does not produce any effect in a healthy subject—that sugar shows itself in the urine. As the power falls shorter and shorter of what is natural, so a less and less quantity of carbohydrate can be taken without running to sugar in the urine. Each case has its own special limit of assimilative power associated with it. When carbohydrate is taken within the limit of power of disposing of it there is no sugar in the urine. When carbohydrate, on the other hand, is taken beyond the limit of assimilative power possessed sugar is eliminated and the amount eliminated stands in proportion to the extent to which the ingestion has exceeded the capacity for proper disposal. As long as sugar is present in the urine evidence exists that the system is being supplied with more carbohydrate than it can properly dispose of. To supply more stands contrary to rational procedure, as it simply adds to the surplus to be eliminated, and in doing so adds to the deviation from the natural state arising from the improper passage of sugar through the system already existing. Experience shows that this passage of sugar through the system not only constitutes the source of the symptoms of diabetes but leads to a growth of the condition at the foundation of the disease—I mean leads to whatever assimilative power exists becoming further diminished. The reverse, however, is what is wanted. If the protoplasmic or assimilative power could suddenly be restored to its proper capacity there would be an immediate disappearance of diabetes. Carbohydrate in ordinary amount could then be taken, and from the natural power of properly disposing of it existing no opportunity would be afforded for its escape with the urine. The aim before us in the treatment of diabetes is to bring back the missing assimilative power, and in many cases amongst patients above the middle period of life this is more or less completely to be done. For its attainment nature requires to be aided by dietetic management directed to keeping the system in a natural state, or, in other words, keeping it free from the unnatural passage of carbohydrate through it; but restriction from carbohydrate food should not be carried beyond what is necessary to keep the urine free from sugar. If it is, a loss of weight occurs, and it is often found in the successful management of a case that after a while a loss of weight begins to show itself upon a continuance of the restricted diet. With freedom of the urine from sugar, it affords an indication that more or less restoration of assimilative power has taken place. A certain amount of carbohydrate can now be disposed of, and it happens, as experience shows, that if it is not given its want is manifested by the setting in of loss of weight. For a system of dieting to be rationally conducted it requires to be shaped to the requirements of each particular case. Time does not permit of my saying more to you upon this matter here, but I have entered fully into it in my recently published work on “The Physiology of the Carbohydrates; their application as Food and relation to Diabetes.”

Under the view I have placed before you there is nothing mysterious or inscrutable about the nature of diabetes. The whole thing lies in a nutshell. Carbohydrate matter ought to be disposed of in such a manner as to be prevented reaching the general circulation as sugar. If not so disposed of it simply passes through the system in an unutilised state and is eliminated as waste material with the urine. The natural disposal of carbohydrate is effected by protoplasmic assimilative action. Persons have hitherto devoted their attention to the effect of ferment action upon the carbohydrates; but this means, looked at in relation to the matter before us, giving attention in the wrong direction. Ferment action prepares for application by protoplasmic action; but it does not in itself in a direct manner lead to application. It is protoplasmic action, therefore, which requires to be studied in connexion with the question of the disposal of carbohydrate matter in the system.

WHY SHOULD PROTOPLASM FAIL TO PERFORM ITS PROPER RÔLE?

Why should protoplasm fail to exercise the power belonging to it of synthesising the carbohydrate into proteid, transforming it into fat, and transmuting it down to glycogen? This is the next point which presents itself for consideration. I do not think it depends upon the protoplasm itself. It is a property of protoplasm, whether it belongs to the animal or vegetable kingdom, whether it exists in complex or simple organisms, to possess and exercise the power of producing the effects I have referred to, and I do not think the failure of action can be assigned intrinsically to the protoplasm, but that it is due to the

conditions under which the protoplasm is placed. I, further, cannot help thinking—and I have thought so for years,—upon the strength of all I know in connexion with diabetes, that the exercise of protoplasmic power is dependent upon the existence of a proper condition of the blood in relation to the amount of oxygen present. The condition of the blood in this respect is influenced by that of the bloodvessels, and the bloodvessels, as we know, are under the control of nerve influence. We are thus carried to the nervous system for the source of the alteration of the blood which, I think, interferes with the proper exercise of protoplasmic power.

From evidence of a varied character adducible, a super-oxygenated condition of the blood may be said to lead to the production of saccharine urine. It seems to interfere through its influence on protoplasmic action with the passage of carbohydrate matter in its proper direction and to favour its passage in the direction of glucose. With dilated arterioles, from a paralysing influence derived from the nervous system, the blood traverses the capillaries in such a manner as not to become properly de-arterialised, thus establishing a super-oxygenated state. As I have said, evidence is adducible that with the occurrence of a super-oxygenated state of the blood saccharine urine is producible.

