half its present price it would still be more expensive to give it orally. But to my mind much more serious than expense is the uncertainty of the oral method of administration. The very fact that a single positive result was obtained shows the possible danger of the method. We have no control over the pyloric sphincter. Also it is conceivable that there might be danger for those with achlorhydria (and it is to be remembered that 4 per cent. of healthy individuals are estimated to have achlorhydria) in that pepsin would not be activated, and in that an oral dose, if calculated from a series of experiments on other patients as being a certain multiple of the subcutaneous dose, might be an overdose for the achlorhydric. Moreover, if the insulin were given with food it is possible that variations in the articles of diet (in spite of keeping the intake of carbohydrate, protein, and fat constant) might influence the proportion of insulin absorbed or destroyed by pepsin.

The experiments are admittedly few in number and confined to one patient. This patient is an expert at diet calculations, however, and has submitted to a large number of blood sugar curves for previous experimental work. She has for several weeks given herself 35 units of insulin daily, and her fasting blood sugar, tested frequently, has remained steady within narrow limits. She, of course, stayed in the laboratory for these special experiments, eating her food off the laboratory bench. The results are recorded in the hope that other workers who have done similar experiments will publish their findings, and so prevent the raising of false hopes in the minds of those suffering from diabetes mellitus.

REFERENCE.

¹ *Journal of Physiology*, 1923, vol. 58, p. 18.

A CASE OF MALARIA ARISING IN THIS COUNTRY.

BY

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ALTHOUGH nearly five hundred cases of locally contracted malaria have been discovered in England since 1917, the vast majority of them can be traced to infection from cases returned from abroad. Indeed, it is probable that malaria is no longer endemic anywhere in England save in the isles of Sheppey and Grain on the south bank of the Thames estuary. The chief interest of the following case is that it suggests the possibility of another endemic focus in the Essex marshes on the opposite bank.

K., a girl aged 12, was admitted to the London Hospital on September 10th, 1923, with the following history:

A fortnight previously she had returned to her home in the East End from a Girl Guides Camp at Pitsea, Essex. Ten days later (that is, five days before admission) she had had a "shivering attack" followed by headache and "stiff neck"; the attack was repeated two days later. She felt quite well and said there was nothing wrong with her. Her bowels had been constipated since the onset and she had vomited once after the first attack. She had had no previous illness.

Her father, while at Gaza in September, 1917, was ill for three days with "sandfly fever"; he was cured by pills and did not leave the battery lines. In September, 1918, he had a similar attack in France; again he was cured by opium and quinine pills without going into hospital. He has had no attacks since and has been in perfect health. The mother is healthy and has had no shivering attacks; one other child, aged 9 years, is healthy.

The patient was a pale, intelligent child in no pain or distress. Temperature 105°, pulse 140, respirations 30. The tongue was furred; the heart and lungs were clear; spleen enlarged 2½ finger-breadths below the costal margin, uniform and rather hard.

First Day.—Rigor at 4 p.m.—four stages: (1) coldness of the feet spreading to the body; (2) trembling of shoulders and legs; (3) short sleep; (4) awoke feeling hot and sweating.

Second Day.—Rigor 5.30 p.m.

Third Day.—Rigor 2 p.m.; parasites (benign tertian) found in the blood.

Treatment by quinine was started. There were no more rigors, and the temperature remained normal. On discharge ten days later the spleen was not palpable.

The length of time (five years) since the father's last attack—if, indeed, he ever had malaria—and the absence of the anopheles mosquito from the East End district in which the patient lived eliminate the father as the source of infection. No history can be obtained of any lodger,

relative, or neighbour ever having suffered from malaria, and everything points to the child's infection having been acquired while in camp. The children slept there in three barns situated in a lonely spot in the marshes south of Pitsea. The nearest habitation, the farm to which the barns belonged, was about half a mile away and was itself some three miles from Pitsea village. Inquiries revealed no recent cases of malaria and no returned soldiers were living in the neighbourhood. The farmer stated that in his youth "everyone" on the marsh had "ague," and he himself had had two attacks, one as a boy and one as a young man, some thirty years ago. He gave a vivid description of a typical attack of malaria. On examination his blood showed no parasites.

As late as October 30th mosquitos were plentiful in the neighbourhood and three were caught inside the barns where the children slept; they proved to be *Culex pipiens*. Pitsea, however, is given as a locality for *Anopheles maculipennis* by the authorities of the British Museum (Natural History).

