with regard to cerebral physio-
logy, we cannot localize the centre involved
here as exactly as in the case of sugar in the urine; but we may justly infer that a biphonic change of some kind is excited by stimulation of the medulla, capable of producing this symptom.

According to the same author albumen is never present after epileptic vertigo. This is what one would expect in the latter the nerve impulse spends its force on the intellectual centres and leaves the lower ones untouched or nearly so, as can be gathered from a comparison of the phenomena in the two forms of the disease.

2. Hammond Diseases of the Nervous System p. 666
3. Rothnagel. Symposium of Med. Vol XVII on Epilepsy,
   Reynolds, op. cit.
4. Reynolds, op. cit.
5. Rothnagel, op. cit.
6. Rothnagel, op. cit.
Chap. II
Chorea

Chorea must like epilepsy be still looked upon as a functional ailment. Possibly as Hammond suggests true may be various forms of the disease. In speaking of it, however, we intend to employ our remarks to those typical cases where involuntary jerky motions of muscles exists, which are increased by attempts to execute voluntary movements.

The connection between rheumatism and chorea has been long written of but not yet firmly established. Certainly very contradictory results have been published. In instances at one hundred and fifty-two cases observed by Steiner only four originated in articular rheumatism.

Putting aside rheumatism then the other causes by which chorea is said to owe its origin are very similar to those which favor
The occurrence of epilepsy. Among them hereditary tendency holds a conspicuous place. As in epilepsy so in chorea the choreic taint need not be present, but only a neuritic tendency.

Hysteria, epilepsy and nervous state are not infrequently the progenitors of chorea. The patient who suffers from chorea says Dr. S. Stiller “are very impresible and emotional and very liable to derangements of the nervous system.” (3)

Radcliffe also finds that in a large proportion of cases near relations have been afflicted with disease of the nervous central irritation probably plays a considerable part in the production of chorea in some cases.

Emotional disturbance—most often caused by excessive fear—is a powerful factor. Not infrequently, moreover, the exciting cause has been traced to reflex action, where the irritation was caused by the presence of...
A cicatrix in postcereulonutum.
A consideration of the various exciting causes, the mutability of chorea with epilepsy, insanity, and other neuroses and above all, the absence of definite pathological lesions seem to justify us in considering chorea as a purely functional disturbance of the nervous system.

Now following the plan laid down at the beginning of this paper let us endeavor to account for the phenomena met with in this disease by an application of the theory of overflow of nerve impulse.

If we consider the condition of the central nerve fibers such that nerve force existing whether generated there or conveyed from the periphery in one portion of the central apparatus tends to overflow and involve the motor tracts of the whole cerebrospinal system we have an explanation to account for those cases where the choreic movements involve
the whole body, and we have a condition not unlike that produced by strychnine poisoning. More frequently, however, chorea involves only a limited number of muscles and in these cases we must consider that the unstable condition only affects certain areas of gray matter.

Patients usually suffer least when kept quiet, that is, where there is least tendency to the generation of nerve force. According to Bristow, any attempt to move the affected part in even any excitement, make the paroxysms worse. Attention to the part may also produce the same result. According to Alt. 'This is a simple effort on the part of the patient to subdue the spasm and to keep his face and limbs quiet is in sufficient to increase the violence of the choreic twitches.'

Let us stop for a moment to analyze the meaning of this. Attention concentrate on
The part means a stimulus originating at its cerebral sensory centre. Now, if the motor condition were what we have assumed, this impulse will spread over the motor centres or centres and produce spasmodic movements.

If the arm is affected, it is desired to flex it, under ordinary circumstances, a stimulus is generated in a sensory centre corresponding to the motor centre of the flexors sent down the corresponding efferent fibres. But if this centre is there (as we must assume the motor centres for the flexors to be) are in such a condition that any impulse generated in one spreads over and all, we can easily understand why an attempt at performing a purposeful movement should result in useless jerks. The impulse cannot be confined to the flexor centres but involves the extensors as well and on convulsive twitchings and irregular movements result.

