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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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

her dinner. The dose was increased on April 1st to 12 units daily, and a course of colloidal manganese (0.5 c.cm.) was commenced on the next day for her septic condition. During this time the index finger of the left hand was incised under local anaesthesia, and the hand and fingers improved markedly, but two further abscesses started forming above the annular ligaments. The urine was being checked daily in the ward, always with a heavy fall of sugar. On April 4th the insulin dose was further increased to 15 units, and, since her general condition was so frail, an ounce of brandy was started on April 6th. An examination on the next day showed that the blood sugar figure was higher—0.427 per cent.—but the urine sugar figure had fallen to 21.7 grams per litre; the acetone reaction was marked, but only a small amount of diacetic acid was present. In view of this report it was decided to give her two daily injections of insulin, each of 15 units. Her general condition was most unsatisfactory. She was quiet and inclined to be drowsy, and there was apyrexia and anorexia; she practically refused the original diet, and was really only taking milk, eggs, and beef tea. In the evening, as she was so drowsy, it was decided to administer further injections of insulin, 20 units at 9 p.m. and again at 1 a.m. On the morning of April 8th she was semi-comatose and extremely weak; the pulse was up to 100 per minute, and the urine was still loaded with glucose. Insulin in 30-unit doses was administered four-hourly throughout the day and night, and sodium bicarbonate was also ordered. The blood sugar improved on the following morning to 0.356 per cent., and the urine sugar to 4.8 grams per litre, while the urine contained no acetone or diacetic acid. Owing to the feebleness of the heart and pulse, alternate injections of digitaline and strychnine were given to sustain the circulation.

On the morning of April 9th insulin in doses of 30 units was administered at 10 a.m. and 2 p.m., and later repeated four-hourly throughout the night. The patient became mentally clear and less drowsy. The pulse remained rapid at 120 per minute, in spite of cardiac stimulants. On April 10th the blood sugar figure was highly satisfactory (0.184 per cent.); there were only traces of sugar in the urine, which was entirely free from acetone and diacetic acid. The insulin dosage was reduced to 20 units every four or six hours. Signs of threatening hypoglycaemia were noted following the insulin injections, and an orange was ordered to ward off these symptoms.

On April 11th the patient's general condition was very grave, owing to the debility and exhaustion. She was mentally clear and not drowsy; the pulse was still rapid (120 per minute), but it was stronger and more regular. The blood sugar figure was estimated twice; at 11 a.m. it was 0.48 per cent., but at 1 p.m. it rose to 0.633 per cent.; no more than a trace of urine sugar was present. There was nothing to account for this huge sudden increase in the blood sugar. Insulin injections (20 units four-hourly) were resumed, and on the following day the satisfactory blood sugar figure of 0.162 per cent. was recorded, and a trace of glucose only in the urine. The patient's general condition was unsatisfactory, however, and she was obviously dying from exhaustion and debility. On whatever spot of her body she rested a bruise appeared, with or without septic pressure sores, and eventually she died at 6.30 a.m. on April 13th. At no time was acetone noted in her breath.

The point of special interest in her case was the marked hyperglycaemia occurring on April 11th. As will have been noticed, the blood sugar content was twice estimated for fear of error; at the first time it was 0.48 per cent., while on the second occasion, at 1 p.m., it had risen to 0.633 per cent.; glucose was absent from the urine, or only present as a slight trace. Her "renal threshold," after studying the figures of her case, appeared to be somewhere near 0.18 per cent.

The problem arises, Why did such a marked hyperglycaemia develop twice on April 11th, with, at the most, only a trace of glucose in the urine? Why was there no corresponding increase of glucose in the urine? There was the possibility of error in the laboratory. I investigated this point, and discovered that the standard solutions for blood sugar estimations had been used, that they were not diluted, that no error of multiplication could arise, and, finally, that the same solution had been checked on a normal blood sugar case the same day. The ward tests for sugar in the urine confirmed the absence of it, thus excluding the possibility of human error in the laboratory.

The extraordinary condition of marked hyperglycaemia occurring temporarily without glycosuria was recorded in a patient who before and after April 11th reacted normally to insulin, and appeared to have a very definite "leaking point" at about 0.18 per cent. The question arises whether such a contradictory state is possible, and, if so, what could have been the cause, unless it was sepsis, with the corresponding toxins.

A NEW PASTE BANDAGE FOR THE TREATMENT OF VARICOSE ULCERATION AND PHLEBITIS.

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(With Special Plate.)

It is a reluctant general admission among those who have interested themselves for a sufficient period in the healing of varicose ulceration that the ideal method of treatment in this treacherous and stubborn condition is prolonged rest in bed with elevation of the affected limb. This is the only completely satisfactory way of eliminating circulatory stagnation from a varicose superficial venous system, or of draining a massive oedema which results from profound venous thrombosis. The ulcer so treated gradually becomes analogous to a healthy wound on any other part of the body, and healing is then rapidly accomplished.

So seldom, however, is this Utopian arrangement possible that numerous ambulatory methods of treatment have been evolved, each contributing to the degree of its success by the efficacy of the same basic factor—that of applying a firm mechanical support which is designed to compress the varices and to prevent the accumulation of fluid in the tissues. These methods may be broadly classified under the following headings: (1) crêpe bandages; (2) woven elastic hosiery of all varieties; (3) rubber bandages; (4) strips of adhesive plaster applied in numerous ways; (5) cloth or chamois leather gaiters; (6) closely fitting plaster-of-Paris bandages (still adhered to by some heroic practitioners); and (7) Unna's paste dressings. It is not our intention to offer a general criticism of these various measures, or to discuss their individual efficiency, costliness, or difficulty of application.

Until three years ago we confined our experiments in the treatment of ulcers and phlebitis to the original zinc-gelatin paste of Unna, and to the numerous modifications of this formula which have since been introduced. The following tabulated group, in which the figures throughout represent parts by weight, will serve to indicate the diversity of opinion as to the ideal paste.

Varieties of Unna's Paste.	Zinc Oxide.	Glycerin.	Water.	Gelatin.	Starch.	Gum Arabic.
The original	10	15	40	20	—	—
Thomas	10	20	20	10	—	—
The soft (Gougerot) ...	10	17	30	10	—	—
The hard... ..	10	30	30	30	—	—
Saint-Louis	10	30	30	25	—	0.5
Héry	10	40	40	10	10	—
Leroy	10	25	45	12.5	—	—
Segard	10	20	40	40	—	—
Brandon	10	16	22	10	—	—
Gilson (Manchester) ...	10	12	22	6	—	—
Smith (Glasgow) ...	10	10	10	10	—	—
Clasen	10	40	30	20	—	—

We are satisfied that the good results following the use of zinc-gelatin dressings are due to the following factors:

1. The medicaments are useful. Glycerin is dehydrating and hence antiphlogistic; it has a definite place in the reduction of oedema. Zinc oxide, again, is both antiseptic and astringent.
2. When applied on light dressing material the paste does not prohibit the evaporation of skin secretions, and it allows air-access to the tissues. These advantages are not obtainable with rubber or elastic webbing bandages.
3. When properly applied the zinc-gelatin bandage forms a satisfactory mechanical support.