The diagnosis of traumatic lesions in the cerebro-spinal axis : and the detection of malingering referred to this centre / by B.A. Watson.

Compliments of the Author.

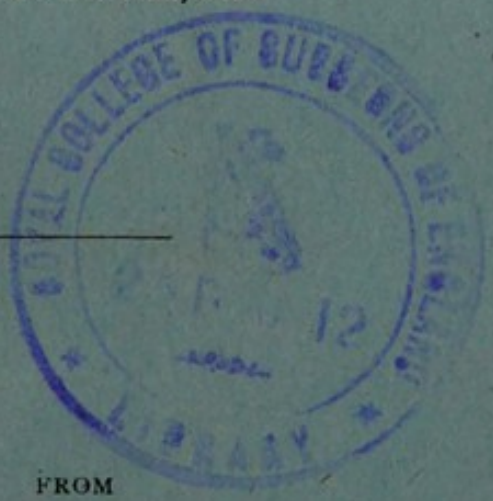
THE DIAGNOSIS OF TRAUMATIC LESIONS IN THE CEREBRO-SPINAL AXIS, AND THE DETECTION OF MALINGERING REFERRED TO THIS CENTRE.

21

BY

B. A. WATSON, M.D.,

CONSULTING SURGEON TO THE BAYONNE HOSPITAL; SURGEON TO THE JERSEY CITY HOSPITAL, ETC.



FROM

THE MEDICAL NEWS,

June 6, 1891.

[Reprinted from THE MEDICAL NEWS, June 6, 1891.]

THE DIAGNOSIS OF TRAUMATIC LESIONS IN THE CEREBRO-SPINAL AXIS, AND THE DETECTION OF MALINGERING REFERRED TO THIS CENTRE.

BY B. A. WATSON, M.D.,
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THE counterfeit cannot be detected without an accurate knowledge of the genuine, and, in addition to this, an exact knowledge of the counterfeit, together with a definite knowledge of the difference between the genuine and the counterfeit, is still imperatively required.

It therefore follows that whoever would attempt the detection of malingering should always be able to diagnosticate concussion of the brain and spinal cord. The proper performance of this task requires a full knowledge of the etiology, semeiology and pathology of the morbid condition under consideration. Let us therefore direct our attention to these essential factors of diagnosis, carefully analyzing each in order that we may more thoroughly comprehend their relation and bearing on each other. It is universally admitted that concussion of the spinal cord most commonly follows falls upon the feet, the buttocks, and less frequently upon the hands when both arms are out-stretched; gunshot wounds

involving portions of the spinal column; a stroke of lightning; heavy blows delivered over the bones of the pelvis or in the line of the spinal column.

The direct effect of these falls and blows is the production of contusions of the soft parts, etc., which are more or less disorganized by these traumatic agents. The force of these agents is not by any means entirely expended in the production of these contusions, but there will be carried along the bony structures a vibratory motion which will also be imparted in varying degrees to the adjacent soft parts. A very fair idea may be gained of these vibratory movements by placing the hand on the apex of a bell, suspended in the air, while its base is struck with a metallic hammer; or by the touch of a properly keyed string of a musical instrument when it has been put in motion. The same vibratory motion may also be very well illustrated by tapping gently with the finger on a glass jar or other vessel which is partially filled with jelly and then watching the tremulous movements imparted to the contents by the slight blows delivered on the vessel. The attention of the medical profession has been frequently called to the peculiarities of concussive or vibratory force by French authors, but its physiological and pathological action has never received that careful study to which it is entitled. A very important factor which presents itself for our consideration in connection with the study of this force is the fact that it is not transmitted equally well by the various organs and tissues of animal bodies; and, consequently, a direct blow delivered on one part of the body may be entirely negative,

or result in the production of very little vibratory action, while on another part of the organism the results will be very marked. Thus a blow delivered directly on the nates will give rise to this concussive force and be transmitted along the spinal column to the bony walls of the skull, and from this structure to the brain itself, which is in such close contact with these bones. A portion of the same force will likewise be expended in varying degrees on those organs in intimate contact with this line traversed by the traumatic agent. The functional disturbances and the pathological changes in the various organs will depend on the physiological functions, weight, texture, etc., of the parts involved, and these need not be enumerated here.

A blow delivered on the feet, while the lower extremities are extended, will be transmitted to the bones of the pelvis and then along the spinal column. A somewhat similar result may possibly be produced by a fall on the hands when the arms are extended; though the force of this blow will be much diminished by reason of the anatomical differences in the parts, particularly the less intimate connection between the bones of the arms and the spinal column than exists between the bones of the lower extremities and this important highway for the transmission of concussive force. It should likewise be remembered that the more concentrated the direct traumatic force, *i. e.*, the smaller the area to which it is applied, the greater will be the amount of concussive force generated, while the other factors remain unchanged. Thus a blow covering an area of four square inches delivered

directly over any part of the spinal column will generate far more concussive force than it would if spread over the whole posterior region of the body. In fact, in the former case, if the blow was a severe one, functional derangements and pathological lesions might rationally be expected to follow from the concentrated nature of the concussive force, but in the latter they will be looked for in vain. There is a very marked difference between the concussion in the case of gunshot wounds produced by musket or rifle balls and that which follows heavy blows or falls; in the former the pathological changes are more circumscribed, while in the latter they may be considered general. Thus, a musket ball having found lodgement in the vertebral column, and in some degree having impaired its outer surface without producing any lesion of the osseous tissues within the canal, the spinal cord will frequently be found in an ecchymotic state at a point near the lesion in the spinal column. In connection with this subject, it may be stated that no single blow, however great its momentum, delivered on the anterior surface of the thorax or abdomen could rationally be expected to produce concussion of the spinal cord, since its force would be expended almost entirely on the visceral organs within these cavities. The study of the effects of lightning, and likewise those of electrical currents of both high and low degree of potency, possesses very great interest for the surgeon in connection with the consideration of concussive force.

The vibratory character of electrical currents is apparently identical with the other forms of con-

cussive force; and it is readily demonstrable that the physiological action, functional disturbances and pathological changes possess essentially the same characteristics. The fact has long been recognized by the ablest authorities that concussion is physiologically indicated by a nervous and circulatory excitation or depression. In accordance with this opinion Verneuil has given the following definition of concussion: "A series of phenomena more or less sudden followed by a mechanical disturbance of the anatomical elements, tissues, and organs, characterized by a temporary excitation or a depression of the functions of the parts disturbed, and likewise producing anatomical changes, are generally observed in cases of functional activity or repose." Mechanical concussive force, like lightning strokes and electrical currents, may produce every possible degree of concussion, varying from the slightest functional disturbances which pass off within a few seconds, to instantaneous death. Further similarity in the effects of these agents is shown by the fact that concussion and even death may be produced by rapidly repeated mechanical blows or a rapid succession of electrical strokes, while a single impulse would be scarcely sufficient to cause even temporary functional disturbance. The pugilist knows that to render his antagonist unconscious the blows should be delivered upon the head. He therefore seeks to deliver in rapid succession blow after blow until his object is accomplished. Experimentation on animals has shown that concussion

of the brain may be caused by slight taps with a hammer delivered on the head in rapid succession.