The view we have taken of the choreic
process is certainly supported by the fact that during sleep the movements generally cease. It will be evident that in sleep our centres cease to generate nerve force and as there is no nerve impulse either intellectual or sensory (except when dreaming) to overflow, there is nothing to excite choreic spasm.

The loss of power too, in the affected parts is consistent with our theory, for extraordinary circumstances an effort of this will is restricted to the terminations of those motor fibres which correspond to the muscles which it intended to use; but in chorea this effort is spread over a larger cerebral tract and is therefore less effectual for its purpose.

A similar explanation—mutatis mutandis—would be applicable to choreic anaesthesia.

(1) Hammond Diseases of the Nervous System
(2) Liebreich’s Cyclopedia: Vol. XIV. p. 421.
Chapter III

Neurinism

It will be all the more necessary to enter into a detailed discussion of this malady as Dr. Edward Living in his work on Neurinism and Sich Headache has entered fully into the subject and given so many arguments in favour of his theory of nerve storms and expiring foci.

His theory resembles the one here advanced enough to make arguments in part gone till for the other. It differs, however, in one or two points. In these pages it has been sought to demonstrate the physiological process concerned in the production of certain diseases, by referring them to waves of nerve impulse, sometimes peripheral, at other times central in their origin, and to show that these may produce disturbance either from being excessive in amount or acting on a health.
nervous system, a moderate in amount, but acting on an abnormal nerve centre. Dr. Livingstone may perhaps be best summarized by quoting his own words:

"On this theory, then, the fundamental cause gall
nerves is to be found, not in any irritation
of the visceral or cutaneous periphery, nor in
any disorder or irregularity of the circulation,
but in the a primary and often hereditary
vic or morbid disposition of the nervous system
itself: this consists in a tendency in the part of
the nervous centres to the irregular accumula-
tion and discharge of nervous force-to disruptive
and uncoordinated action. In fact, and the
concentration of this tendency, in particular
localities, or about particular foci, will main-
ly determine the character of the neurosis
in question. The immediate antecedent of an
attack is a condition of unstable equilibrium
and gradually accumulating tension in the
parts of the nervous system more immediately
concerned while the parasymptom itself may be likened to a storm, by which this condition is dispersed and equilibrium in the time restored.

To apply the designation "nerve-storm" to neural activity, let me repeat that it has been no part of my intention to attempt a physical explanation of the phenomena, or to press the analogy it suggests very much beyond an illustration. It was adopted in the first instance as a concise expression or summary of certain facts in their historical development pretty generally recognized, and which we may now proceed to state somewhat more fully as follows.

It will also be seen from a comparison of this paper with Dr. Living's work that the hypotheses have been worked up to by a different chain of arguments.

To return to our subject - the ordinary symptoms met with in simple cases of myelitis are as follows.
(1) Pain over the eye or temple.

(2) Extension of pain over one or both sides of the head.

(3) The patient becomes dull, indisposed, pale & heavy.

(4) The pulse is slower than natural & weak.

(5) Attacks of vomiting during which the pain reaches its greatest intensity.

Such are the more common symptoms of this headache, but others are occasionally met with. First or frequently we must place disorders of vision. Occasionally defective auricles and anomalies of pulse are met with. Paresthesia and muscular weakness have also been known to result.

The symptoms of migraine have been referred by various authors to vascular changes and Palencher has actually divided the malady into two kinds—sympathetic and neuro-paraesthetic. To suit the fact that at one time we have pain at another, flushing of the face.

