The case presents certain interesting features.

1. The rarity of the condition, especially of bilateral "dislocated head" type of synostosis. With a view to determining whether any cases are accidentally missed 500 radiograms taken at the Royal Infirmary of elbow-joints were examined; I was unable to discover a case of synostosis.

2. The importance of recognizing the condition in view of possible "compensation" for the loss of extension and supination, which the anxious parent may only discover for the first time after an accident.

3. In spite of the definite deformity, the little inconvenience entailed; in this case the blow in the region of the elbow was the first idea that the girl's parents had of anything unusual. The condition therefore does not require any surgical intervention.

4. As regards causation, this case does not fall in with the theory of generalized defect of the controlling factor of chondrification and ossification; there were no other defects, such as exostoses.

5. No hereditary influence could be ascertained.

Since the foregoing was written another case of synostosis has been seen by me, but this case belongs to the first of the three types, and is unilateral.

A child, aged 4 years, was brought to the Royal Infirmary on account of "stiffness after knocking the elbow." There was no sign of bruising, and on examination it was found that extension and flexion at the elbow-joint were normal. Supination was not possible, and pronation was only possible through an angle of 20 degrees. There was no evidence of any dislocation, and the bony points seemed to be in normal position. The extensor ossis metacarpi pollicis and extensor brevis pollicis were somewhat taut. The grip was unaffected, but the carrying of objects involved adduction of the elbow. An x-ray photograph was taken, a tracing of which is seen in Fig. E. There were no exostoses, and no family history of a similar condition.

The case was first seen in May, 1930, by Mr. Chisholm, F.R.C.S.,

and now (August, 1930), after massage and movement, the muscles referred to seem normal. The child is able to carry out normal movements with greater ease than when seen in May.

Here again we find certain important factors, which have been already discussed.

1. The condition is first noticed after an injury.

FIG. E.

2. Inconvenience is slight.

3. No hereditary influence could be ascertained.

4. The signs do not accord with the theory of generalized defect of the controlling factors of chondrification and ossification; there was an absence of exostosis, as in the first case.

I am indebted to Drs. Rayner and Harrison for the use of the x-ray films; and to Mr. Chisholm, resident surgical officer to the Infirmary, for sending me the second case.

AN OUTBREAK OF PUERPERAL FEVER IN A NURSING HOME.

BY

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THE present interest in maternal mortality affords an excuse for the narration of the following observations and facts in connexion with an outbreak of sore throat, puerperal infection, and scarlet fever in a nursing home. The house was formerly a private residence standing in its own grounds; it is drained to a sewer, and is in every way hygienic. There are eleven rooms, seven of which accommodate patients. The staff consists of the proprietress-matron, who is a fully trained nurse with the C.M.B., and a fully trained nurse. When there are sufficient patients another fully trained nurse is employed for night duty. The matron has had considerable experience in maternity work in a former home conducted by her, where she had upwards of 300 cases without a death and without a febrile complication to cause anxiety. In the present home she had 83 cases without incident until the first trouble arose in January, 1930.

CASE I.

A, a primipara aged 21, developed a sore throat and then a rash three days after a normal confinement. There was a slight perineal tear, which was sutured. Only one vaginal examination was made by the matron; the doctor in attendance made none. Puerperal fever was diagnosed, and the patient was removed to a nursing home in a neighbouring city, where she received treatment by scarlet fever antitoxin at the hands of a gynaecologist. After a long convalescence, in which albuminuria appeared, she recovered.

Following A there were 8 normal cases, in one of which slight sore throat developed, but the patient said she often had a sore throat of a similar character. This patient was in the home when B was admitted twenty-two days after A had been removed.

CASE II.

B, a primipara aged 23, had a normal confinement, breech presentation, and had had one vaginal examination. There was a perineal tear, which was sutured. On the third day after the confinement she developed a sore throat, temperature 101° F., and had a generalized patchy rash. She was removed to hospital, where she died seven days after the confinement.

