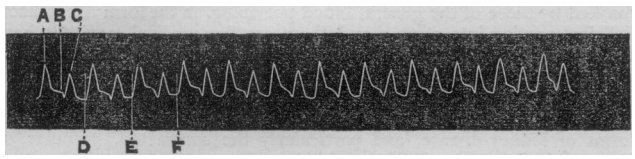
"typhoid" conditions of the system; while, on the other hand, in many cases, it is entirely absent. It has been attributed to a want of arterial tension; and this view was confirmed by experiments with the sphygmograph when that instrument was added to our *armamentarium*. Probably, too little heed has been given, in discussing this point, to the just distinction, first pointed out by Stahl, between a quick pulse and a frequent pulse; *i.e.*, between a rapid contraction of the left ventricle and a frequent repetition of its contractions. The sphygmograph has also discovered the very curious fact, that in most persons' pulses there is a



A The ordinary pulse-beat; B. The ordinary dicrotism; C. An extraordinary dicrotism. These three elements were all produced by one beat of the heart. (D to E and E to F are each single heart-beats.)

slight amount of dicrotism, too slight to be sensible to the touch of even the most skilled finger. Whilst by this instrument dicrotism has been detected in many cases of aneurism, this is the first, so far as I remember, in which it has been palpable to the unaided finger. There are also certain peculiarities in this case which serve to prove, almost to demonstration, that this dicrotism was directly due to the aneurism itself. Such a possibility suggested itself to the ingenious mind of M. Marey; but the circumstances of his case did not afford means of proof; and the dicrotism of aneurismal patients has been commonly held to be due to a relaxed state of the arteries, consequent upon the withdrawal of blood from the general circulation to fill the sac.

The double-beating of the pulse in this case was discovered not by the sphygmograph, but by the finger. The pulse was counted, and the heart-sounds and heart-beats were counted, and the rate of the first was found to be twice that of the last. The instrument did two things. It detected the minor dicrotism (B), and it gave a faithful and permanent record of that and of the major dicrotism (C).

Whilst there was at all times palpable dicrotism in all other parts of the arterial system which could be reached, there was none at any time in the aneurism itself, though the single pulsation of this was very forcible. It is not necessary to enter into the vexed theories of normal dicrotism; it is enough for us to know that all explanations refer it either to the elasticity of the aorta, or to waves or impulses reflected from the periphery of the circulation (the capillaries) or from the aortic valves.

Particular attention is now invited to the fact that, at the usual part of the pulse-trace, there is to be found an ordinary amount of dicrotism; whereas, subsequent to this, there appears a dicrotism, not only in itself of very unusual height, but also, which is much more remarkable, far transcending in altitude the previous dicrotic ascent. From this fact I infer, without hesitation, that the two elevations must depend upon different causes. For it is a physical fact that, in all intermittent effects of elasticity, the first is the greatest. For example, if an India-rubber band be stretched and allowed to contract an inch at a time, the force of the first contraction will be greater than that of any subsequent one. If, therefore, normal dicrotism depend solely upon aortic elasticity, it is impossible that a secondary dicrotic wave can exceed in height a primary one.

Again, in the case of all reflected waves, whether of fluids or liquids, the primary reflection is necessarily higher than any single succeeding one. An echo is a familiar example of this law. If, therefore, ordinary dicrotism depends solely upon reflection from the periphery, it is equally impossible that any secondary wave can exceed in height the primary one. The two waves, then, here cannot be due to the same cause; and, as we may assume that the minor is the normal wave, the major must be due to an abnormal cause. In seeking for this, we naturally turn to the aneurism. We find that it alone, of all parts of the arterial system, is monocrotic. We see, feel, and by the murmur hear, it contracting whilst other parts are expanding. We can estimate the force with which it contracts by feeling that with which it expands. We cannot doubt that this force is adequate to produce the secondary dicrotic wave, or, as we might term it, the tricrotism. We seek for an unusual cause; we find an unusual apparatus of adequate power; is it unreasonable to conclude that here is the cause we seek. If we admit this, it remains for future observation to determine how far this phenomenon may in future assist in the diagnosis of obscure internal aneurisms. At present, I restrict myself to averring that we know of no other disease capable of producing a secondary dicrotism, the trace of which exceeds in altitude a primary one.

