An Address

ON

SOME DIAGNOSTIC FAILURES.

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BY

H. BATTY SHAW, M.D., F.R.C.P.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND TO THE HOSPITAL
FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON.

GENTLEMEN,—I propose to discuss with you some cases which I feel are worthy of notice, not only from the fact that you will hear of much that will excite your sympathy, but because it will give you the opportunity of listening to clinical accounts of difficult cases completed by *post-mortem* examination. When your secretary invited me to address you, several subjects were submitted to him, and the choice of the present subject is his. As to the mode of treatment of the subject I must be held entirely responsible, and I have followed out an old plan of never giving the diagnosis until my audience had had an opportunity of thinking it out for themselves. With this purpose in view, I will endeavour to describe my cases fully enough to give you the evidence which is essential and possible, then I will give you my own opinion of what I thought was the matter, and then your own conclusions and mine may be tested by the points discovered at the *post-mortem* examination. Where it is possible I will give you reasons for the failure of this and that clinical test and the precautions which may be taken to avoid your own and my mistakes in the future.

CASE I.—*An Example of Too Great a Reliance upon Clinical Pathology and Too Little upon Bedside Findings.*

I will ask you to picture to yourselves a well-nourished servant girl, aged 23. She had been noticed by those about her to have undergone some curious mental change during the year previous to admission to the hospital, for she had become almost a child again, and would even stop on her errands to play at any game with children in the streets, such as marbles, etc. She had also become more emotional and readily broke down into tears or developed attacks of hysterical laughter. Three weeks before admission she had developed occipital headache and a severe pain in the right lower chest, which was exaggerated by breathing. As an out-patient she was suspected of suffering from typhoid fever, and the agglutinative power of her blood against the typhoid bacillus was found to be positive in dilutions of 1 in 30, 1 in 50, and doubtfully of 1 in 100. On this latter piece of evidence she was admitted to the typhoid ward of the hospital, and the following signs were elicited: Some dullness and fine crepitations were heard at the right base where the pain was, but all these phenomena cleared up in a few days; there was no expectoration at any time of her illness. The temperature was always raised—from 99° to 102.6° during the stay in the hospital, which was of thirteen days' duration when she died; the pulse was 80 to 100 until the day of her death, when it reached 120 to 140; it was always regular. The impulse of the heart was in the fourth space external to the left nipple line; the area of percussion dullness was increased to the left, and there was a soft systolic murmur at the impulse. Vomiting was a feature throughout her illness. There were no typhoid spots; no distension of the abdomen until the last few days of life, and the spleen was always just palpable. There was some tenderness in the right iliac fossa, and the descending colon could be defined as if loaded with faeces; there was constipation requiring enemata; the appetite was bad. A second Widal test was carried out, and gave positive reactions in dilutions of 1 in 30, 1 in 50, and 1 in 100. The nervous system was quite normal in every respect, except that some days after admission she began to be delirious at night, and the headache persisted. On the day of her death the nervous phenomena developed—namely, a double extensor response and some deviation of the right eye to the right. Ophthalmoscopic examination of the right eye showed that the edge of the disc was a little blurred (? early papillitis), and nothing was discovered in the left eye. The persistence of headache and delirium led to the examination of the spinal fluid, which was drawn off

by lumbar puncture; it was found to be quite normal, showing no cellular content and no excess of albumin. The courses had been established at puberty, but shortly after failed, and never returned except as very "slight shows." There was a great deal of leucorrhoea, which was not offensive in odour, nor did it reveal the presence of the gonococcus on microscopic examination. Pelvic examination was carried out, and revealed no abnormality. The chest was examined radiographically, and appeared to be quite normal. The patient was diagnosed as one of typhoid fever, and so marked was the delirium that the patient was treated by the cold-bath method, which temporarily had the desired effect; the following day, however, she developed diffuse bronchitis, and the respirations advanced from 20–24 to 28–48 a minute; she became more delirious and comatose, and died on the thirteenth day of her stay in the hospital. The urine was free from any abnormality. The only illness from which she had ever suffered was some affection, it was said, of her heart two and a half years previously. The father was alive and said to be gouty; the mother had died of an unknown cause, and her brother and sister were healthy.