In our consideration of the etiology of concussion of the cerebro-spinal axis we have said little in regard to its causation in the brain. It is probably understood, however, that those agencies which have already been mentioned are the important factors in its production in this organ as well as in the other organs of the body. The same general laws pertaining to the etiology of this morbid condition are as applicable here as elsewhere, consequently a blow delivered on the head will be more efficacious in the production of concussion of the brain than would be the concussive force had it been transmitted to this organ from some other part of the body. Furthermore, the weight of this organ and its other anatomical peculiarities must receive due consideration.

In the attempt to describe the symptoms of concussion in the cerebro-spinal axis we immediately encounter a difficulty arising from the fact that the functions of these organs are materially different, and consequently the symptoms observed in connection with a traumatism in the spinal cord will produce entirely different symptoms from those found when a similar lesion exists in the brain. The remedy for this difficulty is very simple and only requires that the symptoms be separately noted as they pertain to lesions in the different organs. A much more serious obstacle is found in the combination of all sorts of injuries with those of *pure* concussion, especially those having their origin in fractures, dislocations, sprains, rupture, and stretch-

ing of ligaments, particularly in the spinal column, and likewise with the concussion in one organ the symptoms of which are erroneously attributed to this morbid condition in another. In illustration of the above-mentioned error, I desire to call attention to the fact that hæmaturia is frequently mentioned as a symptom of concussion of the spine, while I have conclusively shown by my experiments on animals that it bears no constant relation to lesion of the cord, but, on the contrary, following concussive accidents, *indicates a concussion of the kidney*.

Furthermore, visual changes are frequently attributed to concussion of the spine, although as a matter of fact the lesions on which these changes depend are situated either in the brain or the eye. I am now prepared to assert that the greater part of the organs within the thorax and abdomen are equally as liable to suffer from concussive force as the brain itself; and likewise that concussive accidents are much more frequent in these visceral organs than in the spinal cord. This assertion is based on the fact that these lesions are entirely analogous to those observed in the brain, merely requiring that the proper allowance be made for differences in weight, texture, etc., factors which must be taken into consideration in connection with the study of the lesions of concussion in the brain and spinal cord. Furthermore, every true concussion takes its origin in the same peculiar force. Therefore, if it be desirable to speak of concussion of the brain, spinal cord, etc., why should it not be applied to the lungs, liver, kidneys, etc.?

Would it not be absurd to speak of gunshot wounds of the brain and spinal cord, while the term is withheld from analogous lesions caused by the same peculiar force in other parts of the body? An attempt has recently been made to employ the term "nervous shock" instead of concussion; but this departure possesses no advantage and has already caused much confusion. No attempt is here made to deny that certain portions of the nervous system participate in the concussion; although it is self-evident that the functional disturbance and pathological lesions are primarily developed in connection with the *circulatory system*. These preliminary comments are made in preference to any attempt at the mere enumeration of symptoms, which, in order to possess real value, must be fully stated and explained. It should likewise be remembered that there are different degrees of concussion and these differences are indicated by a corresponding difference in symptoms.

It must now be apparent that the limits of this article compel me to economize space, so far as it can be done without defeating the object for which it was written. Permit me, therefore, once more to call attention to the fact that concussion always produces either excitation or depression, *i. e.*, functional activity or repose. The former condition possesses practically little interest, if observed immediately after the application of the concussive force, since it is commonly of short duration, and does not require any attempt at treatment. However, there is another form of excitation occasionally observed in severe cases of concussion of the

brain which commonly makes its appearance from twelve to forty-eight hours after the receipt of the injury. This condition is indicated by a full pulse, flushed face, throbbing of the carotids, headache, hot and dry skin, photophobia, noises in the ears, and sometimes marked drowsiness. These symptoms point to the existence of cerebral hyperæmia, which is commonly of short duration; but in some cases it is the prelude to inflammation of the brain. In the severe cases of concussion of the brain or spinal cord the prompt appearance of *symptoms indicative of depression* commonly suggests the existence of organic lesions, rather than mere functional disturbances; while, as a general rule, the degree of danger is fairly expressed by the depression. It should, however, be remembered that the symptoms of depression, in order to possess any special value, must pertain especially to the functions which are known to be performed by these organs. Therefore, a diminished consciousness, although the patient may be aroused sufficiently to answer questions, the pupils contracted or dilated, relaxation of the sphincters, a feeble and irregular pulse, shallow and sighing respirations, a cold, pale, and clammy skin, are symptoms that point very clearly to the brain as the seat of the injury when the symptoms have followed promptly after the application of concussive force. The symptoms following those above recorded belong to the reactionary period; the object of this paper does not require their mention here. Concussion of the spinal cord is most commonly indicated by the appearance of paralytic symptoms, which frequently

involve the lower extremities, especially the motor power and likewise in many cases also the sensory. Therefore in our experiments made on dogs our attention was promptly directed to the movements of these animals immediately after the application of the concussive force.

The following facts possess an important bearing on the subject now under consideration. From a recent summary made from my "Experimental Study of Lesions arising from Severe Concussion" I shall here venture to cite the following: "Paraplegia is a very important indication of a lesion of the spinal cord and may have its origin either in a disease or traumatism. This morbid condition may be either complete or incomplete, and when due to disease it is commonly slowly progressive; but if due to traumatism it is rapidly manifested and soon reaches its fullest development. In support of this statement examine the following facts: The whole number of cases in which pathological changes were found in the cerebro-spinal axis was fifty; the whole number of cases in which any indications of these lesions were observed during the life of these animals was thirty-three, while our record shows that in the last-mentioned class there were twenty-three cases of paraplegia."