Surely a simpler way of looking at the matter.
to assume that a nerve impulse is either generated at the origin of the supra-orbital branch of the fifth nerve or that it is carried to this point by apparent fibres of their own. At first the impulse spreads and involves the other branches of the fifth, causing diffuse headache. It then affects this centre, probably the cardiac inhibitory and probably the vasomotor centre causing slowing of the pulse and palor. It then reaches the vomiting centre and begins to decline. Occasionally as we have seen it affects the visual centre. The gustatory is very generally involved in the bad dast in the month so said by Eulenberg to be central in origin and to have no connection with the stomach. The auditory centre is shown to be involved in some cases, where pinnitis is a marked symptom. Paresthesia and paralysis very likely point- to exhaustion of certain cerebral tracts, the result of over-stimulation of the
afluent channels concerned in the production of migraine. We do not yet know much about it, but I cannot help thinking that a study of the subject by means of the physiological relation of centers to one another might possibly throw some light on the subject.

(1) On Migraine & Headache by E. Living, p. 336, 337.
(2) D. Lippincott's Cyclopedia of Med. Vol XIV Art. I
Chapter IV
Vertigo

This subject was entered into rather fully in the beginning of this paper. Indeed it was from a study of the general phenomena of vertigo and its accompanying symptoms that the theory which forms its basis was advanced. It remains, however, to consider a few of the details of the matter a little more closely.

Vertigo is a definite sensation and although the subjective phenomena vary still we have quite enough data to show that stimulation of the various fibrils of the auditory nerve (ampullar portion) may produce any form of true vertigo. For instance, if we accept the theory of a recent and accurate observer (From Brown) we find that irritation of the ampulla of the right-horizontal semicircular canal will produce the subjective phenomenon of surrounding objects moving from left
To right, irritation of the left will produce apparent movement from right to left and vice versa. If going over the six canals singly or combined, it is probable that an irritation applied to one or more of them may produce apparent rotation in any plane; in other words any possible form of true vertigo. Giddiness is often complained of where none exists. True vertigo consists of the subjective sensation of movement round an axis. Some persons feel that they themselves are being moved, others refer the motion to surrounding objects, but this depends upon whether their attention is concentrated on themselves or on external objects. Occasionally the symptom is so well marked that the sufferer is actually impelled to which extent from what has been said it seems fair to conclude that there is an encephalic centre stimulation of which will produce vertigo and that it be more or less intimately connected with the
Central ending of the ampullar portion of the auditory nerve.

Now it will be perfectly obvious to the reader that if the existence of such a center be admitted we can easily account for vertigo from both central and peripheral causes.

Dr. Bradbury has reported several cases where giddiness was produced by anaemia and lactation. It is well known as a frequent accompaniment of gross cerebral and cerebellar lesions. Epilepsy is not infrequently associated with vertigo, which may appear before, after, or during the attack as in some cases of petit mal. These facts seem to show that any encephalitic change gross molecular vascular or otherwise may produce stimulation of the vermillion area.

There are also a number of reflex stimuli which may produce giddiness. Pain or infrequently acts in this way. A stimulus applied to a sensory nerve may as we have
been involve a larger or smaller area according to its amount and the stability of the nervous centres. Sometimes in such cases the impulse strikes the vertiginous area without producing other symptoms, at another time it will strike the cardiac inhibiting centre producing syncope. Then again a not uncommon result of great suffering, be it mental or physical is giddiness followed by syncope. This means that the wave of nerve current has reached first the vertiginous and then the cardiac inhibiting centre. These two are as we have seen (Chap. 11, p. 71) in intimate physiological relation.

The other stimuli which may produce the symptoms in question we may discuss under the following heads (1) Visual (especially stomachic) (2) Cerebral (3) Aural.

As this part of the subject was entered into fully in a paper which appeared in the Med. Times and Gazette, it will be sufficient to repeat
Stomachic vertigo has been described most carefully by Townsend in his "Clinical Lectures." It would seem that in these cases the phenomena are generally paroxysmal, and accompanied by dizziness. The vertiginous phenomena are generally accompanied by epigastric distress, which the sufferer compares to sea-sickness. It is indeed nausea in the literal acceptation of the word, says Townsend. Unfortunately I have never had such opportunity of studying vertigo a stomachico case, but this observation seems to show that sickness accompanies the giddiness.