My own opinion of the diagnosis was that the girl had died of typhoid fever, and for this there was the crowning piece of evidence that the Widal test was positive. It would be wrong to say that there were no misgivings, for it was difficult to explain the persistence of the headache when the delirium supervened on such a diagnosis.

The *post-mortem* findings were as follows: There was no trace of typhoid ulceration of the intestine, the only ulceration present being tuberculous, and this was present in two or three places in the ileum, and the peritoneum over these ulcers was soldered on to the adjacent uterus; the heart was quite normal; an infarct was present at the base of the right lung. Evidently the cause of death was due to tuberculosis, for there was great flattening of the convolutions of the brain, the cerebral meninges were thickened and opaque, though no tubercles could be seen (there was no spinal meningitis). Both pleurae were universally adherent, and both lungs and the bronchial glands were freely sown with miliary tubercles. The cavity of the uterus was enlarged and held a drachm of pus, and the cavity walls were caseating; both Fallopian tubes were in a condition of pyosalpinx due to tuberculosis; the spleen showed no tubercles, but the kidneys showed many.

The comment I would pass on the case is this, that too much reliance was placed on the Widal reaction, nor am I in a position to state why it was positive. The golden clinical maxim should not have been disregarded, which says that in typhoid fever the headache gives place to delirium, whereas in meningitis it accompanies it. Too much reliance also was placed on the fact that the spinal fluid was normal. It is well known that the cerebral meninges may be inflamed and the spinal ones escape. It is difficult also to explain why the patient had shown such remarkable mental changes a year before her final illness, but it is conceivable that already meningitis had begun to develop, though not sufficiently to provoke more serious signs and symptoms. The infarction of the lung was probably due to some local thrombosis of a pulmonary artery, and not to an embolus. As for the development of double extensor plantar response, as subsequent details will show, this may quite well be dependent upon purely toxic causes of an origin quite independent of infection. Finally, the tuberculous salpingitis and metritis fully explained the disturbances of menstruation and the leucorrhoea. For the third time in the last few years I have thus met with severe leucorrhoea dependent upon tuberculous metritis.

CASE II.—*An Example of how One or Two Prominent Features Exclude all Thought of Disease of Systems other than the One Responsible for such Symptoms.*

The second case is that of a married woman, aged 40. Several years before admission she had been treated for pneumonia in the left side for three months in a London hospital. She appeared to recover from this entirely. She stated that since the birth of her last child, eight years ago, she had never been well and strong. During the fifteen months prior to admission she had been subject to shortness of breath—at some times worse than others—and had noticed occasionally that she had swelling of both feet, and for six months she had noticed that she had to get up at night three or four times to

pass water. The immediate causes for her seeking admission to the hospital were that swelling had developed in both legs and in the lower part of the body, had missed her chest, but caused a most remarkable puffiness of the eyelids so that she could scarcely see; and, secondly, that her friends had noticed that every now and again her face, instead of being very pale, which was almost natural to her, showed intense blueness. She had had no illness other than the pneumonia referred to, and had escaped the acute specifics of childhood. Her father had died of pulmonary tuberculosis, but the rest of her family, including her two children, were quite well and strong.