It will here be observed that there were ten cases in which there had been noted during the lifetime of these animals some indication of traumatism, but which did not exhibit during that period paraplegic symptoms. It is also worthy of notice that in every instance these paraplegic symptoms were observed immediately after the application of the concussive

force; and, furthermore, that in all cases of complete paraplegia there was not a single case which subsequently improved. In the large majority of cases of incomplete paraplegia there was commonly some improvement observed during the first three days, but in a few instances the paraplegic symptoms became more marked during the first twenty-four hours; however, even these cases soon showed signs of improvement. It is not claimed that even the immediate appearance of paralytic symptoms after the application of concussive force establishes, in all cases, the existence of pathological lesions in the spinal cord. I am strongly impressed with the idea that an injury of the peripheral nerves may be followed by temporary, and commonly, local paralysis. A certain degree of importance should be attached to the existence of either anæsthesia or hyperæsthesia in all cases where lesions of the spinal cord are suspected. The existence or absence of pain in these cases possesses very little interest for the diagnostician. In fact, the existence of pain and soreness ought probably to be looked on as having their origin in a contusion, sprain, or wrench, involving the vertebral ligaments, muscles, etc., rather than indicating lesions of the cord. Having now presented a few of the characteristic symptoms of true concussion of the brain, and likewise of the spinal cord, I desire in this connection to add that it is certainly very unfortunate for the science of surgery that it has hitherto been the custom of nearly all authors, writing on what they have unfortunately designated *concussion*, to group together half a dozen or more traumatic conditions, differing radically from each

other in their *etiology*, *semeiology*, and *pathology*. It requires no prophet to assert positively that so long as this grouping is continued there will be a want of clearness in all these articles; that theory will be frequently introduced in the place of scientific facts; that clinical observation will continue to mislead the profession; that good-natured authors, believing that all their predecessors in writing on this subject have told a little truth, will still further add to the existing confusion in their attempts at compounding these incompatible ideas. Let me ask what would be the inevitable result, if medical authors should group together under the unfortunate cognomen of "Smith's disease," scarlatina, diphtheria, rubeola, roseola, variola, typhus fever, etc.? But this combination is no more objectionable than that grouped under the name of "railway spine," which is so frequently employed by so many of our surgical authors even at the present time. However, the dawn of a new light may be approaching, since Dr. John A. Lidell, in writing on "Injuries of the Back, including those of the Spinal Column, Spinal Membranes, and the Spinal Cord," states that "The traumatic lesions of the back naturally arrange themselves in three groups, as follows:

"1. Injuries of the integuments and muscles, or soft parts generally.

"2. Injuries of the vertebral column.

"3. Injuries of the spinal membranes, spinal cord, and spinal nerves."[1]

[1] The International Encyclopædia of Surgery, vol. iv. p. 668. Edited by John Ashhurst, Jr., M.D. New York: Wm. Wood & Co., 1884.

It must be apparent that this classification presents many important advantages over that commonly employed. Another serious source of confusion has arisen from the fact that the term concussion was, in former times, restricted in its application to the brain, although analogous pathological lesions were produced by the same force in nearly all the organs of the body.

Furthermore, concussion was badly defined by the old authors; because the term was used to indicate a condition induced by a more or less violent shaking of the brain, whereby serious symptoms were produced without lesions of the structures. Recent experiments on animals and a careful study of this subject have entirely failed to support their statements in regard to the non-existence of lesions after concussive accidents in the brain and spinal cord. The results of my experiments were very conclusive in regard to the existence of these lesions. There were fifty cases in which pathological changes were observed in the cerebro-spinal axis, but only thirty-three of these cases showed any symptoms of injury of this centre during life; it will, therefore, be readily seen that in seventeen cases, although pathological lesions existed, they were not made apparent by symptoms which could be recognized in the animals. It is also noteworthy that there was not a single case in which the existence of concussion of the cerebro-spinal axis was even suspected, wherein by our subsequent examination, we failed to discover pathological lesions, *post-mortem* or *microscopical.* These pathological lesions in true concussions are certainly unique in character and nearly

or quite pathognomonic in significance. The only lesions that bear semblance to the pathological changes of concussion are those arising from contusions, and these are localized and not distributed generally throughout the organ, as is commonly the case in the former morbid condition. However, there is found an exception to these rules in the case of concussion following gunshot wounds, in which the injury is commonly localized and more or less closely resembles the ecchymotic appearance arising from contusions. The functional disturbances and the pathological lesions are clearly shown in my experiments to have had their origin in the vasomotor system. In order to illustrate the pathological changes in the spinal cord which have been observed to follow in a mixed or complicated case of concussion of this organ, I will here cite the report of an autopsy, etc.,[1] made on the body of a coal-porter, who had died thirty-four hours after the accident:

"*Autopsy*: There was no external trace of injury. The membranes of the cord were healthy. The substance of the cord was contused opposite the fourth and fifth cervical vertebræ. On section there was found ecchymosis of the posterior horns of gray matter on the left side, and of the adjacent part of the lateral and posterior columns. There were also limited spots of ecchymosis on the right side, one in the right posterior column and one in the right anterior horn of gray matter. The gray substance, generally, was hyperæmic. On removing the spinal

[1] International Encyclopædia of Surgery, vol. iv. p. 790 et seq. Edited by John Ashhurst, Jr., M.D. New York: Wm. Wood & Co., 1884.

cord and membranes nothing abnormal was discovered in the vertebræ until the posterior ligament had been dissected off, when it was seen that the body of the fourth was separated from that of the fifth, and the left articular process of the fourth had been chipped off."

Dr. John A. Lidell, who reports this autopsy, comments on it as follows: "The essential features of this instructive case are as follows: (1) The cord substance was injured by concussion and not by any displacement of the parts. (2) The injury was attended by a number of minute extravasations of blood (ecchymoses) in the gray substance. (3) There were anæsthesia and loss of motion in both lower extremities and in the left arm. (4) There was paralysis of the sphincter ani and sphincter vesicæ, which denoted that the reflex motor apparatus was also paralyzed. (5) The anæsthesia passed away in the course of some hours, the return of sensibility being noted first in the parts most distant from the injury. (6) Hyperæsthesia appeared synchronously with the reaction from 'shock,' and steadily increased in severity. (7) Hyperæmia of the gray substance was found as well as ecchymosis. It should be remarked that the hyperæsthesia was more severe in the right arm than elsewhere, and this part had not at any time been paralyzed." This case was reported by Dr. Lidell as a pure or uncomplicated one of spinal concussion, but I am compelled to question the correctness of the report in this particular, and direct attention to the ruptured ligament which it seems to me may have permitted a partial dislocation to have taken place,

with contusion of the cord, the vertebræ afterward having been restored to their proper position by the natural resilience of the parts. The symptoms of paralysis were certainly more marked than they are usually in cases of pure, uncomplicated concussion of the spinal cord. The following microscopical report conveys a very clear idea of the pathological lesions observed in our experiments on animals in the cerebro-spinal axis when there were no complications in this centre:[1] "Brain intensely hyperæmic, otherwise normal. Every portion of the cord intensely hyperæmic. There were punctate hæmorrhages in the dorsal and cervical regions of the spinal cord, in both the anterior and posterior horns, also in the middle commissure."

The striking analogy existing between the lesions in cases of death caused by electricity and the other cases of concussion may be probably better illustrated by citing the report of the autopsy made by Dr. Carlos F. McDonald on the body of William Kemmler after execution by electricity, since this examination has been more carefully detailed than any of the others. The others, however, entirely agree with McDonald's, so far as given. McDonald's report is as follows: "Rigor mortis marked. On incising the skin over the sternum the blood which escaped was unusually dark and fluid and remained so on exposure. Lungs: Tardieu spots were noticed on the posterior border of the lower lobe of the left lung, also in the middle lobe of the right lung.

