sufficient time to the action of one of the forces of the moment, so that, for example, it is necessary that the tissues should be replaced with great rapidity, the evolution of the young growing tissue stops because the driving power has failed, and growth, freed from its control, asserts itself with all its energy. It is not the tumours that are inherited, but the strength of the driving force that directs development and controls growth. This, in the case of some particular tissue, may fail in several members of the same family for generation after generation, just as may the transmission of some special feature.

THE SIMULTANEOUS GROWTH OF TUMOURS.

This, too, explains the well-known liability of homologous tissues to become almost at the same time the seat of similar tumour-growths. Such an occurrence is most common in connexion with non-malignant tumours, such as fibromata and lipomata, but it is not confined to these. I have known, for instance, duct-carcinoma develop at the base of the nipple in both mammary glands within three months. The strength of the power which has guided the differentiation of the tissues through immeasurable time and which controls their inborn capacity for limitless growth is naturally the same in tissues of similar descent. It maintains its control, or it fails before the action of the forces of the moment in the same way in them all.

As it is the work of the tissues, their own and that of past ages, that step by step has made them what they are, it has been suggested that it is something connected with their work that cuts their evolution short, and allows their power of growth to assert itself unchecked, and that the origin of tumour buds is to be found in a functional disorder of some kind. But while it is quite true the tissues that compose tumours never do any work, the failure to work is the consequence, and not the cause, of the arrest of evolution. Tumour tissues are imperfect, and remain imperfect, and work cannot be expected of such. Their differentiation has been arrested before they were capable of work. Tumour tissues never work because, with the rarest possible exceptions, they never reach the perfection of maturity. The exception proves the rule; a few mammary adenomata attain such a high degree of perfection that secretion has been known to take place in certain portions of them. The vast majority falls far short of this.

THE ARREST OF DEVELOPMENT OF ORGANS.

If it is asked what could interfere with the power of hereditary transmission to such an extent as to cause the arrest of the differentiation of the tissues in one part of the body and the formation of one kind of tumour, or the arrest of their involution in another and the formation of another kind, I can only point to the analogy of the arrest of development of organs and say that I can see no reason why such a power may not fail from mere exhaustion, as, for instance, in old age, either of organs or of individuals, when it is notorious that the liability to tumour-growth becomes greater and greater, or from excessive action, as when an enormous number of young cells is produced with great rapidity in response to some persistent chronic irritation. Whether the action, or want of action, of any organ in the body could have a similar influence over the power of hereditary transmission and bring its work to a sudden stop it is impossible to say. There is one structure in the body at least that appears to be capable of abrogating the laws that control the growth of tissues and of enabling them to increase in size without limit; but we know too little of these things as yet. The study of physiology is of yesterday. At our best we have only a very general acquaintance with some of the most conspicuous individual actions of what may be called the gross mechanical apparatus of the body. We know next to nothing of the essential organs of life, those that really matter where growth and development are concerned. We are only just beginning to grasp the fact that many absolutely insignificant-looking structures, some of them almost microscopic in size, possess the most extraordinary power over growth and even over life itself; and we are only just beginning to realise that no organ or tissue in the body exists for itself alone, but only in relation with every other organ and tissue. It may be that when we know more about these things some structure will be found which has some control over hereditary transmission and the development of tissues as distinguished from their growth. Until then, and it must be admitted the

prospect is not a good one, all our hopes of finding such a thing as a cure for the growth of tumours (for amputation, though it may free the patient from the consequences, can in no sense be regarded as a cure) must rest either upon finding some remedy which, like Coley's fluid, can directly kill the tumour cells without endangering too greatly the vitality of the normal tissues; or upon stimulating to increased energy the normal tissues that surround the tumour, until they are able to treat those degenerate cells that have failed to attain their full development, and have continued to grow at the expense of the organism without being of any service to it, in the same way that the workers treat the drones.

SOME CASES OF PYREXIA.¹

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THE object of my paper is partly to make use of the plentiful clinical material which comes in my way as pathologist to the Royal Devon and Exeter Hospital, and partly to show, what no doubt is known, that it is only by a truly judicial consideration of the evidence obtained by the methods of pathology and bacteriology, as well as by those of clinical medicine and surgery, that we can hope to arrive at a satisfactory diagnosis. If a biased view is taken of these cases, whether from the pathological or clinical side, mistakes are pretty certain to arise.