I do not remember ever seeing such a curious clinical picture as she presented on admission. She was quite unable to lie flat in bed, and was only comfortable when she rested with her chest and head on a pillow on a table placed across the bed. When she raised her head to answer questions, the combination of pallor and cyanosis and extreme facial dropsy was most remarkable. There were ascites and extreme dropsy of the lower part of the trunk and legs. The cardiac impulse was slightly more forcible than normal, and was placed in the fifth interspace, just internal to the nipple line; there was increase of cardiac dullness to the right; there was no murmur. The veins of the neck were very full, filling from below as well as from above, and they had a pulsation which was ventricular in time. There was diffuse bilateral bronchitis, and dullness at both bases behind. The respiration was always rapid, 48 to 64 a minute. The chest wall was very thinly covered, and was free from oedema. There was no cough or expectoration. The pulse was 96 on admission, but four days later rose to 128, and remained frequent till death. The temperature was subnormal till the last four days of life—that is, till after drainage had been carried out. Vaginal examination revealed nothing but the signs met with in cases of ascites. The very scanty urine was examined five times, and always revealed the presence of albumin, from a trace to a cloud; there was no pus or blood, but numerous granular and a few hyaline casts were found. She was only sick on two occasions on one day. The ascites and dropsy resisted all treatment by means of drugs, so that there was no alternative but to try the effect of drainage by means of incisions over the malleoli. By this means sixteen pints of fluid were removed, and by the tenth day after her admission she was feeling very much better, the facial dropsy disappeared, and the chest cleared so much that adventitious signs could be made out at the extreme bases only. The respiration and pulse, however, remained frequent, and eleven days after admission she began to wander, a few petechiae appeared on her feet, and she died thirteen days after admission. The funduses of the eyes were normal. The systolic blood pressure varied from 96 to 100 mm. There was marked pyorrhoea alveolaris.

The only point which seemed clear to me was that one cause of her death was the failure of the right heart, and I made the provisional diagnosis of morbus cordis.

The findings at the *post-mortem* examination were varied in the extreme. The pericardium contained 6 oz. of clear fluid, and was free from inflammation. The heart weighed 9 oz., and revealed no abnormality but a dilated right ventricle and enlargement of the tricuspid orifice. There were old cavities at the apices of the right upper and lower lobes, and the remainder of this lung was hyperaemic and showed many tubercles with fibrosis around them. The left lung and the bronchial glands showed tubercles; both pleurae were universally adherent. The intestines were normal, and there was some ascitic fluid present. The spleen was enlarged, and showed no tubercles. The liver showed some perihepatitis and slight "nutmeg" change. The kidneys were of normal size, each weighing $4\frac{1}{2}$ oz.; the capsule stripped with difficulty, and the surface was red and finely granular; the cortex was of normal width and the renal vessels were normal; cysts were seen in each kidney; microscopically, there was considerable interstitial fibrosis and round-celled infiltration; glomeruli as a rule were normal; vessels free from endarteritis. Some of these changes may have been suspected by you, but the following features were also discovered: The right middle ear was full of pus, there was a soft yellow tumour growing from the inner aspect of the dura mater indenting the right cerebellar lobe but not adherent to it, and of a non-inflammatory nature; there was a chronic gastric ulcer as large as a shilling near the

pylorus and lesser curvature, and having for its base the pancreas: there was tuberculous ulceration of the walls of the uterine cavity; both Fallopian tubes showed extensive tuberculous caseating pyosalpinx, and there were cysts in both ovaries, the larger being the size of an orange.

Comment is hardly necessary in such a case, except to point out the chagrin of the clinician concerned when, after diagnosing successfully one lesion, several other lesions he did not diagnose were brought out at the *post-mortem* examination—sclerosis of the kidneys, otitis media, a cerebellar meningeal tumour and gastric ulcer, and tuberculosis of the lungs, uterus, and Fallopian tubes, and, lastly, double ovarian cysts.

CASE III.—*An Example of the Confusion wrought in the Clinical Picture and of the Harm done to a Patient by Exclusion of Carbohydrate from the Diet.*

A married woman, aged 44, suffering from severe haematemesis—three pints during forty-eight hours before admission—giving the following history: Ten years and five years previously she had had severe dyspepsia, pain, and vomiting. Two years ago she had another bout of pain associated with nausea only, and a less severe attack had occurred four months before admission. There was nothing of moment in the family or personal history, except that it was known she had taken alcohol freely of late. She was well nourished but very pale; the mouth was dry and she was very thirsty. The pulse was 108, respirations 22, temperature normal. There was no abdominal tenderness. The maximum impulse was in the fifth space in the nipple line, and a loud systolic murmur was heard at the apex and at the pulmonary cartilage. The central and peripheral nervous system and the urinary functions were normal. She was at once put on rectal feeds.