[1] An Experimental Study of Lesions arising from Severe Concussion, p. 22 et seq. By B. A. Watson, M.D. Philadelphia: P. Blakiston, Son & Co., 1890.

Heart: Valves and substance normal, ventricles empty. Liver: Blood from cut surface of dark-crimson hue. Gall Bladder: Distended with bile. Spleen: Normal. Kidneys: Both markedly congested. Brain: *Capillary hæmorrhages* were noted in the floor of the fourth ventricle, also in the third ventricle, and in the anterior portion of the lateral ventricle. The perivascular spaces seemed to be distended with serum and blood." Dr. Spitzka's report of the preliminary microscopical examination is as follows: "The brain, spinal cord, and peripheral nerves appeared strictly healthy in every part examined, except in the area corresponding to the discolored (anæmic through the contraction of the vascular channels) area of the Rolandic and pre-Rolandic regions, the ventricular surface, the pons and the medulla oblongata. The latter, which had been the seat of post-mortem preservation of temperature approaching that of the normal human body, were distinctly softer than the observer has ever been accustomed to find these parts in autopsies on persons of Kemmler's age, so soon after death. The hæmorrhagic spots in the fourth ventricle, which were strongly marked, were not accompanied by signs of parenchymatous rupture of large bloodvessels. Hence they may be regarded as having the same significance as the 'taches de Tardieu' found on the surfaces of the organs, notably the heart and lungs. The peculiar softened vesicular zone of tissue underlying the outermost layer of the cerebral cortex is noteworthy as 'the destruction line,' and runs parallel to the free surface of the brain without

dipping with the sulci. Examination of the fresh specimen revealed the existence of vacuoles (probably gas bubbles) in the ganglion cells and in the parenchyma of the destruction line. From the fact that no hæmorrhages had occurred in this softened area, it is a just inference that it was produced after life had become entirely extinct, for the continuance of a blood circulation in a softened brain area is incompatible with the bloodless appearance already noted, and the absence of capillary hæmorrhages in this very district, while they were present in those remote from the site of the electrode."

In order to approach the consideration of *malingering* it is necessary to devote some attention to the traumatic conditions which frequently complicate concussion in the cerebro-spinal axis; since the majority of authors still include these morbid conditions under the head of "spinal concussion," "railway spine," etc. Fractures and dislocations of the vertebræ require little attention at the present time, since they are generally easily diagnosticated, the only exception to this rule being those cases where there is a complete absence of deformity, which may be the case where the fracture consists in chipping off a portion of the articular vertebral surface, etc., or in an incomplete dislocation of a vertebra in which the natural resiliency of the ligaments and muscles has restored the symmetry of the parts. In these cases, it is thought that the careful and observing surgeon will not be compelled to remain long in doubt as to whether the patient before him is a malingerer or suffering from a real traumatic injury—since the prompt appearance of

objective symptoms will certainly be largely dependent on the locality of the injury, while their degree of severity will depend very much upon the extent and character of the traumatism.

There now remain for our consideration in this connection contusions, sprains, twists and wrenches of the back, which on account of their medico-legal bearings are entitled to a more thorough analysis than can be given them in this article. The study of these morbid conditions will be greatly simplified by bearing in mind that their symptoms, the primary and secondary effects, are entirely analogous to injuries of the same character in other parts of the body. It is claimed that the spinal column and the bones which connect directly with it are provided with ninety-nine articulations. The symptoms which characterize the morbid conditions now under consideration are pain, swelling, tenderness under pressure, and occasional ecchymoses. The existence of the symptoms will be limited to the area involved in the traumatism, while the degree of severity will depend on the intensity of the injuries. In some cases there may be constitutional disturbances which will aid in establishing the existence of a morbid condition. The ecchymoses and swelling are objective symptoms, and consequently entitled to due consideration, while the pain, stiffness, and tenderness under pressure may require confirmation. The verification of the patient's statements on these points, and frequently on others, especially questions involving the existence of paralysis, might be definitely put at rest by the proper use of an anæs-

thetic, which ought to be frequently employed in medico-legal cases. The general surgeon no longer hesitates to employ this agent in his private and public practice for diagnostic purposes; and why should it not be thus employed for the purpose of securing justice in our courts?

Drunkenness and syncope may make the diagnosis more difficult in certain cases of concussive accidents, whether it be pure or complicated concussion; but these factors are of short duration, and therefore cannot long continue to embarrass the surgeon.

It is unfortunately too frequently the case that a surgeon commences his examination in medico-legal contests after *having fully formed an opinion*, or at least a bias or prejudice. In many cases, in fact almost universally, this arises from the want of an analytical mind and of any carefully arranged system of examination. These are very serious defects in the expert witness, and when fully demonstrated should disqualify him from testifying. There is another serious defect frequently observed in the members of our profession, which sometimes has its origin in laziness, although occasionally in an inordinate greed, where the physician has been accustomed all his life to give an opinion to a patient without either an examination or thought. It will be perceived that I have not yet reached the case of the malingerer; but I have thus far merely paid my respects to those who aid and assist the malingerer. The malingerer is certainly entitled to a careful and thorough examination, and each surgeon employed in the case ought to proceed in his examination

with the same degree of care and caution that the most conscientious chemist would exercise when required to make a chemical analysis in the interest of justice. It therefore becomes highly important to learn the true history of the litigant, and likewise that of his father, mother, brothers, and sisters. All these matters have a special bearing on the case. It will be readily admitted that any person who has established a good character for truthfulness and honesty ought not to be classed with a mere adventurer. In the examination of all the factors involved in these cases of so-called "railway spine" the fact should never be lost sight of that *correct scientific opinions can only be reached by a systematic, methodical, and painstaking investigation.*

Let us here present a brief *résumé* of some of the points which have been more or less considered in this paper:

1. As used by those who first employed it, the term concussion was erroneously defined, and in its application, unfortunately restricted to the brain. These errors had their origin in a deficient knowledge of the pathological lesions engendered by concussive force.

2. Concussion is a more or less violent shaking or agitation of various organs in the body, producing functional derangements dependent on vasomotor changes and well-defined pathological lesions.

3. Concussion may be either general or special—general when several organs are involved at the same time, or special when the entire concussive force is expended on a single organ.

4. The symptoms arising from concussion will

chiefly depend on the extent of the pathological lesions and the functions of the organs.

5. The frequency with which concussion will occur in any particular organ will depend on the point to which the concussive force is applied; the relative weight of the organ; the character of its texture; the protection afforded by its anatomical surroundings.

