to be renal calculus, suppuration, and discharge of renal parenchyma and its substitution by fat.

The condition under consideration, though a rare one, must not be left out of calculation by the nephrectomist.

CASE.—Alfred Rowen, aged 24, was admitted into the Birmingham General Hospital February 16th, 1883. He was a broad-built, well-nourished man. When seen in the casualty-room at 10 A.M., he had extreme dyspnoea, sitting with his arms leaning on the arms of the chair. The *alae nasi* and muscles of extraordinary respiration were working; his face was covered with beads of perspiration, and a little dusky. His mother, in answer to questions, said: "My son was operated on for stone in the bladder twenty years ago. For many years, he had known that his kidneys were diseased, because he sometimes passed matter in the urine. Occasionally he wetted his bed. Up to three weeks ago, I considered him a strong man; he never ailed, he ate well, and worked hard. Three weeks ago, he passed blood in his water, and vomited blood; he only vomited on that one occasion; at that time, he commenced making a noise in his throat, especially when asleep at night, and so loud was it that it kept awake fellow-lodgers, and, in consequence, he had to sleep in a separate room. He said he could not help it, and that he felt as though he had thrushes and skylarks in his windpipe." During the last fortnight, he had been off work, and a great deal in the house. He had been very drowsy, sleeping during the day in his chair for two or three hours at a time. He had frequently played cards; and, on many occasions, he had suddenly jumped up, and said: "I must leave off, I have got the cramps in my fingers and legs." The fingers and legs would be drawn during these cramps, which lasted about ten minutes at a time.

When examined in the ward, his breathing was very laboured. The dyspnoea was essentially inspiratory. The suprasternal notch, the lower intercostal spaces, and epigastrium, sank in during inspiration. Laryngeal stridor could be heard at the other end of the ward. He was unable to speak, but protruded his tongue when asked to do so. His pulse was 120; temperature 100°, respirations 40. Nothing abnormal could be heard in connection with the heart. On rapidly examining the lungs, mucous crepitant *râles* were heard over both bases behind. Examination of the fauces showed nothing. His legs and feet were not oedematous. Two ounces of urine were obtained, which was found to be alkaline, and to contain albumen and a large amount of pus. He became more comatose, the dyspnoea greater, and the breathing more stridulous. At 1 P.M. tracheotomy was performed, apparently without pain, but without manifest relief. He died at 4 P.M. on the day of admission.

Post Mortem Examination.—The body was well-nourished. The larynx was purple and oedematous. The lungs were oedematous. The heart was normal. The brain was not examined. The other viscera, except the kidneys, were free from morbid change. Both kidneys were imbedded in fat. The left kidney, which was half as large again as normally, on section, was found to be transformed into true adipose tissue. Its capsule was thin. At its hilum, there was a mass of tough, dense tissue, like cicatricial tissue, which extended to the centre of the organ, and in it was impacted a triangular calculus, the size of a tamarind stone; this mass was found to be composed of the obliterated pelvis and ureter, with blood-vessels mostly collapsed, and connective tissue. The adipose tissue was divided for the most part into pyramidal masses by paths of connective tissue, which radiated from the calculus. In this connective tissue ran large blood-vessels. The corpuscles of this tissue were proliferating, and absorbing fat. In two or three spots, near the periphery of the fatty mass, there were small patches of condensed tissue, which the microscope showed to be the remnants of atrophied renal tissue. The ureter remained as an impervious cord. In the right kidney, there were areas in various stages of degeneration; the secreting tissue was reduced to about one-third its normal amount, and this was intensely inflamed. There were circumscribed masses of putty-like matter, and several thick-walled empty sacs; in one sac was a smooth, round calculus, the size of a pea. The pelvis, which was thickened and dilated, contained a calculus, weighing half an ounce.*