On the day following admission—that is, April 23rd—she felt better, but was thirsty, gradually got more blanched, and became very restless. Morphine was administered. Pulse 96, temperature 98.8°, respiration 24. On April 24th she was better in the morning, but as the day wore on she became faint and restless again. The two systolic murmurs could not be separated, a very loud systolic murmur being heard all over the precordium. Pulse 112, temperature 100°, respiration 28. Morphine was again administered.

On April 25th, with blood still present in the stools and of recent date, it was decided to operate. Ether was administered after an injection of scopolamine, morphine, and atropine. A chronic ulcer was found near the cardiac orifice, 2 cm. in diameter. The ulcer was stitched across and gastro-jejunostomy was performed.

On April 26th, the day following the operation, she felt fairly well, and peptonized milk or albumin water was administered, which she retained well. The pulse was still rapid, 116. On April 27th the picture changed completely, and from being a placid woman, reasonable and appreciative of all that was being done for her, she became very restless and violent, and occasional quiet was varied by frequent delirious bouts, and the pulse failed. She presented much of the appearance now of grave mental disturbance, and, to our complete discomfiture, the plantar responses, from being flexor, were now extensor on both sides. Speculation as to the cause of this change was very varied, and included possibilities of delirium tremens, meningeal inflammation, cortical thrombosis, etc. Nothing more could be done than to administer sedatives, and she died at 3 p.m. on that day.

At the *post-mortem* examination the surgical operation was found to be quite successful, but a second small acute ulcer was found not far from the one operated upon. The liver was pale and showed fatty degeneration. The kidney capsule was rather freely adherent to the cortex, especially at the sites of small scars, which caused the surface to be irregular. The heart was dilated, and its muscle showed the familiar "tabby cat" striations. The brain and lungs were quite normal. In contrast with the first two cases, this one showed nothing at the *post-mortem* examination which could account for death or the development of the extensor responses.

I have no absolute proof, but I suspect that the prolonged use of rectal feeds without the use of glucose as an ingredient was responsible for the clinical puzzle and the death of the patient. My reasons for thinking so are that two other cases, one an elderly lady and the

other a child, both developed almost identical features after operation; the one was saved by giving large doses of homely sodium bicarbonate, and the other was given glucose—in the one case overcoming acidosis after development and the other preventing it assuming fatal proportions.

CASE IV.—An Example of how Suppression by the Patient of a Fact in her History completely Misted the Clinician, and also of the Fact that Treatment Destroyed the Patient.

This case is one of the circumstances connected with whose death became a subject of public interest. She was a single woman aged 23, a servant girl, and from a clinical and pathological point of view was of extreme interest. The details of the illness were not without their comical side, though the final opinion to be expressed upon them must be that it was extremely tragic and pitiable.

She was first of all admitted to the diphtheria ward of the hospital because both tonsils were covered with a whitish-yellow membrane. The Klebs-Loeffler bacillus was looked for, but the cultures, etc., were negative. For reasons connected with the cleaning of the ward, she was transferred to a fever hospital. The authorities there were also unable to find the Klebs-Loeffler bacillus, though the appearance of the throat left no doubt as to the nature of the illness. At the end of a fortnight we were requested to take her back, because, although the membrane had disappeared, the patient had vomited incessantly, bringing up blood, so that a diagnosis had been made of gastric ulcer complicating her diphtheria.