6. The fifth statement supplies the explanation of the comparatively frequent occurrence of concussion of the brain, lungs, liver, and kidneys, as compared with that of the spinal cord.

7. Concussion of the brain and spinal cord is always attended with immediate symptoms.

8. Concussion of the brain or spinal cord without immediate symptoms is a mere hypothesis, a myth, and has no real existence.

9. There is no pathognomonic symptom of concussion in any organ of the body, but a thorough, systematic, methodical examination by a skilled surgeon will secure a correct diagnosis.

Finally, I desire to reiterate the fifth conclusion, expressed by Dr. Henry Hollingsworth Smith in a paper which he read before the surgical section of the American Medical Association in 1889, and which is as follows: "No physician should go into court and swear that a plaintiff has had a concussion of the spinal cord, or of its nerves, unless he has proved the disturbance of the normal functions of the cord, as shown in sensation, or motion, or both, and that the symptoms appeared *soon* after the injury."

— THE —

Assurance Medical Society.

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Possible Pitfalls in Life Assurance Examination, and Remarks on Malingering.

By F. PARKES WEBER, M.A., M.D., F.R.C.P., Lond.

London:
Printed by C. E. GRAY
36, Kennington Park Road, S.E.

1918.

— THE —

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Possible Pitfalls in Life Assurance Examination, and Remarks on Malingering.

By F. PARKES WEBER, M.A., M.D., F.R.C.P., Lond.

(Presidential Address before the Assurance Medical Society, January 9th, 1918.

In thanking the Society for electing me their President, I feel that it must be a difficult task for me to worthily occupy a post which has been in turn held by my father, by one of my distinguished masters at St. Bartholomew's Hospital, and by several others whom I admire and honour.

EXAMPLES OF DISEASE WHICH MAY ESCAPE DETECTION AT LIFE ASSURANCE EXAMINATIONS.

Arterial hyperpiesis, with or without granular kidneys. It is certainly quite easy at a single examination to overlook a case of hypertension if one does not employ the sphygmomanometer, even when the brachial systolic blood pressure is as high as 190 or 200 mm. Hg. Here we have the grave risk of premature death from such causes as cerebral hæmorrhage, &c. In many cases the urine may be found on repeated testings to be free from albumin, and tube-casts may be absent or only one or two hyaline casts may be discovered by the help of the centrifugal machine, as they may be sometimes in the urine of healthy persons.* Opthalmoscopic examination may likewise show nothing abnormal in this class of cases. The heart may not appear enlarged by ordinary examination, notably in fat or fleshy subjects; moreover, a moderate amount

*By the help of the centrifugal machine red blood corpuscles may also sometimes be detected in the urine of healthy persons.

of pulmonary emphysema may mask hypertrophy of the left ventricle.

Disease of the myocardium or coronary arteries of the heart in syphilitic subjects. In early cases, without knowing the subjective symptoms, one may easily overlook disease of this nature at a life assurance examination. Moreover, a history of past syphilis may not be forthcoming. The Wassermann reaction is not likely to be taken. A first attack of angina pectoris may be fatal. Other grave symptoms of myocardial disease may suddenly develop in young or middle-aged individuals. Sometimes considerable areas of the cardiac wall undergo degenerative changes owing to obstruction in their supply of arterial blood, and then death is likely to occur rather suddenly before a true cardiac aneurysm has developed.

Malignant tumours in the thorax or abdomen may easily be overlooked in an early stage of their development, especially at a single examination. Symptoms regarded as connected with ordinary dyspepsia may signify commencing cancer of the stomach. In some cases the persistent absence of free hydrochloric acid in the contents of the stomach after a test-breakfast might have furnished valuable evidence as to the nature of the case, though it must be admitted that free hydrochloric acid is probably more often absent in non-malignant conditions than is ordinarily supposed. It is not surprising that an operation for a malignant tumour in the abdominal viscera may occasionally be heard of in bona-fide cases not long after acceptance for life assurance, even when there was no suggestion of a tumour being present at the time of the life assurance examination.

Disease of the suprarenal glands, notably, primary malignant disease (malignant hypernephroma), may escape recognition at first. Tuberculous disease, with caseous degeneration, in one or both glands is often not recognised, even if there are asthenic symptoms, before the characteristic

cutaneous pigmentation of Addison's disease has developed. The following case bears on the question of the difficulty in the diagnosis of "latent Addison's disease," with asthenic symptoms and delayed recovery from infections, before the typical cutaneous pigmentation has developed.

In December, 1899, I saw a young man, aged 24 years, suffering from a pyrexial attack, which was regarded as influenza. The illness was accompanied by some pleurisy of the left base, and was followed by remarkable debility. There were several medical consultations about him, and he gradually apparently recovered. Later on, in 1901, when shooting in India, he was attacked by malaria, probably not a particularly severe attack, and was sent up to the hill-station of Darjeeling. There his condition was one of great asthenia without fever, and he had syncopal attacks, in one of which he died. Some pigmentation was observed in India, though none had been noted during the illness in England. A most careful post-mortem examination was made, and in a report sent over to a life assurance office in England it was stated that the suprarenal glands contained yellow caseous material and calcareous plates, and were matted together across the aorta. It seems to me that suprarenal disease (latent Addison's disease) was probably the true explanation of the asthenic condition connected with convalescence from the previous illness in England, and was perhaps also present when his life was heavily insured at ordinary rates by a great English life-assurance company before the illness in question. More than one English company was affected by his early death.

Cirrhosis of the liver is sometimes very hard to detect, especially in fat or apparently well-nourished persons, with thick abdominal walls which make palpation of the liver or spleen difficult. In such cases the disease, though it may have had an ordinary alcoholic origin, may be not only quiescent, but also actually latent. When the abdominal walls are thin or flabby, the hardened liver generally seems enlarged to palpitation, even when it is in reality more or less atrophic.

Evidently the position of the liver is changed in such cases, for the lower margin can usually be felt projecting below the costal margin. Slight enlargement of the spleen can often likewise be made out, and this together with an occasional subicteric tinge of the conjunctivae, and perhaps (when present), relatively dark-coloured urine, and excess of urobilin in the urine, may facilitate the diagnosis.