I am going to report some cases illustrating many different conditions causing pyrexia. Pyrexia due to enteric and paratyphoid fever, to the pneumococcus and to syphilis; pyæmic and septicæmic cases, including malignant endocarditis; pyrexia due to subdiaphragmatic abscess, portal pyæmia, and to nematoid worms; urinary cases and rat-bite fever.

I will begin by making a few remarks as to the Widal reaction. It is rarely found before the fifth day—generally about the eighth. In very severe cases it may not appear at all, and occasionally even in ordinary cases not till late. If the patient has had enteric fever previously, or has been inoculated with typhoid vaccine, or is a typhoid carrier, a positive result may be obtained, but under these circumstances the proper thing to do is to repeat the test a few days later, and note whether agglutination occurs in higher dilutions than when first done, so that for this purpose it is necessary on each occasion to find the end limit of the agglutination of the serum.

There is also paratyphoid fever to be considered. Professor F. A. Bainbridge in the Milroy lectures thoroughly reviewed the relations of paratyphoid fever A and B to meat poisoning, Paratyphoid fever A is due to bacillus paratyphosus A, an organism of the Salmonella group, and closely allied to bacillus paratyphosus B, to Gaertner bacillus (the cause of food poisoning), to B. suispestifer (found in swine fever), and to the Danysz bacillus so extensively used for destroying rats and mice. These organisms are so closely allied that they cannot well be separated culturally, but this can easily be accomplished by agglutination tests and by the test of complement deviation. The diseases are quite distinct, the food poisoning cases having an acute course with gastro-intestinal irritation, and with an incubation of a few hours, and the paratyphoid fever A and B having an incubation period of 8 to 15 days, and being very like mild attacks of enteric fever.

Paratyphoid fever A is found chiefly in India. Perforation rarely occurs, and the mortality is only 2 per cent., that of enteric fever being about 13 per cent. Sometimes besides the rose spots a purpuric eruption is seen. Headache and a rapid pulse are prominent features, the spleen is enlarged, but Peyer's patches are not obviously involved. Paratyphoid fever B is practically the same, only purpuric eruptions do not occur. Inflammation of gall-bladder and periosteal abscesses have been met with. Severe headache is a frequent and prominent symptom. Pyrexia lasts from 4 to 30 days, average 17; as a rule it is indistinguishable

¹ A paper read before the Devon and Exeter Medico-Chirurgical Society.

from enteric fever. Occasionally the onset is more acute, and ushered in by rigors and vomiting. Labial herpes occurs, and the spleen is supposed to be firmer than in enteric fever. Post mortem there is no inflammation or ulceration of Peyer's patches as a rule. The large intestine is intensely inflamed and shows small ulcers. Mortality 3 per cent. This form is found in Great Britain and on the continent.

Both paratyphoid bacilli are confined to the intestines of human beings, who either have paratyphoid fever or are acute or chronic carriers, either urinary or intestinal. Women are more frequently carriers than men owing to their tendency to intestinal stasis and gall-stones. The agglutination reactions require careful consideration. Group reactions may occur—namely, the serum of a patient suffering from a disease due to a particular organism may agglutinate an allied organism. Cases have occurred where a patient's serum has agglutinated bacillus paratyphosus B in higher dilutions than bacillus typhosus, and yet the latter only has been cultivated from the blood. Hence it is necessary in such cases both to find the end point of the agglutination, which may be as high as 1 in 20,000, and also to carry out absorption tests. By adding to the serum living bacilli of, say, bacillus typhosus and centrifuging all agglutinins of this organism are removed, and if the case is one of enteric fever no agglutinin remains. The co-agglutinins only would be removed in such a case by bacillus paratyphosus B or A. The reverse proceeding to such agglutination tests may be carried out, using known sera and the unknown organism obtained from the patient.

Blood cultures are almost invariably successful in the first week. It is necessary to draw off 5 to 10 c.c. blood, and it is best to inoculate a flask containing 100 c.c. broth with 1 per cent. citrate of soda in it to prevent the blood from clotting. At the height of the fever or later, or in the case of a carrier, the organism may be isolated from the stools by such means as the Conradi-Drigalski plates, but then full cultural tests must be applied. A freshly isolated organism may not be agglutinated by the patient's serum, though it may be by a stock antiserum to the same organism.