Paralysed arterioles of the chylipoietic viscera will lead to an alteration of the condition of the blood going to the liver in the portal vein. There are some cases of diabetes where the fault is limited to association with assimilative action. In these, sugar disappears from the urine when a restricted diet is followed. In others, no amount of restriction of diet will remove the sugar from the urine. Not only is assimilative action here at fault, but a cleavage of sugar from the proteid of the tissues may be looked upon as occurring. Possibly a ferment becomes developed in the blood and produces the effect, for the change is of a nature to be produced in this way. In the diabetes resulting from the administration of phloridzin, a principle obtainable from the bark of the stem and roots of the cherry tree, evidence points to the sugar, certainly in part, being derived from the tissues, and I see no other explanation than that the effect is attributable to ferment action.

And now, Mr. President and Gentlemen, the hour informs me that my task is finished. I much appreciate the patient attention which has been given to these lectures. You all know the subject is one to which I have devoted a life's work. I have confidence, as far as confidence can exist, in what I have placed before you standing in accord with truth. It now remains, however, for the minds of others to be brought to bear upon the matter. Whatever the issue, I can truly say, as one on the College Roll, that I have used my utmost endeavours to carry out the exhortation to the Fellows of the College of our illustrious predecessor, Harvey, “to search and follow out the secrets of nature by way of experiment.”

THE IMPROBABILITY OF A PARASITIC ORIGIN OF MALIGNANT DISEASE.

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In February, 1882, I read a paper on a subject that until then had escaped attention—namely, the fact that malignant disease not infrequently follows with very considerable rapidity an injury such as a fracture or a severe bruise. I called this event, or sequence of events, “acute traumatic malignancy,” and took occasion to foreshadow certain views which I more fully developed in a paper read in March, 1884, before the Pathological Society. Repugnant as I am to repetitions, yet it is necessary that those views be again formulated by short quotations from the latter paper. “The distinctions between carcinoma and sarcoma are not generic, but are merely sufficient to constitute two species of one genus which includes both—viz., malignant growth.” “Where sarcomata were first formed into a distinct group it was asserted of them that they are encapsuled, non-infiltrating, that they do not affect neighbouring lymphatic glands; but being developed from the walls of bloodvessels only infect the system by way of the blood.” “These doctrines are however, untenable. Sarcoma is occasionally without a capsule or often possesses only a very imperfect one; it is whether encapsuled or not, always infiltrating. It grows from intervacular spaces, not from the walls of vessels

and it very frequently, as in the case shown, affects the lymphatics." "The cachexia following sarcoma is indistinguishable from that produced by carcinoma." The view that the malignancy of both neoplasms is identical and that such quality must be regarded as a pathological entity was hardly well received at that time, but it is now in the writings of pathologists tacitly, if not in express words, admitted. In the ensuing remarks, therefore, all tumours possessing that special quality will be considered as a genus, the various arrangements, shape, and parentage of cells serving only to mark species or classes. Tumours which are clinically malignant are distinguished by their richness in cells, packed together closely—i.e., separated by very little and generally imperfect intercellular tissue; or, to put it in a form which more nearly, to my mind, represents the truth, such tumours are composed of cells which have entirely, or in very great part, merged all other functions in that of reproduction, and this, therefore, is excessive. Yet this is only an effect; the cause or causes calling forth this superabundant cell reproduction is and has been for a very long time the object of intense investigation. Stimulated by discoveries as to a microbic origin of many specific diseases, especially by Koch's success in cultivating a tubercle bacillus, a great many pathologists all over the world concluded on some such origin of carcinoma. Sir James Paget's Morton Lecture (1887), of which I must say more presently, greatly abetted this view, showing as it did a strong leaning to, nay, a conviction in its favour. Thus during about eight years research has been chiefly led in this direction, and has been carried along three lines—clinical observation, experiment, and microscopic investigation. I propose in this first paper to examine the clinico-pathological deductions upon which the prevalent opinions regarding the parasitic origin of cancer have been founded, or at least by which they have been supported.