Splenic enlargement. Every medical examiner knows the difficulty in regard to *slight* chronic enlargement of the spleen, when the enlargement is not certainly the result of malaria. I suppose no candidate for life assurance with *great* enlargement of the spleen would be accepted, whatever the nature of the enlargement might be. I cannot resist drawing attention here to a certain class of splenomegaly cases, though a very rare one. A young woman or a young man suddenly complains of considerable pain in the upper part of the abdomen, possibly accompanied by temporary fever and a little ascites. The ascites soon disappears, but the spleen is found to be enlarged and there is probably slight anæmia. Soon the pain passes off, the patient feels well, and there may be no anæmia, but more or less enlargement of the spleen persists. In some cases there may be a little hæmatemesis early in the history of the disease. Later on the patient may seem and feel as well as any healthy person, in spite of the persistence of the splenomegaly. After a year or two the blood-count will probably show nothing abnormal excepting moderate leucopenia. For the clinical aspect of the case at this stage I have suggested the term "Anæmia splenica sine anæmia." Sooner or later, however, grave hæmatemesis is likely to occur, and the case will then probably be diagnosed as one of splenic anæmia or Banti's disease. But in this class of case, as a subsequent post-mortem examination shows, the real cause of the splenic enlargement, hæmatemesis, &c., is a kind of pylephlebitis adhæsiva, with resulting obstruction in the splenic and portal veins.* The obstruction to the return of

* For one of many typical examples of this kind of case, see F. Parkes Weber, "Sequel to the Case of Chronic Splenomegaly of Uncertain Origin, with Persistent Leucopenia, shown on January 12th, 1912," *Proc. Roy. Soc. Med., Clinical Section,* 1916, Vol. IX., pp. 1-6 (including the discussion which followed the paper).

the blood from the spleen may become compensated and the disease may remain quiescent for a long period, but fresh attacks of thrombosis in the portal vein are apt to occur, ultimately leading to complete obstruction. There may or may not be a history of traumatism at the commencement of the disease, and the ætiology of the thrombo-phlebitis in these cases has not been satisfactorily explained.

Pulmonary tuberculosis. I need scarcely mention the well-known danger at single life assurance examinations, of failing to recognise the presence of early pulmonary tuberculosis, especially when (for one reason or another) the medical examination is somewhat hasty or incomplete. *Early cases* are by no means always the most favourable cases of pulmonary tuberculosis. Apart from the general nutrition and build (and possibly the family history) one has no data at first to help one to form an opinion as to how the disease is likely to progress or how efficient ordinary hygienic and therapeutic measures are likely to show themselves. On the other hand, in old cases, and after the disease has been under observation for a considerable time, the resistance of the patient has been more or less estimated. When a person is not overwhelmed by the disease, even when it has considerably progressed, the power of gradually acquired resistance and compensation may prove to be tremendous. A man may be slowly "chased through life" by the disease and yet live to a fair, or even old, age. The disease, although very advanced and after extensive cavernous formation has taken place, may ultimately become quite quiescent or obsolete, and death may occur from other causes. In September, 1909, I saw a gentleman (W.M.M.) with extensive cavernous signs over the upper part of the left lung, back and front. He was 67 years old, but looked rather younger. Cavity-formation had occurred, according to the history, in the upper part of the left lung after an attack of measles at the age of 16 years. At 38 years of age he is said to have had a bad "cold" so that "the hole in his left lung got disturbed," and the pulmonary tuberculosis became active again. Since then he had

been much abroad, in Egypt, Malta, the West Indies, and Madeira, but especially in the Canary Islands. For some time he had a house near Las Palmas in Grand Canary. A tragical complication for the patient was the death of his wife (to whose devoted attention he owed so much), by suicide, a few months before I saw him, in 1909. He died at Pau in January, 1913, at 70 years of age.

Diabetes Mellitus. When diabetics are passed in life assurance examinations, the most probable reason is neglect or mistake in urine-examination, but it must not be forgotten that by means of the modern fasting treatment the urine can often be rendered temporarily free from sugar in the case of young patients (often apparently well-nourished), precisely those who are almost certain to die early from diabetic coma. Such cases would, I believe, often be passed, if fraudulently sent up for life assurance examination after a preparatory course of fasting treatment. In 1909, before the general introduction of the treatment of diabetes mellitus by Allen's fasting method ("alimentary rest"), the urine in a grave case under my care was temporarily freed from sugar in the following way.* The patient was a young woman, aged 23 years, an in-patient at the hospital. The sugar in the urine was reduced by dieting to about 70 grammes for the 24 hours. On September 10th, 1909, the diet was temporarily limited to 200-300 grammes of olive oil in the day, with as much water as she wished for, and a codein-phosphate pill (which the patient had been taking three times a day) was continued. As a result of this "disguised starvation" method sugar completely disappeared from the urine on September 12th. On September 13th I ordered a return to the special diet which she had previously been having for her diabetes, and on the next day the urine was found to contain 4.5 per cent. sugar again. During the days of exclusive olive oil diet the patient

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*Cf. F. Parkes Weber, "Diabetes Mellitus and Life Assurance Examinations," *Brit Med. Journ.*, 1913, Vol. I., p. 73.

did not feel hungry or as if she was being starved. Once or twice the oil, she stated, made her feel a little sick, but she looked well and had her usual, somewhat florid, complexion. She lost only about 4lbs. in body weight. She was not emaciated, and, on the whole, gained weight whilst she was in the hospital. This case was certainly one of grave diabetes mellitus, and afterwards died, as I heard, from diabetic coma, but surely a life examiner, had he examined her when the sugar disappeared, might have been deceived.

Appendicitis. During an interval after recurrent slight attacks of appendicitis, which have not been recognised as such, an applicant may be mistakenly accepted for life assurance at ordinary rates, and not long afterwards may fall a victim to an attack of the same disease, but of much greater severity. Such cases illustrate the importance and well-known occasional difficulty of eliciting and correctly interpreting data in the past medical history, which the applicant himself may genuinely believe to be of no particular consequence.

Syphilis and so-called parasyphilis of the nervous system. Nervous syphilis is supposed by some to have become relatively more frequent since the introduction of the methods of treatment by Salvarsan and Neosalvarsan. The blood-serum and cerebrospinal fluid are naturally very seldom (if ever) tested, regarding the Wassermann reaction, for purposes of life assurance, and it is quite possible that persons actually suffering from headache, etc., connected with commencing nervous syphilis, may occasionally be accepted for life assurance. Future statistics of the great life assurance companies as to the causes of death in early claims will doubtless throw some light on these questions. Similarly, persons with commencing tabes dorsalis or general paralysis must sometimes be unwittingly accepted for life assurance—especially if no history of previous syphilis is forthcoming. I doubt whether as yet Argyll-Robertson pupils are sufficiently examined for and the knee-jerks tested as a routine part of life assurance

examinations. But occasionally at (several) repeated examinations knee-jerks cannot be elicited in neurasthenic, and even in apparently quite healthy, individuals. One must acknowledge that tabetic symptoms, such as tabetic pains, tabetic paræsthesiæ, gastric crises, and tabetic urinary troubles, are frequently supposed to be due to other causes. It may be noted, however, that patients with tabes dorsalis often live a long time, and in some cases the life assurance companies would not be losers if tabetics were accepted by mistake. Tabetics in whom optic nerve atrophy occurs early seem often to have a relatively mild form of the disease in other respects—so much so that, I believe, in France, about Charcot's time, a kind of "arrested tabes dorsalis with blindness" was spoken of—that is to say, a class of tabetics was recognised whose symptoms (i.e., the progress of their disease) were arrested by (or, rather after) the development of optic nerve atrophy.