A man, aged from 30 to 40, eight years ago had enteric fever in Australia; since then he has periodically had attacks of pyrexia, lasting for from 14 days to three weeks. There have also been at the same time pain in the region of the gall-bladder with tenderness, but no definite enlargement. He has been treated by a paratyphoid vaccine without any apparent benefit. Whether this vaccine was made from his own organism or was a stock one I cannot say. Agglutination test: There was complete agglutination to bacillus typhosus in dilutions of 1 in 10 and 1 in 50, partial in 1 in 100, while bacillus paratyphosus A gave complete agglutination in 1 in 10, 1 in 50, 1 in 100, 1 in 200, and 1 in 400. Partial in 1 in 600. Blood culture was negative.

Since I saw him he has been operated on by Mr. Mayo Robson and the gall-bladder drained; he has been quite well since. I believe culture from the gall-bladder proved negative, but pancreatitis was present. The question whether this patient was a paratyphoid carrier must remain unsettled.

The presence of a leucopenia is a valuable confirmatory test of enteric fever. Mr. G. L. Thornton, of Exmouth, I believe has great faith in the diazo reaction. The following are two anomalous cases of enteric fever:—

1. A woman was admitted, under the care of Dr. H. Davy, on Sept. 28th, 1906, and discharged on Dec. 27th. She was admitted for muscular rheumatism. The attack came on suddenly. No joints were affected. Vomiting and indigestion were complained of. Pyrexia was present, 104° to 102° F. Pulse 120; respiration 32. Abdominal pain and tenderness and diarrhoea ensued and the urine contained traces of albumin. Blood count—white cells 6800. Widal positive. The spleen became palpable and spots were observed. The temperature was irregular and not characteristic.

2. A woman was admitted, under the care of Dr. Davy, on Oct. 28th, 1906. She died on the 29th. There was a history of cough for some weeks, with difficulty of breathing. The urine contained albumin and uræmia was diagnosed outside. A severe rigor occurred and dyspnoea was marked. Diarrhoea, epistaxis, and hæmoptysis ensued, and spots were found. The urine contained albumin and casts. Widal positive.

Necropsy.—There was ulceration of the tongue, the heart was flabby, and the spleen was enlarged. Marked nephritis was present and typical typhoid ulcers. A bacillus was isolated from the spleen, having the cultural and agglutinating characters of bacillus typhosus. Here was a case where the toxin of bacillus typhosus acted chiefly on the kidneys, causing "nephrotyphus."

Infection due to Bacillus Coli.

From cases of enteric fever it is a short step to cases of infection due to the bacillus coli communis. Many cases of pyrexia often resembling enteric fever, or possibly tuberculosis, general or urinary, are due to urinary infection by this organism. They generally start acutely, and either

undergo a cure or become chronic. There is often a history of a chill, and in most cases there is not only very marked bacilluria, but also definite pyuria. There is often pain and aching of one or both kidneys, with dysuria and frequent micturition.

Evidently a pyelocystitis occurs, and sometimes a nephritis as well, with occasionally the production of multiple abscesses. In some cases there is a bacilluria with very little pus present. There may be as well as kidney symptoms definite abdominal trouble with pain and meteorism. The bacillus coli is able to produce a massive peritonitis, having very much the same features as that caused by the tubercle bacillus or actinomyces. In one of these cases there was, besides the bladder symptoms, a parametritis or pelvic peritonitis. They undergo a natural cure, or may require vaccine treatment, which, though it may not in every case absolutely eliminate the bacillus from the urine, causes, I believe, nearly always, very great benefit.

A woman was admitted, under the care of Mr. A. C. Roper, on Oct. 13th, 1911, and died on Dec. 25th. There was a history of pain in the left hypochondrium for four weeks with vomiting after food. There was a well-defined, very tender mass in the left hypochondriac region. Leucocytosis 23,000. An exploratory incision was made and multiple renal abscesses found. Bacillus coli communis was isolated from pus. Marked pyrexia was present.