With all the deference and respect which are due to his great name, I cannot but think that the first Morton lecturer has fallen into a fallacy by over-estimating the likeness to cancer of certain specific diseases and by under-estimating their differences. He names these diseases as "syphilis, tubercle, glanders, leprosy, actinomycosis," and points out some undoubted similarities which, however, seem pressed rather far. We judge, he says, of certain fevers, in which as yet no microbe has been discovered, that nevertheless they are of microbic origin, because they are so like one or two other kinds of fever in which bacteria have been found; therefore we ought to judge cancer to be so because of its resemblance in some points to the above-named diseases. But the one great factor upon which above all others we found our deduction of its origin is the infectious,¹ often also the inoculable, nature of the fever or specific disease. Sir James Paget dismisses this non-infectious and non-inoculable quality of malignancy with the simple words, "but these are negatives." Yet surely a stronger "No" would be difficult to pronounce. It is this one fact—viz., the contagious, infectious, epidemic, or inoculable quality of some fevers and certain diseases—which leads to the conclusion that they are the result of bacillar, parasitic, or microbic origin; surely, then, the absence of this special quality far outweighs the presence of certain more or less indistinct resemblances. It is impossible to conceive any disease more uncommunicable than malignancy. To take at present mere clinical evidence (the experimental will come hereafter) it may be asserted that cancer has been known for at least 2500 years. During that period hundreds of thousands of operations have been performed, and doubtless many accidental wounds inflicted; many nurses with sores on their hands have smeared them in cancerous discharges; many men have had connexion with women who had (knowing it or not) cancer of the uterus. In all those years not a single instance of contracting the complaint has been reported.

I now put together as illustrating and completing each other certain clinical facts. Cancer is sometimes in the same patient acquired in some part previously healthy by mere prolonged contact with a carcinomatous tumour; it may thus be carried from one lip or from one labium pudendæ to the other, or transference may be effected along a continuity of surface, as when in visceral cancer nodules of the same disease are found scattered about the peritoneum or more commonly collected on prominent folds

or in recesses of that membrane. Secondary (sometimes falsely called metastatic) growths form in the lymphatic system, often also in more distant and less evidently connected parts, frequently in the liver after cancer of the rectum. These contact, continuity, and secondary nodules have the same micro-anatomy as the primary growth, and some writers have attempted to deduce from that fact a proof of microbic origin. Yet the only deduction to be derived from this similarity leads in a directly contrary direction. For it is readily conceivable that a cell of squamous-celled, of round-celled, or of melanotic cancer transferred to another part of the body by lymph or blood-stream would in that new place grow into a tumour histologically like its progenitor; but it is impossible to conceive that a parasite from any one of these deposited elsewhere must produce, if anything, a cancer like its original habitat unless we be prepared to assume that each form of carcinoma is due to a special genus of parasite which can produce that particular kind, and no other kind, of neoplasm. Let us also consider how very easily epithelial cells take root and grow on any but very unfitting soil. I, like other operators, have again and again transferred large pieces of sterilised epidermis to fresh wounds; every surgeon has practised skin grafting: for the former a reliable nidus is the surface of a perfectly fresh wound, for the latter an ulcer with not too spongy granulations. If, then, an ulcerating cancer lie in direct contact with a healthy skin surface, that surface soon becomes irritated, inflamed, and loses its epidermis; by-and-bye a cancerous epithelial cell adheres there and grows.² The same transference, however, happens in other epithelial growths and conditions, as, for instance, in epithelial warts; also in suppurations—an ulcer of the gum very often calls forth a like condition on that part of the cheek which lies immediately opposite to it. Common skin warts, such as are so frequent on children's hands, are often promulgated from the inner side of one finger to that of another. If a hydatid bursts into the peritoneum, its hooklets and small daughter cysts are not, as a rule, scattered broadcast over the membrane, but are caught and retained in precisely the same places as those which are subject to what I have called continuity nodules. This shows that whatever carries the cancer are also masses—small, it is true, but large enough to be retained by projections or hollows, a quality hardly to be predicated of almost ultra-microscopic living atoms. Moreover, it tends to show that the things carrying the disease are passively subject to the laws of gravity, for they fall to the lowest parts of the cavity, as do all substances which have no power of independent motion, such as a parasite must have in order to infect anything—i.e., in order to penetrate into an epithelial cell or, further still, into its nucleus.