On readmission she was found to be wasted, and the abdomen was much hollowed and retracted, recalling the appearance of the abdomen (boat-shaped) met with in chronic meningitis. She complained of pain in the upper abdomen, but it was difficult to say whether it was due to subjacent ulceration or to the strain of the recti muscles brought about by the incessant vomiting. Despite her wasted appearance she retained a good colour. Beyond the fact that she was rather listless her nervous system was quite normal. The temperature was not normal, but rose now and again to about 100°; there was nothing suggestive in the chart beyond the irregularity of the fever. On one occasion a few râles were heard at the right apex; they soon disappeared, and as there was no sputum at that time tubercle bacilli could not be looked for. The heart revealed no abnormality, and the catamenia were quite regular up to six weeks before admission. The material vomited contained food taken, and was acid. In order to obviate any further vomiting she was fed per rectum, and continued to be so fed for eight weeks. I was so puzzled by the appearance of the abdomen and the symptomatology that I asked the advice of a surgical colleague, who was unable to say she had a gastric ulcer, and proposed not to operate. My own opinions varied; sometimes I felt convinced she had tuberculous peritonitis, at others that she was a case of anorexia nervosa, and always there was the possibility of a gastric ulcer. The case excited great interest, and I subsequently sought the opinion of a medical colleague, and on his expression of the opinion that she had a gastric ulcer, my surgical colleague was reluctantly persuaded to operate. Lateral nystagmus was observed. During the eight weeks prior to operation two superficial skin abscesses had developed—one on the right thigh and one on the back—they were opened, and contained staphylococci. Operation for gastric ulcer was performed on February 27th, and as a preliminary a soft oesophageal tube was introduced into the pharynx, but could not be passed successfully by several different operators; at last, with difficulty, a stiff tube was passed into the stomach. On exploring the stomach thickening was found near the pyloric orifice of the stomach; the stomach was opened, and after ascertaining the presence of an ulcer bordered by rather thick edges and much infiltration, the surgeon performed gastro-enterostomy.

The signs and symptoms which developed after the operation were most puzzling. The temperature now became more constantly elevated; the patient took food by the mouth and was less sick, but she was far from being much relieved. Pain developed at the front of the right chest low down, and a few râles were heard over the liver area. Yellow expectoration was coughed up, and on microscopic examination was found to be free from tubercle bacilli.

On the tenth day after the operation nervous mani-

festations set in, which were extremely difficult to explain. The external recti of both eyeballs became weak, so that there was a tendency for the optic axes to converge, although the patient could deflect each eyeball outwards in concomitant action. The lateral nystagmus noted before operation was still present and more exaggerated. The knee-jerks could not be obtained; there was no ankle clonus, no Achilles jerk, no Kernig's sign, but well marked and persistent double extensor plantar response was observed. There was no paralysis of the limb muscles, no sphincter weakness, and the mental processes were normal; there was no headache. The funduses of the eyes were examined; a fairly large haemorrhage of some days' date and some smaller ones were found in the right retina, and some older ones in the left eye. Four days after the onset of these nervous phenomena the patient sank and died.

Difficult as had been the diagnosis, up to the operation, it was still greater when the nervous signs had developed, and I could but feel that some grave change had occurred within the head, which had led at least to the development of a double extensor plantar response, meningitis being to me a likely explanation. Lumbar puncture was not performed as the patient was so ill.

At the post-mortem examination a small cavity was found at the apex of the right lung, and the lung in the neighbourhood showed numerous miliary tubercles. At the right base an irregular patch of pneumonia was found; the left lung was normal. The oesophagus at the level of the trachea was narrowed and the mucosa destroyed. An ulcer was found near the pylorus, which did not conform in type to the ordinary gastric ulcer, but which caused considerable stenosis. The operation area was quite healthy, and the gastro-enterostomy was quite successful. There was no peritonitis, and all the remaining organs, including the brain, were normal.

At this stage a communication was made by the mother to the effect that, knowing of the critical condition of her daughter, she had ransacked a chest of drawers and found an envelope containing a letter, in which the daughter made the statement that, fearing she would become pregnant, she had swallowed some hydrochloric acid.

It thus became clear what was the nature of the whitish-yellow membrane on the tonsils, and what was the cause of the oesophageal stricture (? basal pneumonia) and the gastric ulcer.

As to the nature of the toxic process which so long after the swallowing of the acid had led to retinal haemorrhages, to the paresis of the external recti, to the nystagmus, and to the double extensor plantar response, little can be advanced beyond the possibility that some or all were associated with the prolonged rectal feeding and acidosis. To complete the story it must be stated that the uterus was quite normal and there were no signs of pregnancy. So far as I know retinal haemorrhages have not been described as a feature of acidosis. The only truly satisfied observer was my house-physician, who had detected evanescent signs which made him think of pulmonary tuberculosis, exactly over the site of the tuberculous lesion found in the lungs.