A woman, aged 28, was admitted, under the care of Dr. Davy, on Feb. 15th, and was discharged on Oct. 11th last. There was a definite remittent temperature at first, but latterly this ceased. History: Well up to Christmas. Illness started with a cold. She shivered and suffered from diarrhoea and vomiting, with headache and pain on micturition. Definite parametritis was present. Uterus fixed in anteverted position. Pus was found in urine, and bacillus coli communis isolated. Treated by autogenous vaccine, and when last seen a fortnight ago was quite well, with no pus or organisms in the urine.

A girl, aged 3, was admitted under the care of Dr. W. Gordon. She was in hospital one month, and was discharged cured. It was noticed that the child passed urine very frequently, and on admission she looked very ill and feverish. Temperature 102° F., pulse 140, and respirations 32. Von Pirquet negative. Urine contained no tubercle bacilli. Bacillus coli communis obtained culturally from the urine, which contained many pus cells. Under treatment by urotropine the child made a complete recovery.

Children are not immune to this condition. Female children are supposed to be more liable to infection, as there is more possibility of direct infection upwards per urethram.

A woman was admitted, under the care of Dr. Gordon, on Jan. 25th, 1912. She is still under treatment. Eight months before admission she had a chill after sitting out on a damp evening. Symptoms: Frequency of micturition and pain. Liver enlarged, spleen palpable, left kidney very moveable. Temperature elevated and tongue coated, and there was definite loss of flesh. Bacillus coli communis isolated from urine. Autogenous (unheated) vaccine was given with very great improvement.

Here there was a definitely moveable kidney, and no doubt a twist of the ureter in such cases may cause, besides pain, attacks of pyrexia. Another patient under my care, whose attack began with a chill, and who at first was extremely ill with pyrexia, resembling enteric fever, also had a moveable kidney and obtained marked benefit from the wearing of a belt.

I will now relate a urinary case under my care where the symptoms at first were so severe that pyæmia, septicæmia, or malignant endocarditis was suspected, but in the end a perineal abscess developed, and when this was opened a rapid cure ensued. Here we were inclined to be too pathological, if that is possible.

The patient was a middle-aged man. Illness began a week before I saw him, with some pain on micturition. About a month before this he was exposed to a very bad smell when undertaking the burial of a woman who had died from cancer of the womb. Symptoms: Marked irregular pyrexia, and recurring rigors lasting about 20 minutes, and generally followed by vomiting. No trouble in lungs or heart. Widal negative. Blood count 13,000 whites, with 93 per cent. polymorphs. Blood culture negative. Spleen slightly enlarged. Treated by antistreptococcal serum and streptococcal vaccine, without any benefit. A week later he complained of pain on micturition, and a perineal abscess developed, and was opened. The temperature fell and the patient soon recovered.

I believe Mr. Russell Coombe has since had a somewhat similar case, where the urinary trouble was an extravasation and where very marked pyrexia was present. My only connexion with this case was that I was asked to examine some urethral pus for organisms.

Pyæmic and Septicæmic Cases.

A man, aged 38, was admitted, under the care of Dr. Davy, on March 6th and died on March 10th, 1912. I also saw him outside with Dr. J. D. Harmer. He was well up to the week before admission, when he began to complain of pain all over the body, but especially over both lower limbs. The left wrist was swollen. A Widal was done three times with negative results. The abdomen was much distended owing to paralysis of the bladder, which was frequently very full. There were

marked pyrexia and delirium; the tongue was dry; everything was passed into the bed, and there were general twitchings of the body and slight rigors. Membranous tonsillitis occurred. White cells 28,000, with 89 per cent. polymorphs. Urine contained many red blood cells, some leucocytes and staphylococci. On blood culture staphylococcus aureus was obtained. An abscess developed in the gluteal region. No post-mortem examination was allowed.

Here first enteric fever was suspected, but the leucocytosis and negative Widal put this out of court, and blood culture and presence of staphylococci in the urine clinched the diagnosis. Whether the primary cause of the pyæmia was a tonsillitis or some deep-seated osteomyelitis cannot be known.

A man, aged 56, was admitted, under the care of Mr. Roper, on June 25th and died on August 14th, 1908. Seven weeks before admission he noticed pain in the calf of the left leg after hard work. There were cicatrices in the leg due to osteomyelitis with removal of bone 40 years previously. An abscess developed in the calf, which was opened. The temperature was raised and irregular in type, 103° to 105° F. He became delirious and vomited, and jaundice appeared with abdominal distension. On blood culture staphylococcus aureus was obtained.