REMARKS.—The case shows that life and apparent health may be maintained, when but a small amount of secreting tissue of one kidney only persists. The left kidney had been transformed into fat for many years; and doubtless, for some years, but a small portion of the right kidney had been capable of secretion. It appears that it was only when the remnant of the secreting tissue was acutely inflamed, about three weeks before death, that uræmic symptoms

manifested themselves. The immediate cause of death is obscure. The laryngeal stridor present on admission, and which was of three weeks' standing, coupled with the fact that the dyspnoea was essentially inspiratory, led to the belief that the dyspnoea was the result of oedema of the larynx, and that the coma was due to imperfect oxidation of blood; the continuance of the dyspnoea and coma, after tracheotomy, went to show that they were due to some other cause, probably uræmia.

THE EPIDEMICS OF ENTERIC FEVER IN THE TRANSVAAL, ZULU, AND EGYPTIAN WARS COMPARED.

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BEFORE comparing the epidemic of enteric fever that followed the occupation of the Transvaal in 1878, and the Zulu war in the following year, with that of the late expedition in Egypt, I will put a few queries as to what is meant by enteric fever, its production, contagion, etc.

Is it to be understood that a specific germ produces a specific and non-failing fever of unvarying type, as propounded by Budd and others? If so, I fear the following facts will lead to more perplexity than ever, and we shall fail to reconcile them with the modern outbreaks of this disease, be they civil or military. But, if the possibility be admitted that there may be more than one form of this fever, in the same way that there may be more than one form of malarial fever produced under certain circumstances, with factors common to different countries, manifesting, it may be, the usual intestinal characteristic complications, then these facts may be reconcilable to new theories, and, under abler hands than mine, backed by statistical knowledge, may be the means of throwing some light on this increasingly complicated subject.

Modern research has effectually dispelled the idea of accident in the production of zymotic disease; and, daily, fresh discoveries pin down to a hard and fast line germs as centres of disease. I do not, therefore, mean to say that no infectious germ is present in enteric fever, any more than in relapsing fever; but, that causes foreign to the usual habits and customs necessary to the health of a man will produce certain symptoms recognisable as a fever which, for want of a more definite nomenclature, goes by the name of enteric at present.

"Contagium vivum seu animale," is as true now as in the days of the Romans, who firmly believed that poisons of malarial diseases consisted of living beings. Have they not received striking confirmation under the hands of Koch and others in their recent researches?

I cannot help thinking that a most important point in the theory of the production of epidemics, be they typhoid, typhus, or any other fever, is the vitality of the germ; always supposing, of course, that the presence of a germ is necessary to the production of infection, or of fever. One peculiarity of these germs is their rapid multiplication. Whether it be by fermentation, multiplication, by division, cell-contact, or some other plan, it is certain that rapid reproduction takes place, and with it infection. Now, can this reproduction take place in a perfectly healthy subject? Can the germs be carried in healthy blood for an indefinite time, subject to all the vicissitudes of climate and life, and still retain their reproducing or infective powers when favourable opportunities occur? Or, must their habitat at once be found in a system so slightly physiologically imperfect, a balance so equal in fact, that the slightest introduction of a poison renders the system pathological? If this latter idea be probable, then I can understand how a soldier taken from his quiet routine work at home in comfortable barracks, to live in a tropical climate, or undertake a campaign, when, in either case, exceptional duties are necessary under somewhat trying circumstances, may be a fit bed for the reproduction on an extension scale, of germs producing fever. But, is this fever typical enteric, or may it not be a variation of it? The atmosphere of Tel-el-Mahuta and Tel-el-Kebir produced no pestilence, although we slept on imperfectly buried bodies at the former place, and breathed a loathsome atmosphere for days at the latter place. Dysentery and diarrhoea there was in abundance, but not enteric fever. True, time was wanting to develop the period of latency; but would the conditions ever have ended in enteric fever without the presence of others capable of producing a similar disease? I use the word "develop" to refer to the reproduction of the specific germ only. Then, again, we have no evidence that enteric fever existed at those stations prior to their occupation by our troops,

* A section of the left kidney is shown in the lithograph.

had, then, the epidemic which subsequently occurred in Cairo, its origin at those stations? I am aware that it is within the limits of possibility that a special germ may have accompanied the troops from England, for I know no limit at present to the vitality of these germs, but it is rather vague.