CASE V.—An Example very probably of Quincke's Serous Meningitis.

A young woman aged 28, a telephone operator, on going home from work on January 13th, felt so out of sorts and "shivery," that she decided to call on her doctor on the way. He found she had a temperature of a little over 99°, and thinking she had a "cold," advised her to go to bed at once, and stay there for a day or so instead of continuing with her duties. She did so, but four days after seeing her doctor her "legs gave way" on trying to get up, and she gradually became lethargic and semicomatose. When I first saw her she had been in this condition for seventy-two hours; she had been persuaded to swallow food with difficulty, and I found her lying on her side curled up in bed. She was incontinent of water, and the bowels were constipated. Two years previous to this illness she had fallen down the spiral staircase in the telephone exchange; her legs were much bruised, and she was unable to work for three months. Even then she never became herself again. She had pain in the back of her neck, and instead of joining with the rest of the family in evening games and occupations, she would sit quietly apart from the rest, rather listless. Her father had died at the age of 52 of heart disease, but her mother, brothers, and sisters were quite healthy.

She was admitted to the hospital on January 22nd, and during her stay of a little over two days her temperature (rectal) never reached more than 97.4°, the heart-rate was (?) 40, 40, 60, 52, 50, 48, (?) 40, and 40 on the occasions on which it was counted, and the respiration-rate on the same occasions was (?) 12, 12, 16, 9, 10, (?) 16, and 10.

She was well developed and well nourished, almost completely unconscious, lay curled up in bed, and resisted all attempts to move her limbs. The breathing, though slow, did not show any periodicity. A faint *tache cérébrale* was elicited. Kernig's sign was obtainable on both sides; attempts to flex the neck did not provoke pain so far as could be told in her semiconscious state. The pupils did not contract briskly to light, and dilated and contracted in slow hippus without apparent cause; the conjunctiva was insensitive or almost so. Occasionally the eyes were kept constantly turned to the right. There was no paralysis of any of the muscles, no spasticity or flaccidity, but general tendency to flexion of the limbs. Occasionally there were slight movements of the arms and fingers. All the reflexes of the arms were absent or greatly depressed. Both knee-jerks and both Achilles jerks were absent, and the plantar responses, when obtained, were flexor; ankle clonus was absent, and with difficulty the abdominal reflexes were obtained in the upper left and lower right quadrants. The eye funduses showed no abnormality of the discs or retinae. Constipation was so marked as to require croton oil for its relief. With difficulty a catheter specimen of urine was obtained, and a good cloud of albumin was discovered on two occasions. There was no sugar. Feeding was carried out by nasal tube. Two attempts were made to obtain spinal fluid by puncture and without success. The difficulty of the case was so great that I asked a surgical colleague to confer with me. We both felt that some intracranial lesion was responsible for the symptoms and signs, and we were greatly influenced by the history of mental change, the pains in the neck, the slow pulse and remarkably slow respiration, the subnormal temperature, and the abolition of reflexes and incontinence of urine. The slowness of the pulse and respiration suggested some subtentorial pressure which was acting upon the medulla. Operation was carried out and the skull opened in the left cerebellar fossa, the opening being continued into the foramen magnum. The dura mater bulged and the cerebro-spinal fluid escaped through a small hole in it at considerable force.

The dura mater was opened freely, and further exploration was deferred until some more favourable opportunity. The opening of the dura mater was promptly followed by a restoration of the breathing to normal—that is, 16 a minute. Unfortunately, however, little else improved, and the patient sank and died within about twelve hours after the operation. I adhered to the possibility of a subtentorial tumour being the cause of the symptoms.

You will scarcely credit it, but the fact remains that the only abnormality found at the necropsy was a mild acute appendicitis, which obviously could not have been responsible for the changes which, beginning two years previously after the fall down the staircase, had culminated in such a curious final picture. The brain revealed no abnormal features, but then it must be remembered that operation with drainage of the cerebro-spinal fluid had probably altered the true condition of affairs. Judging by the clinical features and by the escape at the operation of cerebro-spinal fluid at considerable tension, it seems quite likely, in the absence of other pathological signs, that my patient was an example of Quincke's serous meningitis.