According to our theory of the production of vertigo, we should expect that, when it is preceded by nausea or vomiting, the symptoms would probably be due to reflex irritation through the vagus, and that in such a case the nerve impulse would strike the vomiting centre rather than it could reach the vertiginous area. The following case which I recently met with is an example. The patient—
a medical gentleman is subject to pain, dyspepsia, and vertigo. He is deaf from profligions catarrh. His vertigo is always preceded by nausea and vomiting, and yields to alkalies and bitters. The diagnosis here seems to the old stomach-vertigo, possibly aggravated by ear-mischiefs. Had the vertigo been followed by nausea and vomiting, these facts would have pointed to auditory vertigo. In the same reason it would seem probable that sea-dizziness is generally due to stomach-irritation, and not to the semicircular canals, in which vertigo is one of its symptoms, it usually follows the sickness. Were it due to irritation conveyed through the canals, we should expect the sequence of events to be, first, intense vertigo, and afterwards nausea and vomiting.

2. Ocular vertigo is often well marked in paralysis of the third and sixth nerves, as also in nystagmus. Hughlings Jackson in speaking of this subject in an address on Medical Ophthalmology
The patient judges the position of the external object by the effort he makes to bring the eye to bear upon it, not by any movement really accomplished. The patient judges, to use the language of Bain, by the outgoing current—outgoing, but the observed, not muscle-reaching. The eye does not or is Helmholtz's expression again, the retinal images do not change position. The patient judges by the activity of the centre for the movement—"

Then, again, in a paper on the localisation of movements in the cerebral hemispheres, the same author writes: "In a case of paralysis of the external rectus, there is evidence of disorientation. In striking at an object held on the paralyzed side, the patient's hand goes beyond the object. To bring the eye upon the object, the patient has to send down as much force as would in health be enough to carry the eye beyond it. In the case supposed, the defect is in the conducting nerve-trunk,"
In the energy of the central. It is in the transmission of force, and in the being a lesser amount of transmission; an unusual amount is transmitted part of the way, but it cannot all get to the muscle. The patient judges by the strength of the central excitation, not by what that actually accomplishes.

These facts explain the patient's giddiness, to the production of which, contrary to what is usually supposed, double vision has but little share. Vertigo should denote the taking as accomplished a movement which is only attempted, in which there is no strong or brisk central excitation.

We see from these statements that in palsy of the ocular muscles a very large amount of central impulse is generated by every attempt to recognize the malposition of the globe, but that it cannot reach the muscle for which it is intended, owing to the nerve-trunks not fulfilling its conducting function. Now, what
decrease of this energy! Shut off from its proper channel of escape, it would seem probable that a great part of it is thrown back upon the occipital for the centre, from there overflowing to the adjacent vertiginous area, producing giddiness.

Auditory vertigo is very generally associated with disease of the middle or internal ear, although foreign bodies such as wax may produce it by pressing on the membra tympani. When the internal ear is affected primarily, there need not necessarily be any impairment of hearing in sounds conducted through the air, as in a case mentioned by Pecquet. But more commonly, labyrinthine vertigo is due either to lesions of the internal ear secondary to tympanic disease, or to produced by a change of tension in the end and peri-lymph as a mechanical result of the middle ear lesion. It would be out of place here to enter into a discussion of the forms

NO
of ear-disease which produce giddiness, and the manner in which they act, but I may perhaps be excused in giving briefly the forms of vertigo that have been known to be associated with cerebral maladies:

1. There is the well-known Vertigo Menière's symp.

2. The leading features of which are tinnitus, vertigo, nausea, and occasionally loss of consciousness, whether due to syncope or coma. I would at present leave in an opusquetum.

Althaus in his Text-book on Heromo Diseases remarks that giddiness is but a lesser degree of coma; and Wilks seems to consider auditory vertigo a momentary loss of consciousness.

Whether this aspect of our subject is due to an overflow of nerve-impulse directly to the centres of consciousness, or attacks the latter by influencing the cardiac inhibitory or vasomotor centres, it is difficult to say, still the facts are interesting.