All these points, then—the transference by contact, by continuity of surface, by blood or lymph stream, and especially the fact that in all these cases the progeny is anatomically like the progenitor³—all these phenomena, which have been so much used in order to prop the microbic theory, are, in fact, quite adverse to such hypothesis; in other words, cancer and sarcoma, whatever their origin may be, are carried from place to place by cells or by nuclei detached from the primary growth. In connexion with this part of my subject, too, I must point to what appears to me to be another false analogy in the writings of the upholders of the supposititious cancer parasite. It would, however, be well to begin by saying that I yield to none in the high respect which I entertain for the most careful work of Messrs. Shattock and Ballance, as also for the honest, straightforward manner in which they have reported their results; but they lie open to the observation that even before commencing their work they were convinced of the parasitic theory—their labours have all been aimed at furnishing its proof, they have been undertaken and carried on with a bias. Especially is this bias remarkable in the analogies they draw between tubercle and malignancy. They say,⁴ "What is true of tubercle is almost certainly true of cancer," but this, with all deference, is not so. A bacillus, specific of tubercle, has been found and cultivated. Tubercle has often been successfully inoculated.

¹ The reader is particularly requested to bear in mind the difference between the above term and the word "infective": the former means conveyance of disease from one person to another, the latter that some malady primarily local invades after a time other parts and then the whole body of the patient.

² We have seen that, though this takes place from one part to another of the cancerous patient, such transference never occurs from a cancerous person to a healthy one; this will be further elucidated in the sequel.

³ I suppose this to be what is meant by "spermatic influence" a term which has always struck me as peculiarly infelicitous.

⁴ Transactions of the Pathological Society, vol. xxxviii., p. 417.

Tubercle has been traced to ingestion of milk furnished by tuberculous cows. The eating of tuberculous meat has more than once been followed by infection of the eater. The two first propositions have already been shown to be untrue of cancer. The third proposition in no way applies to cancer. As an instance: In the early part of 1876 I was asked to see a patient, and diagnosed scirrhus of the right breast. She was about five months and a half with child—her first pregnancy. Operation at that time was declined; in due course she bore a healthy child, which she suckled from both breasts. After about five months the supply, having been for some time insufficient, almost entirely ceased. I was called in again, not having seen her in the interval. The scirrhus had not greatly increased in size, but there were two enlarged glands in the axilla. I removed the breast and cleared out the axilla; she made a good recovery. The tumour was found to be a very hard scirrhus with a red point of softening in the centre. At the end of 1891 this patient died from broncho-pneumonia following influenza. The infant, who was suckled from a cancerous mamma, was strong and healthy. I see her, now about seventeen years and a half old (not as a patient), from time to time, and never in my life saw a stronger and heartier looking girl. As to the fourth proposition, animals, especially rats, who are of all quadrupeds particularly prone to cancer, have again and again been fed with lumps of scirrhus, the result being uniformly negative. Further, as to the “constitutional origin” of cancer without local manifestation and its comparison with general tuberculosis the supposed analogy does not hold. There is in malignancy nothing comparable to that condition called “scrofula,” “strumous diathesis,” or “constitutional tendency,” which so often enables us to predict imminent tuberculosis; there is none of that anæmic failing of the vital powers so often immediately precedent of such attack. Again, is there such a thing as general or systemic malignancy without local manifestation? It is to be supposed that everyone has seen in the post-mortem room, as I have not infrequently done, in the medulla or cancelli of bones, in the retro-pleural or retro-peritoneal spaces, malignant tumours so deeply situated as to be undiscoverable and unsuspected during life. If in such case malignant cachexia with multiple tumours had destroyed life, shall the morbid anatomist be misled into concluding that the case is one of general or systemic “carcinomatosis” without primary local manifestation?

Lastly, must we conclude from the fact that bacilli form from their own excreta or from their reactions on the fluids of our systems a poisonous compound that infects the blood—must we conclude from this fact that malignant cachexia is necessarily produced by the same or analogous agency? The late Mr. Marshall was wrong in saying in his Morton Lecture that carcinoma and sarcoma are acid in their reaction. A freshly excised malignant tumour, or, still more strikingly, such a tumour incised for diagnostic purposes before removal, is decidedly alkaline. The acid reaction never, in my experience, appears until after three—more commonly after five—hours. There are between malignant cachexia and the toxic effect of bacteria many points of difference which are plain to careful observation, but hardly amenable to verbal description. The odour of a newly incised scirrhus or round-celled sarcoma is that of a cadaver of from four to six hours' demise, its alkaline reaction, the numerous degenerations microscopically observable, the high probability (to say the least) that degeneration is not the only condition, but that the decease of many cells—an interstitial death of cell tissue—must constantly be taking place: all these point to the same conclusion, as does the difference of symptoms—namely, that malignant cachexia is a slow poisoning by a peculiar form of poison evolved not by the constantly reproduced, but by the continually dying, cells of the malignant tumour.