I am indebted to Dr. Robert Hutchison for permission to publish this case.

LITERATURE.

Malaria at Home and Abroad. By S. P. James, M.D., D.P.H., Lieutenant-Colonel I.M.S., 1920.

Reports to the Local Government Board, New Series, Nos. 119 and 123.

Annual Reports of the Chief Medical Officer of the Ministry of Health, 1922 and 1923.

Map showing distribution of *Anopheles maculipennis*. British Museum (Nat. Hist.), 1918.

SPONTANEOUS RUPTURE OF THE LOWER UTERINE SEGMENT.

BY

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SPONTANEOUS rupture of the lower uterine segment must be a very rare occurrence, and perhaps on this account the notes of the following case may be of interest.

I was asked to see a patient, aged 30 years, at 5.45 p.m., on account of abdominal pains which had commenced an hour earlier. She was then in the thirty-sixth or thirty-seventh week of her first pregnancy. The pains had the character of first-stage labour pains. External examination showed the child to be lying in the left occipito-anterior position. No vaginal examination was made as at the routine examination two or three weeks before no abnormality had been discovered. At 6 p.m. the pains became much more severe and I commenced the administration of chloroform through Junker's inhaler. At 6.15 the patient, although under the influence of chloroform, made a violent expulsive effort and the child was born. The infant weighed only 5 lb. and its head was very soft. Immediately following the birth of the child a large haemorrhage occurred. The patient was turned on to her back and the fundus of the uterus grasped; this was found to be well contracted. A hand was then passed into the vagina, and a laceration was found which admitted the whole hand and which extended through the cervix and lower uterine segment into the left broad ligament. The placenta was rapidly removed from the cavity of the uterus and the cervix drawn down to the vulval orifice. Two volsellae were then placed side by side at right angles to the cervical axis so as to take a broad grip of the walls of the tear and the cervix was then allowed to ascend to its normal position. The volsellae thus made a partial torsion of the line of laceration and controlled the bleeding. There was no damage to the vaginal walls or to the perineum. The usual methods to combat the shock were then adopted. Dr. Thomas Wilson saw the patient in consultation at 8.30 p.m., and it was decided to leave the volsellae *in situ* for forty-eight hours, and unless the haemorrhage recurred to carry out no further surgical procedure. The tear healed soundly and the patient made an uninterrupted recovery, but a certain amount of tenderness has persisted low down in the left iliac fossa ever since.

Seven months later the patient found herself pregnant again. No complications arose during the pregnancy, but during the thirty-fifth week it was found that the foetus was lying in the left sacro-anterior position. As spontaneous version did not occur, at the commencement of the thirty-ninth week external version was done under a general anaesthetic. The resulting position was right occipito-anterior. The child was born two days later; it weighed 7½ lb. Except for a slight opening up of the cervical laceration the labour was without complications.

The cause of the extensive laceration at the first labour is obscure. No complications had arisen during the pregnancy, and as the urine had remained free from albumin it would appear that toxæmic causes need not be considered. The tear extended so far upwards that it was impossible to

determine whether it began in a rupture of the lower uterine segment and spread downwards or in a tear of the cervix and spread upwards. The small size of the baby and the precipitate second stage suggest that the tear must have been caused by insufficient dilatation of the lower part of the cervix and external os, and raise the suspicion that this part of the uterus at least may not have been of full adult development. The juvenile form of cervix so often seen in association with acute antelexion or primary retroversion of small uteri is fairly common, and it may be that this patient had such a cervix. The lateral situation and longitudinal extension of the laceration appear to favour this explanation of its origin, and so does the fact that in the

second labour a $7\frac{1}{2}$ lb. child was born without reopening the old laceration. The menstrual history of the patient up to the first pregnancy also suggests that the uterus was not quite normal. Menstruation started at the age of 14 years, was quite regular every twenty-eight days, lasted four days, but only four pads were required at each period. The patient always had severe abdominal pain before the onset of each period and occasionally passed small clots. Since the birth of the first child pain has been practically absent and the loss has been much more profuse.

I am indebted to Dr. Thomas Wilson for much helpful advice, both in the care of the patient and in the preparation of this note.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, 1923.

SECTION OF ORTHOPAEDICS.

T. H. OPENSHAW, C.B., C.M.G., M.S., F.R.C.S., President.

DISCUSSION ON THE OPERATIVE TREATMENT OF OSTEO- ARTHRITIS.