ABSTRACT OF A CLINICAL LECTURE

ON THE

PATHOLOGY AND TREATMENT OF THE TYPHOID STATE IN DIFFERENT DISEASES.

By CHARLES MURCHISON, M.D., F.R.S.,
Fellow of the Royal College of Physicians; Physician and Lecturer on the Practice of Medicine at the Middlesex Hospital; etc.

I PURPOSE in the present lecture to direct your attention to several cases of disease, widely different in their nature, but in all of which there has been a similar group of symptoms, constituting the condition ordinarily known as the "typhoid state."

CASE I.—William D., aged 23, admitted October 26th, 1866, on the seventh day of an attack of typhus fever, with well-marked eruption, a dry, brown tongue, great delirium, and restlessness, and signs of congestion of the lungs. Marked improvement on the fifteenth day. On the eighteenth day, return of the febrile symptoms; an inflammatory swelling over the left parotid, and albumen in the urine. On the nineteenth day, frequent attacks of convulsions, which continued to recur until death by coma on the twentieth day.

After death.—Blood dark and fluid; great hypostatic congestion of lungs; spleen large and soft; kidney presenting the characters of a recent acute nephritis. A large quantity of clear fluid, containing urea, beneath the arachnoid, and in the lateral ventricles.

CASE II.—John F., aged 27, admitted December 3rd, 1866, on the ninth day of an attack of typhus, with well-marked eruption, a dry, brown tongue, and albuminuria. On the thirteenth day, violent delirium, followed by stupor, floccitatio, and involuntary evacuations, which continued until death on the seventeenth day. No convulsions.

After death.—Blood dark and fluid. Extreme hypostatic congestion of both lungs, the two together weighing sixty-eight ounces. Both kidneys greatly enlarged, weighing together fifteen ounces and a half, their surfaces smooth, and capsules non-adherent, their cortices extremely congested, and the tubes gorged with granular epithelium. Much clear fluid, in which a considerable quantity of urea was detected, at the base of the brain, and in the lateral ventricles.

CASE III.—John S., aged 44, admitted into the London Fever Hospital, on October 12th, 1863, on the seventh day of an attack of typhus, with copious eruption. On the ninth day, acute delirium, followed by stupor, complete unconsciousness, low muttering delirium, and involuntary motions. On the thirteenth day, urine very scanty and smoky, containing epithelial and blood-casts. During the following night, a fit of convulsions, lasting a quarter of an hour, and death by coma half an hour afterwards.

After death.—Blood dark and fluid. Extreme hypostatic congestion of the lungs. Spleen large and soft. The two kidneys together weighing fourteen ounces and a half, their capsules separating readily, and their cortices quite smooth, but very friable, intensely congested, with blood dripping in large quantity from the cut surface; all the uriniferous tubes gorged with opaque granular epithelium, and many of them containing extravasated blood. Much clear fluid in the lateral ventricles and at the base of the brain, which was not examined for urea.

CASE IV.—James P., aged 73, admitted November 13th, 1866, with an eruption of pemphigus over the greater part of the body. Many years before, he had suffered from "typhus," and from a rheumatic attack, but he had never had dropsy. Two years before, the eruption had appeared on his legs, and within the last three months it had spread over his body. Two or three days after the patient's admission, he became very restless and delirious at night; his tongue was dry and brown, and coarse moist *râles* could be heard over the back of the lungs; the urine was scanty, but, though examined repeatedly, contained not a trace of albumen, and there was no dropsy. Death by coma occurred on November 21st.

After death.—The blood dark and fluid. The kidneys contracted and granular, their capsules firmly adherent, and their cortices having well-nigh disappeared. Œdema and hypostatic congestion of the lungs. Much clear fluid, containing urea, at the base of the brain, and in the lateral ventricles.

CASE V.—John K., aged 49, admitted October 27th, 1866, with a gangrenous ulcer of big toe, the result of an injury, jaundice, and albuminuria. About November 15th, the patient became very restless and delirious at night; the tongue was dry and brown; there were signs of hypostatic congestion of the lungs; and the gangrene began to spread up the leg. On November 20th, convulsions occurred, and, after about twenty fits, the patient died comatose on the 21st.