Necropsy.—Liver was much enlarged and congested. Small foci of pus in portal spaces all over the liver.

A man was admitted, under the care of Dr. Davy, on March 29th and died on April 10th, 1910. At the end of January last he noticed a pain in the right side in the region of the lower ribs; he took to his bed and remained there three weeks, and then returned to work, but in another three weeks he had again to go to bed owing to pain and difficulty of breathing. Diarrhoea was present. He looked very ill and thin on admission, and his pulse was 104 and temperature 102.8° F. The liver was displaced downwards and the spleen was enlarged. There was partial dulness at both bases with fine crepitations and bronchial breathing. Rigidity in the epigastrium. On April 6th a rigor occurred (temperature went up to 106°), followed by profuse sweating. Later very definite dulness on the right side posteriorly suggested pus, and on resection of a rib much foul-smelling matter was evacuated.

Necropsy.—The liver was riddled with abscesses of various sizes. The drainage-tube was in one of them. The appendix was gangrenous.

Though there never had been anything pointing to appendicitis, this was the origin of the trouble, resulting in suppurative pylephlebitis and death.

Septicæmia is not always fatal, as is shown by the case of a girl, under the care of Dr. Gordon in 1906, who had irregular temperature, with rigors, lasting on and off for over two months, and who developed in succession thrombosis of veins of the thigh, legs, and arms. She became very anæmic and suffered from palpitation and severe pains in the chest and around the spleen. In due course she made a complete recovery. Treatment was antistreptococcic serum and drop doses of cyllin.

A woman was admitted, under the care of Mr. J. D. Harris, on Oct. 6th and died on Oct. 15th, 1905. On the day before admission she had a slight accident, tumbling up stairs. This was followed by a pain in the left side. She did not sleep that night on account of pain. Pyrexia was present. Widal negative. The abdomen became markedly swollen and tympanitic and tender. Diarrhoea began on the second day after admission and was of pea-soup character. Vomiting was severe. Leucocyte count on Oct. 12th 40,500, 87 per cent. polymorphs. On Oct. 13th it was again 40,000. There was a history of long-standing indigestion with occasional attacks of abdominal pain. Dulness was present at both bases, with a friction sound on the right side. Impulse of the heart was displaced inwards and upwards. A needle introduced drew off foul-smelling pus, a rib was excised and a pint of pus evacuated. Death occurred next day.

Necropsy.—Hour-glass stomach, with a perforated ulcer communicating with a sub-diaphragmatic abscess. Pus also was present in the hollow of the pelvis around the uterus and appendages.

I have treated by vaccines in all 4 cases of malignant endocarditis, obtaining by blood culture a streptococcus—in every case, I regret to say, unsuccessfully. So far I only know of two cases of this disease which have been cured by vaccine treatment, and in neither of these could the fact of endocarditis be definitely proved. One was the case under Sir James Barr, where Captain S. R. Douglas set up a laboratory upon the premises and did the opsonic index twice daily. Here the organism was a streptococcus. The other was a case under Dr. J. Michell Clarke, where the influenza bacillus was obtained by culture. In both of these cases, instead of an actual endocarditis being present, there was the possibility of the bruit being due to relative mitral insufficiency.

The cardinal signs of malignant endocarditis are: signs of valvular lesion with changes in character of bruits, presence of organisms in the blood, occurrence of embolism in various organs, with symptoms of intoxication—namely, pyrexia, headache, malaise, and rigors.

A girl aged 19. There was a history of having had influenza over two months before I saw her. She had been in bed ever since, with elevated temperature, malaise, and loss of appetite. She had coloboma iridis and congenital heart disease, probable congenital stenosis of the pulmonary orifice, with perforation of the ventricular or auricular septum, as there was a harsh rasping murmur over the mid-sternum, also heard all over the præcordium. She suffered from pyrexia, from hæmaturia, and albuminuria, dry pleurisy with marked dyspnoea and pain, and was greatly emaciated. Besides being treated by vaccines she

had per rectum antistreptococcic serum, which caused no benefit, but resulted in a severe attack of urticaria and rheumatic pains in joints. A petechial rash appeared and ulceration and perforation of the septum nasi, evidently due to an embolus. Finally, a severe attack of parotitis developed, and she died about six months after the onset of illness. Blood cultures showed a plentiful growth of a streptococcus of the salivarius type.