This brings us to the question: Is enteric fever miasmatic, pythogenic, contagious, or a combination of these? I recollect a case in the Citadel Hospital, Cairo, where a man was admitted with a contusion over the nose. The cut did not heal rapidly, and he remained a considerable time in hospital. Three weeks after admission this man sickened and died from enteric fever, a most rapid and virulent case; the truth of the diagnosis being verified by *post mortem* examination; and yet we are told that direct contagion is doubtful, for the poison, like that of cholera, so Pettenkofer and others say, must go through an intermediate stage of development before it becomes infective. Another example. Many of our nurses in the hospitals at the Citadel, Abbasseyab, and Alexandria, were attacked with a mild form of this fever, and so were several of the orderlies. It may be taken for granted that all the usual sanitary precautions recommended in such epidemics were adopted, and yet direct contagion would seem to have been present. It must be remembered that these same sisters had not gone through the campaign, so that the cause of their illness could neither be laid to the water of the sweet water canal, nor to the atmosphere of Tel-el-Mahuta, or Kebir. Possibly it might have been of the "abortive" typhoid type spoken of by Liebermeister, where the temperature rose to 104° for a few days and suddenly subsided, leaving the patient free from fever, and in a fair way to convalescence. Many such cases did occur towards the end of the epidemic. But might this not have been another form of this fever of a contagious character? The local conditions in the hospital were alike for all, and gave one the impression of a system becoming saturated with a fever-stricken atmosphere, and breaking down in consequence. Towards the end of convalescence, in more than one case I saw this "saturation" so to speak, or, to be more correct, "hospitalisation," where patients, kept all through the disease in one room, would persist in a supernormal temperature, and nothing would break it, neither dieting or medicine; the very walls seemed to absorb the fever. A change to another room, or house, would break this at once. May not this have been the same fever seen among the nurses and orderlies?

We have hitherto been dealing with the contagion question. Now is enteric fever pythogenic? Decomposition, pure and simple, may, cause dysentery and diarrhoea, but can it produce the real enteric fever without the presence of the specific germ? Any answer must necessarily be speculative, so I will leave it, and content myself with comparing the epidemics that have come under my personal observation.

If the advocates of the theory that simple decomposing organic matter will produce the fever under consideration really believe what they teach, how can they reconcile their doctrine to the epidemic in the Transvaal? There were no dead bodies there, for a very good reason, there were no killed and wounded, and the water was as pure as one could desire; and yet we got the inevitable epidemic, and that among troops seasoned by residence in Malta and the Cape, but unseasoned to tent-life, and with it to the duties and fatigues inseparable from campaigning. If putrefying animal matter will produce enteric fever, why do not the Kafirs get it, who eat meat green from decomposition, and highly offensive to the smell? The Kafir is as keenly alive to the truth of the old saying, "Cleanliness is next to godliness," as any civilised being, yet he will eat the meat in a putrid state; and, in spite of this, enteric fever is unknown among his race. Again, surely the custom of eating game "high," as it is called, which prevails in the ranks of society, would have some influence in the production of this fever, if such were possible; but it is not so.

Now, let us look at the epidemic of this fever in Egypt. That the water was bad—unfit for drinking purposes, in fact—will be readily admitted. The foulness, however, was due chiefly to mud. I grant that dead bodies were occasionally found in the canal, but it was the exception, and their removal soon accomplished. The connection between the water and the common intestinal diseases is plain enough; not so enteric fever, however, for, even if faecal matter, in addition to the other foulness, were present, it by no means follows that the epidemic which followed in Cairo was so caused. It has yet to be proved that human excrement alone will produce this fever. And again, in the Transvaal and Zulu wars, where the water could not but be pure, the usual epidemic followed.