After death.—The cause of the jaundice was ascertained to have been catarrh of the bile-ducts, from irritation of a calculus. Both kidneys were enlarged, and presented the characters of a contracting fatty kidney. Blood dark and fluid. Marked œdema and hypostatic congestion of both lungs. The fluid in the lateral ventricles, and at the base of the brain, was found to contain urea.

CASE VI.—James McW., aged 42, admitted October 15th, 1867, with symptoms of gangrene of the bladder, succeeding those of the passage of a renal calculus. On October 26th, a rigor, followed by fever, with dry, brown tongue, restlessness, and delirium at night, signs of congestion of the base of the lungs, and death, after an attack of convulsions, on November 7th.

After death.—Gangrene of the bladder; great dilatation of the right ureter. Sacculation of the right kidney, with complete destruction of its secreting tissue; numerous small abscesses in left kidney. Congestion of the lungs. A considerable quantity of clear serum, containing urea in the lateral ventricles, and at the base of the brain.

CASE VII.—Henry P., aged 28, admitted September 13th, 1866, with all the ordinary symptoms of rheumatic fever in an intense degree, which had lasted about ten days. There was no cardiac complication. On September 17th, he became suddenly worse; he was heavy and stupid, and could scarcely be roused; the urine was loaded with lithates, but contained no albumen; the tongue was dry and brown; there was now faint pericardial friction. On the following day, he became violently delirious; the temperature was 107° Fahr., and at 5 P.M., he died comatose. An hour before death, the temperature was found by Dr. Murray to be 110.2° Fahr.; indicating an unusually rapid disintegration, or combustion of tissue.

After death.—Blood dark and fluid. A thin layer of recent lymph over surface of heart, but only half an ounce of fluid in pericardium. Both lungs exhibited in a marked degree the characters of hypostatic congestion, as seen after death from typhus. Spleen ten and a half ounces, soft and pulpy, like a "typhus-spleen". Both kidneys enlarged and hyperæmic, weighing together twelve ounces and a half, but surfaces smooth, and capsules separated readily. No fluid in cerebral ventricles.

In this case the products of tissue-metamorphosis, as indicated by the high temperature, were probably in too large quantity for healthy kidneys to eliminate.

CASE VIII.—Esther P., aged 26, admitted November 5th, 1867, with febrile symptoms, a dry, brown tongue, and violent delirium. She had been ill for five days before admission, and presented all the symptoms usually observed in a bad case of typhus fever; but, on examination, no eruption could be discovered on the skin, and though there was scarcely any cough, there were all the physical signs of pleuro-pneumonia from base to apex of the left lung, and also at the right base; the urine contained a considerable quantity of albumen. For four days, the patient seemed to get worse; she had no sleep, was extremely delirious, and was with difficulty kept in bed; but at the end of that time, an improvement commenced both in the symptoms and in the physical signs of the lungs; the albumen entirely disappeared from the urine, and on December 6th, the patient left the hospital well.

In the cases now submitted to your notice, the group of symptoms known as the "typhoid state" was the same, notwithstanding the varying nature of the primary maladies. These symptoms are, a quick soft pulse; a dry brown tongue; the symptoms and physical signs of hypostatic congestion of the lungs; impairment of the mental faculties; stupor passing into coma; delirium, which at one time is acute and noisy, at another low and muttering, and not unfreqnently associated with muscular tremor; involuntary evacuations; and occasionally subsultus, carphology, or even general convulsions. The precise grouping of the symptoms will vary in different cases even of the same disease, while in diseases essentially different it may be identical.

The *post mortem* appearances met with in such cases are a dark fluid condition of the blood; hypostatic congestion and œdema of the lungs; old disease or recent congestion, with epithelial engorgement of the tubes of the kidneys; enlargement and softening of the spleen, and, unless the typhoid state have been of very short duration, an accumulation of serous fluid in the lateral ventricles and at the base of the brain, the veins and sinuses of which are usually full of dark blood.

