of sanocrysin. He was discharged on August 15th, 1928, with a normal temperature and pulse 72 to 92; there was no sputum. The physical signs and *x*-ray appearances were much improved.

9. A woman, aged 33; admitted March 3rd, 1928; onset December, 1927, "influenza." Most of the right lung was affected, and the left middle zone; L3, S2. Sputum was scanty. Evening temperature 99° F., pulse 84 to 120. She received a total of 4.5 grams of sanocrysin. When discharged, on June 5th, 1928, there was no sputum and the physical signs were much fewer; the improvement had been very rapid in a short time.

10. A woman, aged 24; admitted October 29th, 1927; onset August, 1924, pleurisy. There was extensive disease on the right with cavitation, and slight disease on the left; L3, S2. Sputum 3 to 4 oz. The evening temperature was 99° F., pulse 88 to 108. She was given in all 6.6 grams of sanocrysin. She was discharged on April 12th, 1928, with the physical signs much improved, sputum 1/2 oz., bacilli fewer but still present. The general improvement was maintained. She was readmitted on October 20th for a second course of sanocrysin (3.6 grams). When discharged, on December 22nd, there was further improvement in the physical signs and the sputum was negative.

Cases in which Patients became worse and where the Disease was probably aggravated by Sanocrysin.

11. A woman, aged 34; admitted June 25th, 1928; onset February, 1926. L3, S3. There was extensive disease of both lungs. The evening temperature was about 100° F., pulse 88 to 108. Small doses of sanocrysin were given after the temperature had been allowed to settle. After the fourth injection the patient had haemoptysis with fever (see Chart 3). There was no previous history of haemoptysis. The physical signs had increased.

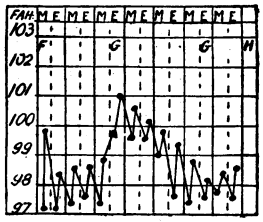

CHART 3.—F, 0.1 gram sanocrysin (fourth injection). G, Haemoptysis. H, Signs of extension of disease.

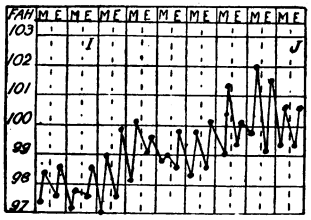

CHART 4.—I, 0.3 gram sanocrysin. J, Fever continued for fourteen days longer.

12. A woman, aged 22; admitted May 3rd, 1928; onset December, 1926. L3, S2. Pulse 96 to 132. The left lung was extensively involved, and the right upper zone was affected. Dermatitis occurred after the second injection, and treatment was stopped for four weeks. Seven doses were then given at weekly intervals. The temperature was rarely above 99° F. After the last injection the temperature began to rise and the patient had a continuous pyrexia with increase of physical signs (see Chart 4).

Conclusions.

We recognize the possible fallacies in attempting to draw conclusions in a disease where the unexpected so often happens, even in the absence of any form of treatment, and especially where more than one form of treatment has been employed. But after mature consideration we feel justified in making the following statements.

Sanocrysin cannot be regarded as a specific in the treatment of pulmonary tuberculosis. In suitable cases it shortens the period of routine treatment—that is to say, a similar degree of improvement may be obtained in considerably less time with sanocrysin than without.

With sanocrysin there is a greater possibility of the improvement following institutional treatment being maintained than there is without its use.

In carefully selected cases it is of value as an adjuvant to other methods of treatment. It is of definite worth in clearing the "better" lung in bilateral disease and allowing the establishment of an artificial pneumothorax on the "bad" side. It may also be employed with advantage in collapse therapy, of any variety, when there is a tendency for the "good" side to break down.

Used with care it is of value in the treatment of the acute exacerbations in chronic fibroid disease.

By ridding the sputum of bacilli even for a short time it must—theoretically, at any rate—limit the spread of the disease.

Sufficient time has not yet elapsed to allow us to dogmatize on the permanency or otherwise of the improvement.

CASES OF DIABETES MELLITUS WITH A LOW RENAL THRESHOLD.