Tropical heat in some way, unexplainable at present, must have a powerful influence on this disease; for, go where we will—India

China, Egypt, or South Africa—all produce their typhoid fever among recent arrivals in the country, especially when campaigning is commenced in the hot months; for, strange to say, it does not accompany the troops for ever. We had no more enteric fever in the Transvaal after the epidemic had once expended its energy, even after a few weeks' rest, when we again set out on the march to Sikukuni's country, under as adverse circumstances as could well be conceived, as far as rations and tents were concerned. One great point, however, was always in our favour; a bountiful supply of pure water from the clear mountain streams was always procurable.

I am refraining from giving minute details as to the supposed causes of the outbreak of enteric fever during the Egyptian occupation, for they were exhaustively dealt with by the special correspondents to the BRITISH MEDICAL JOURNAL at the time. I hope now to bring forward a few more points not then noticed, more particularly bearing on the water as a supposed cause, and to compare them with other epidemics where the water was not in that suspicious state.

There were several peculiarities about the Egyptian epidemic worthy of notice, not the least being the absence of the usual "curves" in the temperature-charts. One case I recollect, of an officer who began a very serious and prolonged attack with a persistent night and morning temperature of 104° for three days, verified by more than one thermometer; then it broke down, and gave a few characteristic variations, but never settled down to the typical "ups" and "downs," so to speak.

In another case which I recollect came in, there was a high morning and low evening temperature, which persisted for many days, when suddenly it changed round to the usual evening exacerbations, without materially influencing the progress of the case in any way. Now, either we must discard temperatures as a diagnostic sign altogether, or the cases met with may not have been the true enteric fever, uncomplicated in character.

It can hardly be said that medicines influenced these cases, for little or none were given other than simple ones to combat some irregularity of the constitution; and, even had they been given, it would be hard to believe that they would have influenced so pointedly the course of the temperatures. Another marked peculiarity which may be said to have been almost persistent in this epidemic, was the slowness of the pulse, rarely rising above a hundred in the minute, except under excitement, or, later on in the case, from debility. It is to be regretted that *post mortem* examinations were not more systematically made. I know from experience how difficult and how unpleasant these are in tropical countries; but the idea was forced upon me that, had a skilled pathologist accompanied the expedition, as was done in the Crimea, many points might have been solved, among them being this slow action of the heart. One more peculiarity, and I have done, as far as this question is concerned. Relapses are said to occur in about 8 per cent. of enteric cases. Both in South Africa and in Egypt relapses were the rule instead of the exception, and yet the cases under observation certainly did not come within the category of relapsing fever as noticed by Griesinger in Cairo in the year 1850, in connection with the bilious typhoid and typhus then raging. They were simple relapses, accompanied by the usual gastric disturbances; and yet we were supposed to be dealing with plain unvarnished enteric fever.

We will turn to South Africa, and see how the case stands. It was about the middle of March, 1877, that the expeditionary force destined to overawe the Boers, and enforce the proclamation of annexation to be issued by Sir Theophilus Shepstone, left Pietermaritzburg, Natal. This force consisted of the 1st battalion 15th Light Infantry, under Colonel Montgomery; half a battery of Artillery, and details of Royal Engineers, Natal Mounted Police, etc., making a brigaded total of 1,000 men and two guns, under the command of Colonel, now Sir Charles Pearson, K.C.M.G.