The typhoid state is developed more commonly in true typhus fever than in any other disease that we are called upon to treat in this country; and accordingly, all diseases in which the typhoid state is apt to be developed are constantly mistaken for typhus, so that the cases registered in official returns as deaths from "typhus," furnish no reliable test of the prevalence of this epidemic fever. The cases sent as typhus to the London Fever Hospital, and in fact, to all Fever Hospitals, from year to year, testify unequivocally to the fact there is scarcely an acute disease to which flesh is heir which may not in the way pointed out be mistaken for typhus. Next to true typhus, enteric, pythogenic, or the so-called "typhoid fever," is the malady in which the typhoid state is oftenest met with; and this is one reason why it was so long, and is still so often, confounded with typhus. In many cases of enteric fever, it is true, the typhoid state does not appear at all, or only late in the disease, and in the latter case the attack is often, though erroneously, described as "gastric fever passing into typhus." Cases of pneumonia are not uncommon where the symptoms of the local disease are completely masked by those of the general blood-poisoning, in the form of maniacal delirium passing into coma; and there are few medical men who have not met with examples of acute rheumatism passing into a condition indistinguishable from the typhoid state of typhus, and presenting similar morbid appearances in the solids and fluids after death. Another class of cases very commonly mistaken for typhus is constituted by certain forms of kidney-disease, and particularly the contracted, granular, or "gouty" kidney. In this form of kidney-disease the patient may at no period of his life have suffered from dropsy, and the urine may contain only a minute trace of albumen; or, as in Case IV, even none at all. The symptoms ordinarily associated with one's ideas of kidney-disease are absent, and therefore when cerebral symptoms supervene, the primary disease is overlooked, and the case is regarded as one of typhus; or, if the attack be sudden or ushered in with convulsions, as one of apoplexy. The "serous apoplexy" of the pathologists of a past age—cases diagnosed as apoplexy during life, but where no apoplectic clot was found in the brain after death—were probably, for the most part, examples of contracted kidney. As physician to the London Fever Hospital, numerous cases of contracted kidney have come under my notice during the last six years in which the cerebral symptoms—the typhoid state—were indistinguishable from those of typhus, and where, failing any previous history, the only points of distinguishing from typhus have been the absence of any eruption on the skin, and the fact that the temperature has been but little elevated above the healthy standard, except when some local inflammation has complicated the primary disease in the kidney.

I shall now endeavour to explain to you why it is that a similar state of the system is so often produced in the course of diseases so widely different, and to show, in fact, that the pathology of the "typhoid state" in all diseases is probably identical, whatever be the cause of the primary disease.

To take, first, the cases of contracted kidney, it is now generally admitted that the cerebral symptoms are due to a poisoned condition of the blood. There may be differences of opinion as to whether the actual poison is urea or some compound of ammonia; but for our present purpose, it is immaterial to inquire which of these views is the more probable. It is sufficient to know that the blood is poisoned with the products of the retrograde metamorphosis of the nitrogenous tissues, which it is the function of the kidneys to eliminate from the body. The destruction of tissue is not increased beyond the standard of health, but the disease of the kidneys renders them incapable of separating from the blood the normal quantity of the products of that metamorphosis.

Take next, the class of febrile diseases. It is well known that convulsions—a symptom characteristic of an extreme degree of the typhoid state—is not uncommon in scarlatina, and it has long been acknowledged that this symptom is independent of any cerebral lesion, but is due to poisoning of the blood in consequence of inflammation of the kidneys. Convulsions, however, are met with in many other febrile diseases besides scarlatina. I have known them occur in typhus fever a hundred times or more, and I have published evidence to prove that their pathology in typhus is the same as in scarlet fever. The urine contains always more or less albumen; the kidneys after death exhibit traces of disease, old or recent; and in not a few instances (as in CASE I) I have discovered urea in the fluid of the cerebral ventricles. But convulsions, under such circumstances, are only an extreme symptom of the typhoid state. In a large proportion of cases of typhus, even where no convulsions occur, albumen is present in the urine during the second week of the disease where cerebral symptoms are most apt to appear; and in several cases where death has been preceded for some time by the typhoid state, but not by convulsions, I have found (as in CASE II) urea in the cerebral fluid.