I now will task your patience by one further instance of diagnostic failure. The case is one which personally I feel is a milestone in my progress or the reverse on the road to becoming a physician. Like most beginners I have hitherto felt that molar and molecular death should be accompanied by the discovery of some physical or chemical signs indicative of the cause. In some of the cases I have rehearsed such cause was physical and in others there was reason to believe it was chemical. In the present case I am compelled to admit that the cause was one which conformed to neither, and, for want of evidence to the contrary, I am obliged to admit that at last I have seen death occur, not from anatomical disturbance, including trauma, and not from chemical poisoning, using the term in a broad sense, but from disturbance of vital function by depressing circumstances—indeed, I

have at last seen what might be called a physiological death. I do not say I was ignorant of such possibilities, but simply that I had never had the opportunity of seeing such a case and of verifying it by *post-mortem* examination.

CASE VI.—An Example of Death caused purely by Mental Depression and Excitement.

A gentleman who had amassed a considerable fortune decided at the age of 60 that he would retire from active work and spend the rest of his days in leisure and in the discharge of duties other than personal ones. He had always been a perfectly healthy man, had never known illness, was a keen sportsman, and led a life which was hygienically perfect. He came of a most healthy stock, free from any trace of insanity or mental weakness. When I say that he invested his wealth in one large commercial enterprise at the age of 60 in order to free himself from keeping watch over a multiplicity of investments, it may be questioned whether after all he had not betrayed his one and only departure from mental balance and sanity.

The fact remains that up to this date his business acumen and judgement had been of a first-rate order. The investment he made turned out badly, and though he was far removed from bankruptcy by this turn in his fortunes, he began to sleep badly a month before he died. Restlessness developed and within a week before death he showed his first delusion. Acute mania developed, and without showing any physical signs of ill-health, he died in a second attack of cardiac failure, the first occurring twelve hours beforehand. The *post-mortem* findings were *nil*. Such forms of death are familiar to alienists, but they are not familiar to those of us who do not spend our energies in the care of insane patients. The experience was to me unique, and seems to bring home to me that my diagnostic failures are after all nothing more than confessions of ignorance of pathology, of physiology, or of faulty history taking. This last case impresses me, and will do so for all time, with the fact that mind is superior to matter, that it can triumph over matter—in fine, the “ego” is after all an element which, if robbed of its normal balance, can play havoc with its tenement.

A COMPARISON OF INFLAMMATION AND TUMOUR FORMATION.*

BY

CHARLES F. M. SAINT, M.S.DURH., F.R.C.S.ENG.,

ASSISTANT SURGEON, HOSPITAL FOR SICK CHILDREN,
NEWCASTLE-ON-TYNE.

PROFESSOR RUTHERFORD MORISON has said that “the tissues react to irritation in two ways: by blood changes and cell proliferation.” Inflammation is defined as “the reaction of irritated and damaged tissues which still retain vitality,” or “a series of vital changes which occur in the tissues in response to irritation.” The irritants are either non-infective or infective; the former comprising mechanical, electrical, chemical and thermal agents, and such things as *x* rays, etc.; the latter including the micro-organisms.

Inflammation may be either acute or chronic, though all gradations are met. Both varieties may be produced by the non-infective or infective irritants. When due to the non-infective irritants the type is determined by the severity of the irritation, whereas, when due to the infective irritants, it is generally determined by their nature—for example, acute inflammation is most commonly produced by the pyogenic bacteria, while chronic inflammation is usually dependent upon tuberculous or syphilitic infection. The degree of inflammation depends, in the non-infective variety, upon the quantity of the causative stimulus, whereas in the infective it depends mainly on its quality.

In *acute inflammation* the blood changes are at a maximum, while the local cell proliferation is, speaking roughly, just sufficient for the repair of the tissues damaged in the process. In such a case the cell proliferation may lead to the regeneration of the tissue destroyed, or, as more usually occurs, the damage is

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