2. The real form of auditory vertigo is when the
patient tends to stagger backside, generally towards the end of the ear which was afflicted.

3. He may also have patients affected with a more or less chronic giddiness.

In his article on Vertigo in "Regnolli's System of Medicine," Dr. Ramsay H. speaking of acute stomach vertigo, says, "Curiously enough, it is rare that patients complain of oscillation in defective hearing, or of tinnitus aurium, although both these complaints are very common in the chronic stomachal form of vertigo." Again, as a common combination of symptoms, the same author names "vertigo, tinnitus aurium, and partial deafness."

Now, these remarks, coming from such a high authority, would suggest to consider the following questions:

1. Has the ear examined in every case where this combination of symptoms existed?
2. Has the deafness permanent or transitory?
3. How many of these cases cured by treatment...
directed to the stomach alone? I may have mention that dyspepsia in old, feeble, recognized by aural surgeons as a cause of deafness.

4) Then again, associated with aural disease we may have a giddiness produced only by such acts as getting up quickly or turning round suddenly. In some people, however, the same thing happens in perfect health, so that we cannot attach much importance to the symptoms unless it be very well marked, or has only existed since ear-mischiefs began.

In connection with auditory vertigo a very difficult problem is suggested by three cases of chronic middle-ear disease which caused paroxysmal vertigo without any apparent acceleration of the ear-mischiefs. An accumulation of nerve-energy, either in the auditory nerve or its central terminations, must probably be assumed.

Journal of Anatomy and Physiology 1874
(2) "Vertigo or Dizziness" by Bradbury. Graduation Thesis.

(3) Eyon, "Recherches Experimentales sur les Fonctions des Canal Semicirculaires".


(5) Juneau, "Clinical Lectures".


(7) Hughlings-Jackson, Address on Medical Ophthalmology, delivered before the Med. Society of London.

(8) Hughlings-Jackson, "On the Localisation of Movements in the Cerebral Hemispheres".

(9) Kranitz, "Labyrinthinic Vertigo" (West Riding Asylum Report, vol. 6).


(12) Ramskill, article on Vertigo ("Reynolds System of Medicine")
Chapter V

Some General Applications of our Theory.

Without thinking it either desirable or possible to bring our thing to bear upon all nervous symptoms, we may be allowed to give one or two of its more general applications.

Let us, for a moment, consider the process concerned in the production of voluntary movement—let us say in the case of example of the arm.

In the case supposed, a central impulse is generated in the brain and through the spinal cord and nerves conveyed to the muscles which are required in the performance of the movement. Now, if from some cause, such as sclerosis, the nerve current cannot reach its destination what result are we to expect?

The first consequence of such an impediment would undoubtedly be degeneration of nerve energy—so, in fact, an attempt, on the part of the central nervous apparatus, to overcome the obstruction. This being impossible, the great amount of nerve impulse generated must find an outlet. This it does by
overflowing to other centres and as a result we have involuntary and purposeless movements of a whole limb instead of voluntary and purposeful movement of one group of muscles. How far this application of our theory may account for nervous phenomena observed at the bed side I leave those who have ample opportunity of clinical investigation to judge.

Epileptic form neuralgia is a disease, which though uncommon furnishes an interesting example of overflow of nerve impulses. As was before pointed out the sensory nerves of a part are intimately connected with the motor centres. Thus the prick of a pin produces, as a matter of course, movement of the part hurt but not necessarily on the parts of the body unless the pain be very severe on the person experimented upon very nervous. This seems to show that sensory impressions—especially of sharp and sudden—are conveyed by sensory nerves and from their central terminations tend to spread to other centres more or less corresponding to the distribution of the sensory fibres involved.
These cases where neuralgia of the fifth nerve exists, twitching of the face we must consider that the nerve stimulus applied to the sensory center has overflowed and reached its corresponding motor tract, producing the symptom in question. A very interesting comparison may be drawn between the above and the results of neglected toothache. It may not be generally known that the pain caused by a carious tooth sometimes ceases to be felt in the offending part (the tooth) but still spreads itself over other branches of the fifth nerve. The first branch thus affected is usually the one supplying the ear so that toothache is simulated in reality we should say algia or produced, if matters be allowed to go on, the whole of one side of the face, head and, not unfrequently, the mamma of the same side, become extremely painful and the general health may suffer as, for instance, in the following case:

Mrs. had been complaining for six or seven...
weeks of intense neuralgic pain all over one side of her head and face. There was no swelling and apparently no pain in speaking in the lower jaw, but the ear was said to be very painful. It was also complained of as painful to the touch (i.e. the auricle). On examining the tympanic membrane there was evidence of chronic middle ear catarrh (thickening and indrawing of the drumhead with spasticity). The walls of the meatus were hyperaesthetic and slightly reddened. As a palliative one of Greber's zinc lozenges was introduced. This had little or no effect & the pain got worse. I also learned from the patient that the pain had at first been deep in the ear. Knowing that the most common cause of aural neuralgia is to be found in the presence of a carious tooth, I recommended a dentist to be consulted. Before the patient could make up her mind to do so, the pain had extended to the region of the clavicle and mamma of the same side. A carious tooth was extracted and on the
Following day all pain had disappeared. In this case the pain had at times been so severe that frequent hypodermic injections of morphia had been resorted to. Loss of sleep and appetite had seriously impaired the patient's health. How at no race the chain of events in this and similar cases, which are by no means uncommon. The diseased tooth had sent waves of impulse up towards the Gasserian ganglion, and from the symptoms we must include that the almost the whole of the nerve current overflowed and other branches of the fifth and was then conveyed to the centre of consciousness and eventually were again overflowed and produced the sensation of mammary pain.

It may be asked why part of the overflow is supposed to have taken place in the ganglion and part in the higher centres. Our answer is that had the first overflow taken place in the brain we should have had all the symptoms present but one besides—hoastrache.
In the same way, we know that the pain in the knee in cases of hip joint disease is not caused by central reflex action, but that the overflow must occur lower down for pain to often absent from the offending joint.

The pain in the mammary region, however, requires the hypothesis of overflow in the higher centres on physiological grounds. My in some cases have impulse lends to overflow from sensory motor centres and in other attacks only strong sensation is a problem which will not here attempt to answer. In the case which has just been described what were the sensory fibres of the fifth involved but also the spastic branches as shown by the hyperaemia of the neural. Writer's cramp seems to furnish an example of overflow from one motor centre to others. Here all other centres are stable except that governing the act of writing from which impulse overflow producing choreic movements of the hand or forearm.

Let us now turn to death from shock and consider the subject according to the line of enquiry which he
been followed throughout this paper. That may be caused by a stimulus applied to the periphery of an ordinary sensory nerve, or to the higher centers according as it results from injury or emotion.

The explanation of the phenomena seems very simple. In either case an enormous amount of nerve impulses exist in the brain. In the case of excessive emotion, it is generated there, in the case of a crushed limb it is carried thither by apperent nerves. In either case it seems probable that the first effect of this is increased, but very transient, functional activity produced by the overflow of nerve current to various centers. At the beginning of shock the pulse is slow, as pointed out by Fourneau-Gondy. In overflow of nerve current impulse is the cardiac inhibitory centre. After a very short time of increased activity exhaustion of the centre takes place with
loss of consciousness, diminished reflex action and death.
This is quite in accordance with our knowledge of physiology which makes it certain that the greater the functional activity of a part the sooner will it become exhausted.
A check the early stimulation of the cardiac inhibitory centre by weakening the heart-contractions furnishes another element which hastens the exhaustion; for a little consideration will show that just when the activity of the cerebrospinal centres is greatest their blood supply is curtailed.
Proably in death from check nerve exhaustion and nerve starvation each play their part.

"Surgical Enquiries" Chapter 1.