It was in the days when the ranks were composed mostly of twenty-one years' men—men who had served for some time previously in most trying climates. Of course, there was a sprinkling of six and twelve years' men; but the majority were seasoned soldiers, who had seen a goodly lot of service, and some of it in Malta, not the best school in the world for campaigning. The average age of the force I am unable to give; but it could not have been as low as that in Egypt (twenty-four years about), for the short service had not then become general. Taking them all round, they were as fine a body of men as I ever saw begin a march. The truth of this was borne out by the few "casualties" on the line of march. A fortnight's marching brought us to Newcastle, on the borders of the Transvaal. Camp was pitched on a hill overlooking the town, and separated therefrom

by a river of considerable size—one of those numerous rivers arising in the noble Drakensberg close by, and which intersect the colony so plentifully. A rest of three weeks or so, awaiting orders to resume march from Sir T. Shepstone, was useful; and then the force set out for Pretoria, distant about two hundred and twenty miles. For the first thirty, the country traversed presented a series of mountain-spurs, each ravine having its mountain-stream fresh from the hills on the left. It is quite possible that, had a microscopic examination of the water been made, algæ and diatoms could have been discovered; but, as Professor De Chaumont says: "To condemn water on account of the presence of these living creatures, would be really to condemn all water, even rain." They may, therefore, be said to have contained pure water. Soon, however, this hilly country ceased, and a flat uninteresting plain took its place; a few stagnant pools supplanted the beautiful running mountain streams, and precautions had to be taken to prevent the men from using them. This was not difficult, as they did not suffer greatly from thirst. Night marching was resorted to; the men carried their rifles and ammunition only, and hourly halts were called. The result was little or no sickness, except the usual abrasions and excoriations. Diarrhoea was almost unknown, and sunstroke absolutely so. Even on this high flat plain—3,000 feet above the level of the sea—one of those mountain-streams was met with about every ten miles, when the men could drink their fill with impunity, and replenish their water-bottles. I cannot imagine campaigning under more favourable circumstances. The heat certainly was great at mid-day in the bell-tents; but the nights were cool—so cool, in fact, that I often marched on foot in preference to riding. A fortnight sufficed to cover the two hundred and twenty miles, and Pretoria was reached on May 4th, 1877. Our camp was pitched above and on a higher level than the town, and had running through it a clear mountain-stream, contamination being out of the question; the sanitary state of the camp and neighbourhood receiving special attention.

We arrived at Pretoria on May 4th. On the 14th of the same month, there were nine cases of enteric fever in hospital, and two officers down with it, and this in a force of a thousand men all told. That it was enteric fever I will answer for, for I made *post mortem* examinations on all fatal cases with a small bush-knife; and the truth of the diagnosis was demonstrated by the condition of the small intestines, which were extensively attacked by the characteristic ulcerations. It was a sudden outbreak, and as suddenly ceased, but the cases were numerous enough in proportion to warrant the application of the term epidemic to it. Some positively affirm that for the production of enteric fever a specific agent must be present, and that common fecal matter may produce diarrhoea, even of a febrile character, but not enteric fever. If this be correct, whence did the specific agent come in this case? The Transvaal had only been inhabited by Boers for a quarter of a century, and, as far I know, enteric fever was unknown among them. It comes to this, then either there was a specific germ carried with the troops from one knows not where, awaiting certain favourable circumstances for its growth, in which case this germ would be virtually everlasting, indestructible, capable of retaining vitality under almost any and every circumstance, in air, clothes, etc., capable of conveyance any distance and in any climate; or, there is no special germ producing enteric fever, but producing varied forms of fever with enteric complications, which fevers can be produced without specific agents, provided certain elements are in force dependent upon, and inseparable from, campaigning in tropical zones, or under somewhat similar states when an individual goes from a home to a tropical climate. We will now look at the enteric fever which accompanied the Zulu war. It must be remembered that the men forming this force (twenty-five thousand strong) came out straight from England. Operations were carried on in a comparatively young country, so far as European residents were concerned, and a persistence of this fever was not ascertained. Whence did the specific germ come from in this case? The large force was divided into several columns, one at Lower Tugela, one at Helpmakaar, and another at Utrecht. In all these columns, enteric fever showed itself; yet they were totally separate from one another, and the little communication between them that did exist was of the most indirect and roundabout form. Again, cases occurred at Maritzburg; men were left behind just after landing, before the poison, if it had been absorbed in the country, could have had time to develop. Others, again, were attacked after a lengthened life in tents, when they came back into the towns and took up their residence. How is this accounted for, if the specific agent theory be correct, or amenable to modern views? Rather was it not, as has been suggested by others, that "the altered circumstances under which the men lived led to an excessive phy-