There had been no contact between B and the aforementioned patient with slight sore throat. The home was thoroughly disinfected and aired, and no new cases were admitted for a week. The matron was advised to use gauze face-masks when in attendance on patients, and she and her assistants carried out this instruction. Sixteen normal cases followed, and seventy-two days elapsed before C was admitted.

CASE III.

C was a primipara aged 30. Labour was instrumental, and there was a perineal tear, which was sutured. Two days later this patient developed a temperature and a severe pain in the sacral region, which was attributed to rheumatism. This pain was so intense, especially on movement, that the perineal toilet could not be carried out satisfactorily. This pain was presumably secondary to a slowly developing streptococcal infection of the torn perineum. The patient was three weeks in the home, and was seen twice by a gynaecologist. She returned to her own home and then went to a private nursing home, where it was decided that she had had pelvic infection. Recovery took place, but convalescence was prolonged.

While this patient was in the home three others were admitted, and these had normal confinements and puerperia.

CASE IV.

D, a primipara aged 23, was admitted on the day C left the home and had a normal confinement two days later. Two vaginal examinations were made by the matron. The doctor in attendance made none. There was no perineal tear. Three days later the patient developed a temperature and a severe sloughing vaginitis from streptococcal infection. She was removed to hospital, where she died ten days after the confinement.

CASE V.

E, a primipara aged 30, had a normal confinement one day after D's confinement; a perineal tear was sutured. On the seventh day after the confinement she developed a temperature of

104° F. and a rapid pulse. She was removed to hospital, and recovered after prolonged convalescence.

The only patient now in the home was sent to her own home, and the place was closed to further admissions for a week and thoroughly disinfected and aired. The throats of the matron and assistant nurse were examined, but no evident signs of inflammation were present. A swab from the throat of the assistant nurse was taken, and the bacteriological report stated: "Streptococci present—non-haemolytic."

Three days after the removal of E the matron developed a sore throat, and three days later the well-marked rash of scarlet fever. She was removed to the isolation hospital, and the home was again disinfected and the restriction for admission of patients extended for another week.

DISCUSSION.

How did the infection in these cases arise? None of the infected cases occurred in the same room; all were in different rooms, which did not communicate directly with each other. One doctor attended B and C, but there was an interval of seventy-two days between, and this doctor had attended several cases in the interval without incident. The other cases were all attended by different doctors. The matron and nurse had attended 18 primiparae and 8 multiparae between A and E, and all had normal puerperia. In view of these facts it would be difficult to assert that the antiseptic technique followed in the home was faulty, without discovering the fault in the cases that were infected, and this could not be done.

The possibilities of infection from milk were considered, especially when the matron contracted scarlet fever. There did not seem any possibility of her being infected from contact with an infected person. She had only been out in her private car for two short runs in the fortnight previous to the onset of the disease. The milk was supplied from a dairy in bottles, and was said to be pasteurized before delivery. In spite of this the possibility of a milk infection in cases of puerperal fever with sore throat was kept in mind, and the matron was advised to boil the milk supplied to future patients.

While autogenous infection might explain the illness of A and B, it would be wholly erroneous to assume such infection in C, D, and E, and in the matron; who was attacked by scarlet fever. All these cases occurred within a month, and were attacked one after another, so must have been related to some common source of infection. Did that infection enter the home by a visitor or was there a "carrier" among the other patients? It is impossible to say.

THE BLOOD CALCIUM IN DIABETIC RETINITIS.

BY

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In a recent publication Cammidge¹ reports that he has found a low calcium content of the blood in cases of diabetes with retinitis, and has suggested that this calcium deficiency might be the cause of the retinitis. He states that in 81 per cent. of his retinitis cases the blood calcium figures were low enough to be considered "distinctly pathological." It is a little difficult to gather what he considers abnormal figures, because few are mentioned in his publication. In the one case recorded in any detail 5 mg. per cent. is stated to be a "subnormal value," and 6.5 mg. per cent. "the maximum normal figure." Presumably these figures were obtained on whole blood, in which the normal content is usually said to range from 5 to 7 mg. per cent.² It is more usual to estimate the serum calcium, which varies from 9 to 11 mg. per cent., though some authors extend this normal variation from 8 to 12 mg. per cent.