(To be continued.)

THE Royal British Nurses' Association announce their first course of educational lectures for the coming autumn. This comprises five subjects, each to be treated by an experienced lecturer, as follows: Elementary Anatomy, Mr. W. J. Walsham, F.R.C.S.; Physiology, Dr. A. Schofield; Hygiene, Dr. Louis Parkes; Practical Nursing, Miss Isla Stewart, Matron of St. Bartholomew's Hospital; Domestic Economy and Ward Management, Miss de Pledge, Matron of Chelsea Infirmary. Particulars as to fees, dates, hours, &c., can be obtained of the Secretary, 17, Old Cavendish-street, W.

NEUROLOGICAL FRAGMENTS.

By J. HUGHLINGS JACKSON, M.D. ST. AND., F.R.S.,

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No. X.

ON SLIGHT AND SEVERE CEREBRAL PAROXYSMS (EPILEPTIC ATTACKS) WITH AUDITORY WARNING; SLIGHT PAROXYSMS WITH DEAFNESS AND THE SPECIAL IMPERCEPTION CALLED “WORD-BLINDNESS”; SPECTRAL WORDS (AUDITORY); INABILITY TO SPEAK AND TO WRITE.

IN a paper published in *Brain* in July, 1888, “On a Particular Variety of Epilepsy (‘Intellectual Aura’),” I remarked (p. 191), “There is, I am convinced, in or after certain paroxysms of epilepsy temporary ‘word-blindness’; certainly in one patient of mine, who had a ‘warning’ by noise. I could not make out that this patient was at the same time ‘word-deaf,’ but thought his temporary deafness was ordinary deafness. Still there may have been ‘word-deafness.’” In this note I give details of this case. I have in the title of this note used the term “cerebral paroxysm” as well as the term “epilepsy.” We have not only to try to determine how far a patient's paroxysms approach or reach the type epilepsy, but also how they are departures from normal states. The term “epilepsy” is time honoured, and thus it often seems to be taken as a satisfactory statement of a case to say that it is one of epilepsy with this or that warning—as if epilepsy were some entity with different warnings in different patients. I submit that it is well also to consider that the warning in any paroxysm signifies the seat of a discharging lesion, that the warning is in that sense localising. Whilst, on the study of paroxysms by type, we may speak of some cases of epilepsy beginning by warnings of smell, others by warnings of colour, and others by warnings of noise, we should also recognise that there are in the cases instanced three varieties of cerebral paroxysms—one depending on a discharging lesion of a cortical centre for smell, another on such a lesion of a cortical centre for vision, and the third on such a lesion of a cortical centre for hearing. In other words, we should study the epilepsy, or cerebral paroxysm, of each patient on its own merits. To prevent misunderstanding I will use both the term “epilepsy” and the term “cerebral paroxysm.”

In the case related in this note there was, I submit, a discharging lesion of some part of the auditory centre of Ferrier and of that part alone (left half of the brain, I suppose). The discharge presumably spread from this fulminating point to normal cells—not necessarily to those morphologically nearest, but rather to those “physiologically nearest” (I mean to those normal cells which are united to the highly unstable cells of the discharging lesion by lines of least resistance). Those who have pondered over diagrams intended to illustrate the cerebral mechanism of speaking, reading, and writing, and of reception of the speech of others will see what is here meant. Probably in this case several of the phenomena mentioned were the after-effects of the discharge, were correlative with exhaustion of cells discharged in the paroxysm, and were post paroxysmal, post epileptic. There very likely occur cerebral paroxysms with an auditory warning without the special imperceptions word-deafness or word-blindness, and without inability to speak; but more careful inquiries have to be made in such cases. Much, no doubt, depends on whether the discharging lesion is of a part of the right or left half of the brain. Cerebral paroxysms with the phenomena mentioned have occurred in one case under my observation without any sort of auditory warning.

An intelligent man twenty-one years of age consulted me in March, 1882 for epileptic attacks; he had, he told me, the first at Christmas, 1881. This fit began by a noise, auditory warning; he became deaf; he could not see people speaking to him (their lips moving), but could not hear them; he ran to the door and fell on his face; he became unconscious and bit his tongue: his mother said he foamed at the mouth and “struggled”; he was, or was partly, conscious again in about ten minutes. I did not hear of any actions after this fit. He had had ten or twelve severe seizures since, in some of which he bit his tongue. The fit at Christmas,