BY

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THE renal threshold, or leak point of the kidney for sugar, is usually about 0.17 to 0.19 per cent. blood sugar, and variations are recognized to occur in diabetes mellitus. Most commonly any change consists in a raising of the threshold above 0.2 or even 0.3 per cent. blood sugar, with the result that urine tests for sugar give little indication of the satisfactory control of the disease, making the patient appear sugar-free when the blood sugar is high. Much more rarely diabetes has been reported to be complicated by a lowered renal threshold. One case each has been reported in the last two years by Voigt,[1] Shapland,[2] and by Curran and Mills,[3] their articles leaving the impression that the combination of diabetes mellitus and a lowered renal threshold is a very rare condition. Consequently the following eight cases seem worth recording, as the condition leads to confusion and difficulty in the practical management of the cases.

Diagnosis of the Condition.

This presents no difficulty when a case of diabetes is being followed by blood, as well as by urine, sugar tests. As the result of treatment it is found that, whereas the blood sugars are becoming normal, the urine does not become correspondingly sugar-free. Positive proof that the renal threshold is abnormally low can be obtained by simultaneous tests of the blood and urine for sugar on one or two occasions. For example, after insulin has been given at 8 a.m. the blood and urine sugar may be tested as follows:

Time:	10 a.m.	11 a.m.	12 noon.	1 p.m.
Blood sugar per cent. ...	0.19	0.12	0.085	0.080
Urine sugar	+++	++	trace	0

This proves that the threshold is below 0.12 per cent., but not so low as 0.08 per cent. The condition can often be proved quite definitely by estimating the blood sugar only once and testing the urine secreted afterwards. In the above example the leak point could be proved to be below 0.12 per cent. by finding sugar in the urine passed at noon after the bladder had been emptied at 11 a.m.

Description of Eight Cases.

An individual description of these eight cases seems unnecessary. All the patients had had typical symptoms of diabetes mellitus and in all an abnormally high blood sugar had been found, either on ordinary diet or after a glucose tolerance test. In three cases diet alone controlled the blood sugars; in five insulin was necessary. Five were males and three females, and their ages varied from 19 to 68. In two cases there was a family history of diabetes.

The son, aged 14, of one of these patients has no symptoms of diabetes, but on two occasions has shown an abnormally high blood sugar curve with slight delay in the return to normal. Unlike his father, this boy has a raised renal threshold and passed no sugar at a blood sugar concentration of 0.225 per cent.

The level of the renal threshold was fairly closely observed in these cases, and was respectively 0.15, 0.14, 0.11, 0.1, 0.1, 0.09, 0.09, and 0.08 per cent. It is obvious that the last few cases were hardly ever free from traces of sugar in the urine. The amount of glycosuria was in every case related to the level of the blood sugar, and the more the latter exceeded the threshold the more sugar appeared in the urine. This is contrary to what is usually stated to occur in renal glycosuria in non-diabetics, where

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the amount of glycosuria is said to be usually constant and independent of diet and the level of the blood sugar. This has not been true, however, in my experience of non-diabetic renal glycosuria.

In two of these diabetics the lowered threshold has remained constant for five years, and in none, observed over shorter periods, has it been found to vary. There is every reason to think, therefore, that the peculiarity is a permanent one, as renal glycosuria seems to be.

In addition to these eight cases, a lowered threshold appeared temporarily during pregnancy in two diabetic women, but became normal again after labour.

Practical Importance of the Condition.

These cases seem, on the whole, to follow the same course as diabetes with a normal renal threshold. They have not seemed more liable to ketosis than other cases, because they have not lost sufficient sugar in the urine to affect materially the ketogenic-antiketogenic ratio of the diet as fed. But one of the pregnant women, who was losing some 30 grams of sugar a day by the third month of pregnancy owing to the low threshold, excreted more ketone bodies than previously. The fasting blood sugar level might have been expected to be lower than usual from the loss of sugar, but this was not noticeable. However, two of the youngest patients, who had for years after the original discovery of their diabetes paid little attention to their condition, seemed to deteriorate much less rapidly than the usual careless juvenile diabetic. One of these patients gave little heed to diet for seven years, was thirsty, and had a blood sugar of nearly 0.3 per cent. when tested, and yet became rapidly controlled on diet without insulin, and has remained so since. Another, a girl of 12, had diabetes for seven years without any marked progression, and always responded at once to dietetic treatment. It seems possible that the low leak point acted in these cases as a safety valve, which tended, by the loss of sugar in the urine, to lower the blood sugar and relieve the strain on the islet cells. This is my impression in these two cases, but, of course, they may have been exceptionally unprogressive for other reasons.