MALINGERING.

Knowledge in regard to the simulation of symptoms and diseases is of extreme importance for workmen's and accident assurance. The combination of simulated with genuine signs or symptoms is often especially difficult to detect. Functional nervous diseases and simulation of organic diseases are not rarely associated with each other. The artificial skin affections which I have seen have been chiefly in young women with abnormal psychical states. Unfortunately in these cases, as in other cases of artificially produced disease, though the artificiality of the disease may often be recognised, the exact means whereby the condition is produced frequently escapes detection.

Most of us recently have had to pay attention to the question of various forms of simulation in men. Amongst prisoners of war simulation of disease for purposes of repatriation tends, of course, to be regarded as fair play and as rather creditable than discreditable, if it is successful. In such persons, however, as in accident insurance cases and "traumatic

neurasthenia," there may be a real functional nervous element combined with the simulation of disease. In some cases a functional nervous disease may constitute the chief trouble, not combined with actual simulation, but fostered by a desire not to recover until liberty is regained, just as ordinary traumatic neurasthenia may be kept up and fostered by a desire to obtain monetary compensation, etc. An intelligent and highly skilled business man, aged 36 years, had right hemiparesis and what seemed to be a kind of myoclonus (paramyoclonus) multiplex. He could not speak, except in a whisper, and could not walk. He was allowed by the military authorities to return to his native land, and I afterwards heard that not long after his return sudden recovery of speech and power of walking followed the shock of a fall from a tram-car.

This reminds me of some remarks in a recent paper by E. Farquhar Buzzard,* in the course of which the author stated that he mistrusted the complete genuineness of many of the reported sudden recoveries from functional nervous disorders (including functional loss of speech, &c.) following (i.e., as result of) shocks. He suspected that the patients began to be tired of their loss of speech, &c., and began to be sure that their trouble was no longer really necessary; he thought that they then gladly accepted some "shock" or other exciting circumstance as a kind of "excuse" to recover suddenly. I wish some beneficial "shock" had occurred in an aggravated case of "chorea rhythmica" (functional) which was under my case for a long time.† The patient was a man, aged 38 years, of Hebrew origin. When he was being examined by a doctor, the rhythmic movements of the neck and arms used to become increased in rate and amplitude and force. They quieted down somewhat when he was left to himself, and ceased altogether during sleep. They became more violent when anyone tried to restrain them by force in

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* E. F. Buzzard, "Warfare on the Brain," *Lancet*, London, 1916, Vol. II., p. 1097.

†F. Parkes Weber, "Chorea Rhythmica in a Man," *Proceedings of the Royal Society of Medicine, Clinical Section*, 1916-1917, Vol. X., p. 6.

any way. He said he could not walk, but he was able to move his lower extremities when he was sitting or lying down. He gave one to understand that he could speak only in a very slow and jerky manner, as if battling against some obstruction in articulation. On the other hand, he could eat, drink, and dress himself fairly well, when left alone, without any assistance. He was at first not losing weight, but afterwards he lost slightly. He was apparently free from any visceral disease. His knee-jerks were exaggerated; no ankle clonus could be obtained; his plantar reflexes were difficult to obtain, but, when obtained at all, they were of the flexor type. Obviously psychical factors played an important part in the case. The patient was removed to another hospital and I lost sight of him, but perhaps he has been allowed to regain his liberty, and in that case rapid improvement may have taken place.*

In some cases I believe it is very difficult to distinguish between what one might call bona fide functional paresis (with limping on one lower extremity) and actual simulation, and equally difficult is it to distinguish between genuine painful remnants of sciatica and simulation.

A remarkable case of simulated amblyopia was complicated by a temporary attack of genuine optic neuritis. The case was that of an intelligent man, aged about 45 years, with special knowledge of chemistry. He was repeatedly examined by two ophthalmic experts, and there can be no doubt whatever that typical optic neuritis was actually present for a time; it could not have been simulated. He, however, apparently managed for purposes of simulation to have his pupils generally under the influence of atropin, but occasionally, when examined unexpectedly, his pupils were found to react to light. On an occasion, later on, when he met one of the eye-doctors unexpectedly, he committed the involuntary mistake of addressing the doctor, before the latter had spoken or given the patient any opportunity of recognis-

*I hear that he has been liberated and that improvement has taken place. F. P. W.

ing him, except by sight. As to the possible causes of the attack of true optic neuritis in this case it may be remarked that the patient's blood-serum gave a positive Wassermann reaction, that he complained of (possibly genuine) headaches, that he was given antisyphilitic treatment, and that the Wassermann reaction afterwards became negative.

One day this patient's urine was found to have a specific gravity of 1.057, which I think was probably due to some common table salt having been emptied into it. It is quite possible that the patient had noticed that on ordinary urinometers used in England the portion of the scale above 1.030 is marked **Diabetes** on the reverse side. Owing to this curious statement, perpetuated on nearly all these instruments, as used by doctors and in hospitals, the idea may suddenly have occurred to him that by artificially raising the specific gravity of his urine he might simulate an important symptom of diabetes mellitus. If so, he probably gave up the idea when he found that, owing to the absence of sugar from his urine, the disease in question could at once be excluded.

Dr. Hingston Fox, in proposing a vote of thanks to the President for his paper, remarked on a few points by way of suggestion. "Increased blood pressure would be detected by using the sphygnomanometer in all cases of suspicion, and in all cases after the age of 45. Degeneration of the coronary arteries is an important condition, and hard to recognise. Abnormality in the rhythm of the heart may help in some cases. A case some years ago may illustrate the risk of overlooking early malignant disease of the stomach. A gentleman of about 65 was examined, not by me, for life assurance for a very large amount. He was a thin, spare man, with dyspeptic symptoms, but he was passed as a first class life, the amount being distributed over a number of companies. In about a year death occurred from malignant disease of the stomach. Where dyspeptic symptoms exist great care must be exercised. Apropos of pulmonary tuberculosis, the President's case of a cavity existing from youth up to the age of 70, reminds me of one I saw more than 30 years ago