A girl was admitted, under the care of Dr. Davy, on Feb. 9th and died on March 24th, 1905. There was a history of congenital heart trouble with cyanosis on exertion or exposure to cold. She was ill for three weeks before admission with headache, fever, and loss of flesh. There was a loud, harsh systolic bruit, heard best over the pulmonary orifice. There were marked cyanosis and dyspnoea. White cell count 27,000.

Necropsy.—A small cavity in the left lung. Infarctions in lungs and spleen. The main branch of the pulmonary artery was plugged with ante-mortem clot. The right ventricle was dilated and there was a congenital interventricular opening, some endocarditis of the tricuspid valves, but the valves of the pulmonary artery were replaced by a mass of vegetations, which were composed mainly of micrococci.

Malignant endocarditis is very likely to be developed in cases of congenital heart disease. In some cases this disease lasts for many months, and may show marked remissions. In others the course is extremely rapid. I saw with Dr. Davy a very acute case where there was plugging of the femoral artery, and gangrene ensued. Coma and death soon took place. Here the mitral valves were involved. Pyrexia with a change in the character of bruits in heart disease does not always mean malignant endocarditis, as is shown in the case of a man under the care of Mr. H. Andrew, who developed rapidly a marked aortic bruit with pyrexia. Blood culture proved negative, and he recovered.

Cases of Pneumococcic Peritonitis.

A woman, aged 35, was admitted, under the care of Mr. C. E. W. Bell, on Feb. 11th last and died the next day. She was seen by Dr. G. P. D. Hawker outside. She was taken ill with pain in the right shoulder and chest, with rigors and fever. She got up next day, though no better, and then began to have pain in the abdomen. Next day she took purgatives and loose stools resulted. The following day abdominal distension was noted, but the patient would not come in. On admission her temperature was 101° F., pulse 110, and respirations 28. There was a flushed appearance suggesting pneumonia, and much distension of the abdomen, especially in the lower part. A tympanitic note was found over the abdomen, except in the right flank, where it was dull. There was slight dulness at the right base posteriorly, with a few crepitations. Exploratory laparotomy was performed. The intestines were found to be covered with thick fibrinous lymph. Films and cultures proved the pneumococcus to be the cause.

A man, aged 56, was admitted, under the care of Mr. A. C. Roper, on May 19th, 1910, and died the same day. He was at work till the evening of the day before admission. About 5 p.m. he began to complain of pain across the abdomen. During the night this increased. He says that previously he had been quite well and able to do his work. On admission the temperature was normal in the mouth and 99° in the axilla. Respirations 44. The pulse was quick and weak. There was marked meteorism. The flanks showed shifting dulness. Bronchial breathing at the left base, with moist crepitations.

Necropsy.—The abdomen was full of stinking pus, but there was no perforation. The lower lobe of the right lung showed red hepatization, and films demonstrated capsulated diplococci, evidently pneumococci.

A woman was admitted in a collapsed state, under the care of Dr. Gordon, on March 10th, 1906, and died the same day. She had been ill for three days before admission. The onset was marked by sudden abdominal pain and dyspnoea, with diarrhoea and vomiting. The abdomen was much distended, especially in the region of the appendix.

Necropsy.—Creamy pus was present in the peritoneal cavity, with adherent lymph. There was no perforation of the stomach, duodenum, or intestines. No appendicitis or cholecystitis or perforation of gall-bladder. No pyosalpinx or pancreatitis. Films and cultures from pus showed pneumococci.

Attacks of bronchitis with pyrexia are often caused by the pneumococcus, and here I may mention in parenthesis that marked benefit is obtained by vaccine treatment. I recently made a pneumococcic vaccine for Dr. J. H. Iles for a man with chronic bronchitis and pyrexia, due, I believe, to pneumococcic infection, with, I am told, a good result.

Pyrexia due to Various Causes.