And if this be the pathology of the typhoid state in scarlet fever and in typhus, it may fairly be inferred that the typhoid state in all febrile diseases, whether they be due, in the first instance, to a specific poison or to a local inflammation, admits of a similar explanation. As in the contracted kidney, the blood is contaminated with the products of tissue metamorphosis, but the cause of the contamination is different. It is now well known that in all febrile disease the disintegration or combustion of the tissue is greatly increased beyond the healthy standard. To this is to be ascribed the increased temperature, which is the pathognomonic symptom of the febrile state, and the rapid emaciation. The

products of this increased metamorphosis are mainly eliminated by the kidneys, and appear in the urine. As long as the kidneys are equal to the increased work thrown upon them, the blood is properly depurated, and the typhoid state is warded off. But if the kidneys be unequal to the task, either from the large amount of effete material to be eliminated, from previous disease in the secreting tissue, or from congestion resulting (as it often does) from their increased work, then the blood becomes contaminated, and convulsions or the typhoid state supervene. This explains why albumen so commonly appears in the urine in the course of all acute diseases of a severe form, and why it is so justly regarded as an unfavourable symptom. It also explains why albuminuria is looked upon as a contraindication to any serious surgical operation. The surgical fever which follows an operation is attended by an increased metamorphosis of tissue; and if the kidneys be diseased, the products of the metamorphosis will be retained in the blood, and induce the typhoid state with all its dangers.

If the views now submitted to you as to the pathology of the typhoid state be correct, they furnish material for reflection as to its appropriate treatment. This ought to be directed towards depuration of the blood of the products of tissue-metamorphosis, avoiding, at the same time, all measures which will irritate the kidneys, and interfere still more with their secreting functions. At the present day, existence of the typhoid state is usually regarded as in itself an indication for liberal administration of alcoholic stimulants. Considerable experience of the typhoid state in all diseases has induced me to doubt the wisdom of the practice, and from an experimental inquiry at the London Fever Hospital, in which the patients admitted with typhus fever on alternate days have been treated with and without alcoholic stimulants, I am satisfied that the benefits of late years ascribed to alcohol in the typhoid state have, to say the least, been greatly over-rated. Before drawing conclusions as to the good effects of alcohol in typhus, it is necessary to watch the course of the disease where no alcohol has been given, and this precaution has too often been overlooked. At the same time, you must not go away with the impression that stimulants, in moderation, are injurious in all cases where the typhoid state is present. Not unfrequently this condition is accompanied by great impairment of the heart's action, indicated by extreme weakness or irregularity of the radial pulse, a diminution of the cardiac impulse, and weakness with shortening of the first sound. Under such circumstances, stimulants in moderate quantity are unquestionably of use. It is the indiscriminate administration of large quantities of brandy in all cases of the typhoid state that I am induced to condemn, both from theory and from practical experience. Such a practice, it appears to me, is calculated to prove injurious by irritating the kidneys, and thus impeding the elimination of the products of tissue-metamorphosis with which the blood is contaminated. The administration of stimulants must be regulated by the condition of the pulse and heart, and not by the mere existence of the typhoid state.

OBSTETRIC MEMORANDA.

[UNDER this head, we shall, from time to time, as materials come to hand from correspondents, publish records of cases remarkable in themselves, or illustrating points of interest in obstetric practice, therapeutic or manipulative. We shall probably in this way preserve from oblivion the notes of very many useful and instructive occurrences in private practice; for the great obstetric experience is that—for the most part hitherto unwritten—of the great body of general practitioners throughout Great Britain. We will only ask those who may forward cases for record, to relate them with the utmost brevity, and equally to condense any appended remarks.]

CASE OF EXTRAUTERINE PREGNANCY.

By HENRY EWEN, Esq., Long Sutton.