siological action, showing itself primarily on the intestines, and, overstepping the bounds of health, became pathological, expending its force in the form of a fever with the above manifestations?" The water in this case could hardly be blamed, for it was practically pure. True, the streams before reaching us had to pass through a few Kafir villages, for it follows that they must live near this commodity as well as other people; still, I venture to repeat, that they are as cleanly in their habits, and as refined in their tastes, according to their lights, as the most refined civilian of the day. Contamination, therefore, was unlikely from them, even supposing, for the sake of argument, that fecal matter could produce enteric fever. In my notes on this fever made at the time, I find my impression then was that the epidemic was caused by drinking stagnant water. Subsequent experience has led me to doubt this impression, for it is very doubtful if much stagnant water was used by the men, even if it could have produced true enteric fever.

It comes to this: either the germs, if this theory be entertained, were carried from afar, or it is not necessary to have the presence of a specific poison to produce a fever, under special circumstances, manifesting intestinal complications. Here was an epidemic of enteric fever with pure water and fairly good sanitation. In Egypt, we also get it with impure water, but with other conditions similar. One thing was common to both: service under canvas, in a tropical or subtropical climate, under altered conditions from those in force in the native country of the men employed. Has the water so much to answer for? Are there not other and important factors at work producing similar results, and with equally fatal force? I offer no opinion, but ask others to make their own deductions.

ON THE ACTION OF VERY DILUTE SOLUTIONS OF ESERINE, AND THEIR USE IN THE TREATMENT OF WEAKNESS OF THE CILIARY MUSCLE.

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SOLUTIONS of the extract of Calabar bean, or of its active principle eserine, have for some years been used for the purpose of contracting the pupil or stimulating the ciliary muscle; but, as far as I can ascertain, these solutions have always been of a strength far greater than that of those, the use of which it is the object of this paper to advocate. Apparently, also, they have been recommended only in cases of decided paralysis of the pupil or the accommodation. Two important papers on the subject have been published. The first was read by Dr. Argyll Robertson before the Edinburgh Medico-Chirurgical Society in February 1863, when the use of solutions of the extract of Calabar bean was first recommended for cases of weakness or paralysis of the ciliary muscle. The second was by the late Mr. Socolberg Wells, in the *Medical Times and Gazette* for May 16th, 1863, recounting the effect on the pupil and accommodation of a strong solution of the extract (one minim corresponding to four grains of the bean; I may here mention that the yield of eserine is uncertain, so that the relative strength of it and the extract of the bean must be reckoned by the results of their application). Experiments were made upon Mr. Bowman, who graphically relates his own experience, and upon a patient with paralysis of the ciliary muscle and iris. Irregular and somewhat painful spasms of the muscle commenced after ten minutes, and continued for some hours, causing the nearer approach of both far and near points of vision. In Mr. Bowman's case, the pupil remained fully contracted for eighteen hours, and did not return to its normal size for three days. Attempts to use the eye while the drug was in full action caused much distress and pain in the eyeball. Mr. Wells ends his paper with the following remarks: "My next experiment will be to test the curative influence of the Calabar bean upon paralysis of the pupil and accommodation; and to ascertain whether we cannot succeed, by periodic applications of a very weak solution of the bean, in exciting the sphincter pupillæ and ciliary muscle to gentle contraction, without fatiguing and weakening them by over-stimulation." Accordingly, on reference to the last edition of his work on *Diseases of the Eye* (1873), I find that he relates his experience of the drug applied in cases of ciliary paralysis. But he seems to have used comparatively strong solutions, for he says that the drug should be applied of "a strength sufficient to cause considerable contraction of the pupil, and should be used at intervals of several days." Mr. Nettleship, in the appendix to his work on *Diseases of the Eye*,