Cases of lymphadenoma occasionally exhibit marked pyrexia, as in a boy under the care of Dr. Gordon, who periodically got attacks of fever lasting a week, and at the same time the glands of his neck became much swollen. A peculiar organism was obtained from his blood, but a vaccine did no apparent good, and it was impossible to definitely connect this bacillus with the condition. Lymphadenoma, like pernicious anæmia, splenic anæmia, and probably leucæmia, especially acute lymphatic leucæmia, are due no doubt to a toxin, and consequently pyrexia occurs. This may also be found in cases of anæmia due to pyorrhoea alveolaris, which should not be forgotten.

Pyrexia of a remittent or intermittent type, which may be mistaken for typhoid fever, tuberculosis, or malaria, occurs in the course of syphilis. I have seen two cases of this. One was that of a man under the care of Mr. Roper, who was admitted with swelling of the knee-joint. Pyrexia occurred

and a Widal proved negative. Wassermann reaction, however, was positive, and with mercury the temperature rapidly fell and the joint trouble cleared up.

The other case was that of a boy, admitted under the care of Dr. Gordon, with congenital syphilis and choroiditis, who always gives a very marked positive Wassermann. He was admitted in a semi-conscious state with cerebral symptoms. These cleared up, and he was discharged, but later was readmitted with a temperature of 102° F. and swollen glands of the neck.

I have had three cases of trichinosis. The first one Mr. J. D. Harris and I published in THE LANCET, and a reprint of which is here for anybody who wants it. This patient had swelling of the eyelids, with tenderness of muscles, headache, and pyrexia. A history was obtained of his having partaken of diseased pork. A woman who also ate this pork was ill. Both these patients had marked eosinophilia, and the trichinae were demonstrated in the pork, and also in a dead rat found on the farm. I have lately examined the blood of another patient, under the care of Dr. C. J. Vlieland, who in all probability had trichinosis. Her blood showed 33 per cent. eosinophiles. Evidently trichinosis is much commoner than is generally supposed.

Pyrexia may be due to round worms as well as to trichinae. Osler calls the condition typholumbricosis. The following case is possibly such a one:—

The patient was admitted, under the care of Mr. Harris, on Nov. 11th, 1911, and was discharged on Jan. 11th, 1912. She was supposed to be suffering from appendical abscess. Laparotomy was performed and the appendix was found to be normal. The patient was in the fourth month of pregnancy. She developed jaundice, high temperature, and rapid pulse and respiration. Leucocytosis 47,000. Widal negative. Examination of lungs negative. On Dec. 11th acute parotitis developed on the left side. This subsided only to be followed by a similar condition of the opposite side, which also subsided. On the 28th another rise of temperature corresponded with the passing of a round worm 10 inches long, the temperature dropped, and the patient was later discharged on account of her pregnancy.

I have seen one case of rat-bite fever where there were periodic attacks of pyrexia, with pains in the limbs of a rheumatic character, and an urticarial eruption.

Toxæmia and uræmia may cause pyrexia, as is shown in the following case:—

A woman, aged 40, was admitted, under the care of Dr. Davy, in 1906. She had been ill for six months and suffered from severe abdominal pain with vomiting. There had been diarrhoea for the last 14 days, with much thirst, giddiness, fainting, and headache. She was passing very little urine, only three to four ounces; the face was puffed and the abdomen swollen; slight jaundice was present. The urine contained albumin and bile. Leucocytosis 22,000. She became very drowsy, with twitching of hands and unequal pupils. Diacetic acid and B-oxybutyric acid were found in the urine. Absolute coma ensued, with a temperature of 103° to 104° F. Mr. Roper transfused her with a quart of 2 per cent. sodium bicarbonate, and she rapidly recovered. Treatment besides transfusion consisted of hypodermics of strychnia and pilocarpine and vapour baths.

In conclusion, I must thank you for your attention to my somewhat lengthy paper, but I trust that the cases I have related to you may have shown how many difficulties there are in arriving at a diagnosis in a case of pyrexia, and may be of some help to you in solving such difficulties in the future. I should also like to thank the physicians and surgeons of the Royal Devon and Exeter Hospital for their kindness in letting me make use of the notes of their cases.

Exeter.