OPENING PAPER

BY

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ARTHRITIS DEFORMANS is somewhat difficult of definition; it undoubtedly embraces cases due to a variety of causes. McCrae attempts to define it as a group of cases of arthritis which have as prominent features a tendency to chronicity and to the production of more or less permanent changes in the joints or structures about the joints, those forms of arthritis with a definite known etiology (for example, gonorrhoeal arthritis) being excluded. I would make this difference—that it is perhaps better from the practical point of view to retain those cases excluded in the latter part of this definition, if they are chronic and deforming. When considering cases of arthritis from the surgical point of view, one is bound to found one's views of treatment largely on the practical result that the arthritis has produced and not entirely upon the etiological factors. McCrae says it is important to remember that arthritis is always secondary to a process elsewhere; the joint condition is not primary. In this he has perhaps paid insufficient attention to the occurrence of arthritis in a deformed joint—for example, the frequent occurrence of arthritis in coxa vara and coxa plana, in genu valgum, and in joints which have been damaged by intra-articular fractures, or whose alignment has been interfered with by a fracture farther away from the joint.

It is difficult to determine how many diseases are really included under arthritis deformans. It is quite certain that "in all chronic joint changes the same cause may produce varied results and the same results may come from different causes." There appear to be many causative elements, of which we may class as the most important:

1. Infection of the joint with a micro-organism of low virulence.
2. Toxaemia, possibly from a bacterial poison, possibly from a chemical poison.
3. Trauma, including not only direct damage to the joint structures by injury, but also the reaction to an abnormal strain and an interference with the proper fit of the joint surfaces, due to old-standing injury or deformity.

The physiology of joints is not yet very well understood. The secretion of synovial fluid is still somewhat of a mystery; there are no glands for this secretion, and it is

formed apparently by the cells lining the synovial membrane, presumably through some form of proliferation and mucoid change. It is believed that the cartilage covering the bone surfaces is in a constant state of cell growth towards the articular surface. The bone underlying it and around the margins of a joint we know to have potentialities of growth and alteration throughout life. The whole of the interior structures of a joint must thus be looked upon as possessing potentiality for growth, which shows itself in the reaction to any of the above-mentioned causes of arthritis.

Changes in the cartilage in the shape either of thickening or of erosion and ulceration, changes in the synovial membrane in the shape of proliferation or of fibrosis and thickening, and changes in the bone in the shape of overgrowth at the joint margins, may all occur in a chronic arthritis due to any of the many different causes. In addition there is a far greater potentiality for the formation of new tissue in joint structures than is commonly realized. We know that cartilage may not only thicken, but may also extend over osteophytes around the joint margin, that new masses of cartilage may develop in or under the synovial membrane, and that small portions of cartilage or synovial membrane flaked off and lying in the joint cavity may undergo growth into cartilaginous loose bodies, and bone may develop in their interior. We must discount the actual nature of the pathological changes in every case when we try to estimate the causation. It is quite clear that these many changes are of the nature of a reaction of the joint structures to an injury, or to a bacterial or chemical poison, and are none of them specifically due to any one of these causes.

The classification into clinical types—that is, into those mainly characterized by periartritic changes, and others of the atrophic and hypertrophic types—is useful, but is not of any special service from the surgical point of view.

Operative treatment has a very definite place in the treatment of arthritis deformans, but in the main it concerns itself with the removal of mechanical causes and with the treatment of particular functional disabilities. Operative treatment must always take a secondary place to a careful medical investigation and treatment of any primary cause. The first steps in treatment should be:

1. The determination, as far as is possible, of the existence of any specific infection or toxaemia.
2. The determination of the existence of any mechanical cause.
3. The treatment of an infection or toxaemia, if it has been found to exist.
4. The treatment of the more acute periods of inflammation by rest.

The operative procedures which may be useful in arthritis deformans may be classified under the following heads:

1. Operations to correct pre-existing deformities—for example, osteotomy to secure abduction of the hip in coxa vara, osteotomy of the femur for genu valgum, genu varum, and genu recurvatum.
2. Operations to remove an intra-articular cause of the arthritis—for example, removal of a permanently displaced internal semilunar cartilage or of a cartilage which is constantly being displaced, and which by its mechanical effect is producing secondary arthritic changes in the knee-joint; removal of a loose body or of a foreign body from the