MRS. P., aged 39, a farmer's wife, and a pale and delicate person, had been married eight years, and pregnant seven times. She lives six miles distant from Long Sutton, Lincolnshire. She sent for me on February 6th, 1865, complaining of pain on the right side of the abdomen, where there was some fulness, and severe neuralgia along the course of the anterior crural nerve of the same side. She supposed she might be pregnant for the fifth time. I saw her from time to time up to the early part of May. As the progressive enlargement of the abdomen was not central, but on the right side, I suspected it might turn out to be a case of extrauterine pregnancy. The development of the ovum went on up to, or nearly to, the full period of gestation, but no semblance of labour-pains occurred. In the autumn of the same year, she again became pregnant, and was delivered of a living child long before I reached the house on the 22nd of June, 1866. As she was comfortable when I arrived, she was unwilling that I should make any special examination. She made a good recovery. In the early part of the present year, she again became pregnant. On October 29th, 1867, she sent for me, having had accidental hæmorrhage to a not serious extent. On the 30th, I was sent for about midnight, as labour had commenced. She expected to have kept up another month. The presentation natural; labour was progressing slowly. On the 31st, about 2.30 A.M., she was delivered of a small female infant, likely to live. About a quarter of an hour after the birth of the child, hæmorrhage to an alarming extent took place. I immediately introduced my left hand into the uterus, and removed the placenta and coagula, making pressure at the same time by my right hand externally, and thus secured a fair contraction of the uterus. Brandy was given from time to time. She became very faint, with pallid lips and face; vomited several times; but there was no jactitation. The pulse never exceeded 100 in the minute. I remained in the house four or five hours before I thought it prudent to leave her. In the evening she was comfortable. The pulse was under 90. Whilst my left hand was within the uterus, with my right hand applied externally over the abdomen, I could most distinctly feel the extrauterine fœtus of the fifth pregnancy, occupying the right hypochondriac, umbilical, and iliac regions. The axis of the fœtal body corresponded with the axis of the maternal trunk. My attendance was not required after November 5th, when she was going on favourably.

REPORT OF THE OUT-PATIENT MIDWIFERY PRACTICE OF ST. BARTHOLOMEW'S HOSPITAL, DURING THE YEAR 1866.

By W. G. KEMP, M.R.C.S., Resident Obstetric Assistant.

DURING the year 1866, there were attended from St. Bartholomew's Hospital 1025 cases of midwifery, amongst the out-patients. Of these there were—vertex presentations, requiring no interference, 981 cases; twins, 9 cases; vertex and funis, 4 cases; breech, 7 cases; footling, 5 cases; arm, 2 cases; face, 2 cases; requiring delivery by forceps, 9 cases; foot, hand, and funis, 1 case; foot and hand, 1 case; placenta prævia, 1 case; complicated with ovarian tumour, 1 case.

The deaths of mothers were 3, or 1 in 341⅔ cases. Two of these were from puerperal causes, and one from causes apart from the puerperal state. The deaths of children were, at full time, 16, or 1 in 64; premature, 4. One was born dead at the full time; the fœtal heart could not be detected during labour. Of the maternal deaths, depending upon puerperal causes, one was from pelvic cellulitis coming on after miscarriage of twins, at the third month of gestation, and one was from accidental hæmorrhage, caused by separation of the placenta, due probably to fatty degeneration, as there was no history or evidence of the patient having had a fall. The death was due in the third case to phthisis, which proved fatal on the sixth day after delivery.

Twin cases occurred 9 times out of the 1025 cases, or once in 114 cases. In 7 of the 9 cases, both children were born alive; in one case both were dead, being born prematurely at the third month; and in one case, the first was alive and the second dead. Of the presentations, in four cases, both children presented with the vertex; in two cases, with the feet and vertex; in one, with the feet and breech; in one, with the breech and elbow; and in one, both were premature.

Head- and funis-presentations occurred four times, or once in 256 cases. In two cases, the child was born alive; in two cases, the forceps was used, as the cord could not be replaced within the cavity of the uterus; and in one of these cases, the child was born alive; one case, the funis was replaced within the cavity of the uterus, and the child was born alive. In the fourth and last case, no interference was deemed requisite, as the child was thought to be dead, the fœtal heart not having been heard during labour, and the cord being cold and flaccid.

Breech-presentations occurred seven times, or once in 147 nearly; in five out of the seven cases, the child was born alive; in one case, it was born dead; and in the last it was premature, delivery occurring at the sixth month.

Foot-presentations occurred five times, or once in 205 cases. In four of the cases, the child was born alive, and in the other case it was premature, seven months and a half, and had been dead for some time, as it was partly in a state of decomposition.

Arm-presentations occurred twice, or once in 512½ cases. Turning was performed in both cases; in one under the influence of chloroform; and in both cases the mother and child were saved. Chloroform was administered in the first case on account of the violence of the pains; but in the second it was not thought necessary, as the parts were well