It would appear that these cases respond to treatment in the usual way, but they certainly present some difficulty in their management. It is impossible to keep the urine sugar-free if the threshold is below 0.1 per cent. The usual urine sugar tests give little or no indication of the patient's real condition, which can be known by blood sugar tests alone, difficult to carry out frequently, and sometimes impossible. It is very disappointing to the patient, and the practitioner, to find sugar almost constantly present in spite of treatment, and the patient, at least, is apt to be depressed. It is best for these patients not to be taught to test their urine or to co-operate as much as usual in their treatment. If the threshold is lowered to only 0.14 per cent. or so, this may be almost an advantage in treatment, as one may then be certain, if the urine is sugar-free, that the blood sugar is normal.

It is difficult to see any etiological connexion between the lowered threshold and diabetes in these cases. It is highly improbable that the diabetes produces in some way a lowering of the threshold, for any change that is known to take place in diabetes is in the direction of a rise in the threshold; nor is there any clear evidence that a pre-existing condition of renal glycosuria may tend to produce or be followed by true diabetes. Indeed, Hjärne[4] has recently published a hereditary study of renal glycosuria which tends to show the absence of any connexion between diabetes mellitus and renal glycosuria.

The most likely explanation of these cases is the fortuitous development of true diabetes in cases of renal glycosuria. It is now well known that a lowered threshold is fairly common in normal individuals, though what percentage of the population are renal glycosurics it is impossible even to guess; but the number of cases of renal glycosuria that are discovered must be very small compared to the number existing. The above eight cases have occurred among some 500 diabetics—an incidence of 1.6 per cent., not a high one, probably no higher than in the non-diabetic population. Quite likely among my 500 diabetic cases others with a lowered threshold have escaped discovery, because it requires close observation and not infrequent blood sugar tests (not possible in every case) to establish the condition. There seems no reason to think that the combination of diabetes mellitus with a lowered renal threshold is not a mere coincidence.

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VOMITING IN INFANCY.

BY

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VOMITING is a very common presenting symptom during the first few months of life, and one about which the mother is not unnaturally anxious, as it signifies to her that her child is not getting sufficient nourishment.

In the act of vomiting the walls of the stomach contract, the diaphragm is pushed downwards in the full expiratory position, while powerful contractions of the abdominal muscles take place; at the same time the cardiac sphincter is relaxed and the gastric contents are expelled, chiefly as the result of the pressure exerted in the abdomen by the diaphragm and the abdominal muscles, aided to some extent by reversed peristalsis. As a general rule the pyloric sphincter is closed, but in some cases it may become relaxed, with the result that bile and intestinal contents are expelled with the vomit. The simplest method of bringing this mechanism into play is to overfill the stomach, especially if this is done rapidly before the walls of the stomach have time to adapt themselves to the sudden increase of gastric contents. Thus it is extremely common for breast-fed babies to vomit a small amount at the end of their feeds. Provided the gain in weight is within normal limits no treatment is required, and the symptoms may be regarded as one of Nature's "safety valves." If, however, it is found that the baby is gaining weight excessively, a little water should be given beforehand and the actual length of time spent over each feed decreased.

Vomiting from Birth.

All cases in which such a history is given should be regarded seriously, as a congenital obstruction of the duodenum is a possible cause. It is also important to realize that the symptoms of congenital pyloric obstruction may date from birth. As a general rule the vomit in the former is bile-stained, and jaundice may occur, but the distinction between the two conditions is really only of academic interest, as surgical intervention is indicated in both.

Various congenital malformations of the oesophagus that produce intractable regurgitation of food dating from birth are occasionally encountered. Some form of fusion of trachea and oesophagus is the most common deformity, and screening with the *x* rays may sometimes show conclusive evidence of obstruction. Treatment is of no avail.

Vomiting with an Acute Onset.

The sudden onset of vomiting, associated with diarrhoea, in an apparently healthy baby suggests immediately the possibility of an infection, of which the most likely site is the gastro-intestinal tract, particularly in artificially fed children. It is important, however, to realize that parenteral infections may produce exactly similar symptoms; the most common of these are pyelitis, bronchitis, and otitis media. Recent work, both in this country and in America, has drawn attention to the great frequency of acute pyelitis in babies, and unless the urine is examined the condition is frequently missed. In severe cases the onset is sudden, with marked fever, vomiting, diarrhoea, and convulsions or rigors; the latter are uncommon in children, and should always suggest an acute kidney infection. Other cases have a more insidious onset, with vomiting and persistent loss of weight as the outstanding features. It is essential in