with the late Dr. Wilson Fox. The latter remarked to me then that cavities were capable sometimes of "living dry"; that is of passing into a condition in which they continue for a long time practically unchanged, doing no harm, and the patient ultimately dying of something else. The case in question was that of a lady with such cavities, and she is still living, about 75 years of age. In life examinations the taking of the patient's temperature is very advisable if there is any suspicion of tuberculosis. With regard to glycosuria, we must recognise that if a patient is sufficiently well coached, and ingenious, and has a manner capable of carrying his plot through, it may be impossible for the most acute examiner to detect the condition, if the sugar has been temporarily excluded from the urine by some such means as those alluded to by the President. But happily for us there are few persons who could carry the thing through so effectually. Most careful examiners would discover, from their enquiries as to diet, as to when the patient had last seen a doctor, and so forth, that there was something not quite straight about the case. They might even, like Dr. Pavy, give the candidate a plate of jam to eat, and tell him to return in an hour, when the matter would be cleared up. With regard to syphilis, I must plead guilty to not examining for the Argyll-Robertson phenomenon, as a matter of rule, in all cases of life assurance. I do, however, strictly examine the knee-jerks. As an instance of their absence in some non-tabetic cases, an extremely hard-working professional man—he worked, in fact, much too hard, and dieted himself very badly—came to me to be examined for life assurance. I was quite unable to get his knee-jerks, although I saw him more than once, and tried all the various little dodges for inducing reluctant knee-jerks to show themselves. I was, of course, suspicious of the case, but could find no other symptoms of tabes; the eyes were practically normal, there was no inco-ordination, and I had good reason for thinking him free from a history of syphilis. I saw him many times subsequently—in fact, he put himself under my care to a certain extent—but there were no further symptoms, and I became convinced that it was, in

act, a somewhat peculiar case of neurasthenia. On one or wo occasions, at a later stage, I obtained a little knee-jerk n one side, but with this exception they were always absent, nd I believe they continue to be absent. Still, there is no loubt that in nineteen cases out of twenty, the absence of nee-jerks would have a very definite organic cause. The President's remarks with regard to the long life of some abetic cases command my full assent. I have known cases f men who have lived to be nearly 80 years of age; but they were in good circumstances and took very good care of themelves.

Dr. Arthur T. Davies, in seconding the vote of thanks, quoted a case related in Hilton Fagges "Text Book of Medicine," vol. II. page 151, where a man seen by Sir Thomas Watson, between 40 and 50 years of age, was on his way home from Scotland (where he had been deerstalking and shooting grouse) when he was seized one night in a London hotel with deadly faintness, very rapid breathing, and severe pain referred to the sternum. He had before been gradually losing flesh and strength, but the only definite symptoms of which he complained were sour eructations, loss of appetite, and repugnance to solid food. Sir Thomas Watson could detect no disease; he found the epigastrium full and pulpy. The next night the gentleman had a similar paroxysm and died. The larger curvature of the stomach presented throughout its whole extent a mass of scirrhus, while the cardiac and pyloric orifices were free. Dr. Davies remembered the case of a well-known man who died eighteen months after examination by a very able examiner, and who had only complained of a few dyspeptic symptoms, and was found post mortem to have malignant disease of the stomach. It seems quite impossible for the medical examiner to avoid falling into such pitfalls with nothing to guide him; even if he suspects serious disease he cannot insist on making the applicant have a "test meal," and even this test, as pointed out by the President, is by no means an infallible one. An insurance examiner is not, after all, strictly speaking, a clinical

examiner. With regard to the case of tuberculosis mentioned by the President, it seemed to him that the reason for his being able to live so long was because he was in the position to travel to and reside in such places as the Canaries, Egypt, and elsewhere. Had the case occurred in a lower class of life it would probably have ended earlier. With regard to tabes and the prolongation of their lives, a friend of his had told him that he had seen such a case who, after examination by the Army doctors, had been sent to the front.

Dr. Otto May: With regard to the question whether disease of the coronary arteries produces any characteristic change in the rhythm of the heart, I do not think any useful rule can be established. I imagine there might well be a perfectly normal rhythm coupled with the presence of fairly extensive disease. I am convinced that there is more to be learnt from the estimation of the heart's functional powers by means of certain exercises than by all the methods of physical examination. Most of us, I imagine, have had unfortunate experiences in accepting lives which have become early claims from heart failure, where the ordinary methods of physical examination of the heart have failed to show any defect. With regard to the question of tabes dorsalis, I know of one interesting case in which a Company took a tabetic subject deliberately, and they were well justified by the result. The subject in question told the agent that he was not well, and that he was suffering from nervous disease; and the examining doctor found that there was a loss of knee-jerks and Argyll-Robertson pupils. In spite of that he was let in at first-class rates, and he is still alive, aged 75. I know him to have had tabes for over thirty years, and the company in question seem to have realised that tabes is compatible with a very long spell of life. This, no doubt, is an exceptional case, and I do not advance it as a justification for accepting all tabetic subjects at first-class rates, but each such case may well be considered on its merits, and not declined merely on the strength of a diagnosis of this disease.

Dr. Collier: With reference to the cases of heart degenera-

ion mentioned by Dr. May, I came across an interesting case ome four or five years ago. The examination was made on Friday, and a report came through the following day accordng to which everything was absolutely normal; the family istory was perfectly all right, and the man was recommended or acceptance at ordinary rates, and was in fact so accepted. The acceptance was posted on the Saturday, and would have eached him on the Monday morning; but on the Sunday he ied. The post-mortem examination revealed the atheroma f the coronary arteries, and some dilatation of the heart.

Now the examination in that case was carried out by a doctor who was well recognised as being exceedingly careful, nd whose reports were constantly taken. My experience has been that many people over 50 years of age have weak heart ounds; when one listens carefully they are found to be disinctly more weak than those of a great number of average nen out of the street. The blood pressure test may disclose a state of things which may be called normal, say 120 or 130. The other point is that one always gets a high diastolic; he only real test in such cases is the heart's response to effort. It is necessary to see what is the result of his actually raising himself up so many stairs, after which the heart should be again carefully examined with the patient occupying both the upright and the recumbent position. In my opinion t is impossible wholly to guard against such claims; they do not necessarily imply want of care in making the examinaion, and it is no doubt often the case that with the greatest possible care the examiner has been unable to detect some defect which has existed.

The President (Dr. Weber), in replying, said: In the course of the discussion some interesting points have been brought out. Dr. Withers Green has asked me a question with regard to local hyperpiesis, but I cannot help thinking hat he was really referring to local thickening of the arteries, which is not quite the same thing. Local thickening of the arteries is, of course, well known to occur in the arms of workmen who use their arms a great deal, but that is not the ame thing as hyperpiesis.

Dr. Withers Green: What sort of pressure do you get?

The **President**: During rest from work there would not necessarily be abnormality in blood pressure. With regard to Dr. Hingston Fox's remark as to the knee-jerk being occasionally absent in apparently healthy persons, and persons who live long, it is certainly true that in some such persons a knee-jerk cannot be elicited on repeated examinations. With regard to cirrhosis of the liver, I agree with Dr. Hingston Fox that when cirrhosis of the liver is suspected, a dark colour of the urine and excess of urobilin may give a useful hint; but I am not prepared to say that the urine is dark coloured in every case of hepatic cirrhosis, or at all times. With Dr. Hingston Fox's consent I shall insert a reference to these points in the text of my address.