BRIGHTON GUARDIANS AND OUTDOOR MEDICAL SERVICE.—Following on the retirement at Brighton of one of the medical officers under the guardians the board has decided to reduce the number of medical areas in the union from four to three, and raise the salaries of the two officers whose districts are enlarged from £125 to £155 per annum. The medico-political committee of the Brighton division of the British Medical Association wrote to the guardians, stating that while offering no objection to the rearrangement the committee respectfully suggested that the additional salaries are inadequate and should more nearly approximate to the sum hitherto paid for the same work, and pointing out that there had been no epidemics during the last few years, and that the medical work in the town generally had been considerably below the average. The guardians, however, were influenced by the result of the working of the Old Age Pension Act, for whereas on Dec. 31st, 1910, there were 1034 persons in receipt of outdoor relief, the figure on Nov. 12th, 1912, was 546.

THE SUPPRESSION OF THE CONVULSION IN ECLAMPSIA.¹

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FIRST, it must be expressly stated that this note does not attempt to deal with the treatment of the cause of eclampsia. It relates solely to the suppression of what is usually the leading symptom in the eclamptic condition—the convulsion. The treatment of eclampsia cannot be carried out on rational lines so long as the pathology is obscure. Therefore, treatment must be symptomatic, and as the leading and most serious symptom of eclampsia is usually the convulsion, the control of the latter has been the chief aim and object of obstetricians for many years. Seitz² divides cases of eclampsia into three classes: (1) Cases in which the poison acts mainly on liver, kidneys, heart—coma; (2) as in (1), but acts also on nerve centres—convulsions; and (3) acts on nerve centres chiefly—convulsions predominate.

In the majority of cases convulsions occur, and control over them is necessary, because the fates of mother and child depend largely on the severity of the fits, and, when the latter are severe, on their frequency also. As a rule, the foetus always dies when the fits have been severe—killed possibly by a toxin related to eclampsia, more probably asphyxiated during severe convulsion. For the mother the actual number of fits is not of supreme importance unless they are severe. Seitz³ states that the highest mortality occurs in cases in which there have been from 16 to 30 fits (30 to 35 per cent.); the next highest mortality in those in which from 30 to 70 fits have occurred (33 per cent.). Severity is always of the gravest import, especially when the intervals between the attacks are marked by deep persistent coma or a condition of unconsciousness with never-ending restlessness, both indicating that to the eclamptic state there has been added some cerebral condition. And undoubtedly some eclamptics die, not from eclampsia, but from oedema or congestion of the brain, or from forcible rupture of cerebral vessels during the fit. Prutz⁴ gives the following figures as a result of necropsies on eclamptics: Oedema of brain, 42 per cent.; congestion, 30 per cent.; apoplexy, 3 per cent.; unaltered brains, 10 per cent.

Control of the Convulsion.

1. Reliance has been largely placed on chloroform, chloral, morphia, and veratrum viride or veratrine. The most recent upholder of the chloroform, chloral, and morphia treatment is Stroganoff, of St. Petersburg, whose reported results are truly astonishing. No doubt the drugs named have an influence in the treatment, but it should be pointed out that each patient is treated under conditions infinitely more favourable than have ever before been made use of, for she is placed at once in a totally darkened room that is isolated from all possibility of disturbance by external influences. There she remains with doctor and nurse—she is not disturbed in any way until light chloroform anaesthesia has been induced, even for hypodermic injections, catheterisation, administration of enemata, &c.—in a word, each one of her senses is guarded from every stimulus that might precipitate a convulsion in an eclamptic. Further, her nervous system is blunted by the administration of morphia and chloral, whilst chloroform is ever in reserve should a fit threaten.

Treatment begins immediately on admission, every eclamptic brought to the hospital receiving at once a hypodermic injection of morphia, gr. $\frac{1}{4}$. She is then anaesthetised and operative delivery is carried out if considered advisable. If the necessity for this does not exist the patient is placed in the isolated dark room and narcosis is maintained with morphia and chloral until well after delivery. Baths and hot packs are not given, any necessary washing being done with warm moist cloths. Stroganoff states that the careful execution of treatment on these

¹ A paper read before the Liverpool Medical Institution on Nov. 21st, 1912.

² Archiv für Gynäkologie, Band xxxix., p. 425.

³ Loc. cit.

⁴ Zeitschrift für Geburtshilfe und Gynäkologie 1892, Band xviii., pp. 1–52.