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It seemed important to confirm this new observation on which a therapy might be based, and the point has been investigated in known cases of diabetic retinitis attending our diabetic clinic; no selection of cases was made, but the first ten patients who happened to attend were taken, and our results show that we have been unable to detect any abnormality in the serum calcium contents of these patients. The tests were performed in duplicate by the Kramer and Tisdall method.

Results.

The average serum calcium of these ten cases was 9.6 mg. per cent.; the lowest figure was 8.6, and the highest 11 mg. per cent. Seven cases fell between 9 and 10.5 mg. per cent., and in no case, therefore, was an abnormally low or high figure obtained. Admittedly the number of cases investigated is small, but the uniformly normal results make it clear that the blood calcium has no diagnostic or etiological importance in these cases.

A few other facts of etiological importance in retinitis should be mentioned. Five of the patients were males and five females. The average age was 61 years, the youngest being 44 and the oldest 71. Seven cases were severe enough to require insulin. The minimum ascertainable duration of the diabetes was two years, the maximum twenty years. Three patients showed traces of albuminuria without other evidence of Bright's disease; in them the retinal picture was that of diabetic retinitis alone. Gross arterio-sclerosis was not a noticeable feature. The mean systolic pressure was 150 mm. Hg, the diastolic 90 mm.; the highest systolic pressure was 185 mm., the lowest 100. It is clear, therefore, that true Bright's disease and hyperpietic arterio-sclerosis were not important etiological factors in these cases, which appear to be really "diabetic" in nature.

Summary.

We are unable to confirm the observation that retinitis in diabetics is accompanied by a low blood calcium content, which we have found, indeed, to be normal.

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- 1 Cammidge, P. J.: *Proc. Roy. Soc. Med.*, 1930, xxiii, 292.
- 2 Harrison, G. A.: *Chemical Methods in Clinical Medicine*, J. and A. Churchill, 1930.

MARKED HYPERGLYCAEMIA WITHOUT GLYCOSURIA.

BY

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SINCE some lay down the cast-iron rule that each person has a definite "renal threshold," the occurrence of a case presenting at one time marked hyperglycaemia without glycosuria deserves to be recorded, if not because it is unique, at any rate by reason of its rarity.

A married woman, aged 55, was admitted to the Colonial Hospital, Gibraltar, on March 26th, suffering from a bad septic hand; she was very sallow, unhealthy, and anaemic, and there was marked pyorrhoea alveolaris, with dental caries. The heart was apparently normal, the pulse rate varying from 84 to 90 per minute. Pyrexia was slight or absent, a surprising point in view of the extensive septic lesions in the left hand. The urine was found to be loaded with sugar, and the diacetic reaction was positive. The total quantity of urine was never excessive—at no time more than three pints—and the patient never suffered from undue thirst. For treatment, fomentations and eusol baths were given locally, an iron and arsenic tonic was administered for the anaemia, and a light diet, consisting of bread-and-butter, two Moorish eggs, pudding, beef tea, and one pint of milk, was ordered temporarily. The patient was so unhealthy, and her general condition so bad, that it was considered too risky to place her on any strict diet without further examination and consideration.

Besides the routine daily analysis of the urine in the wards, a specimen was sent to the laboratory on March 28th, when 26.3 grams of glucose per litre were recorded. There was no reaction to acetone or diacetic acid. On the following day the blood sugar and urine sugar estimations were repeated. The blood sugar had advanced to 0.366 gram per litre, while the urine contained slight amounts of acetone and diacetic acid.

On March 30th an injection of 10 units of insulin was given tentatively, and repeated the following morning one hour before