cirrhosis in post-natal life consists in the further change in the large bile ducts and gall bladder. An attempt to explain this additional lesion may be made as follows: The bile ducts are extremely small at birth, and any inflammatory change will, from the small size of the lumen, produce stenosis much more readily than later in life. An analogous effect is seen in the fact that laryngeal obstruction in diphtheria is more frequent in young subjects than in older patients, quite apart from the much greater frequency of the disease in the young. The opposed inflamed surfaces of the bile ducts will also come in contact more readily, and, as in catarrhal appendicitis, obliteration might result.

The following considerations bear on the hypothesis that the disease is primarily a congenital cirrhosis.

1. The almost constant occurrence of cirrhosis in these cases of bile duct obstruction in infants as compared with the infrequency and irregularity with which cirrhosis follows obstruction of the larger bile ducts in later life. The mixed character of the cirrhosis explains the discrepancy in the recorded cases, some authors speaking of biliary, others of multilobular, cirrhosis.

2. The large size of the liver—this resembles hypertrophic biliary cirrhosis. In simple obstruction of the larger bile ducts in adults, the liver, though swollen from retained bile in the early stages, is usually small after death.

3. The large size of the spleen—a phenomenon not met with in uncomplicated biliary obstruction. The large size of the spleen is best explained as the result of toxic bodies reaching the organ by the splenic artery. In hereditary syphilis, where it is probable that the poison reaches the liver by the umbilical vein and is derived from the maternal circulation rather than that the ovum is infected by a syphilitic spermatozoon, there is a similar splenic enlargement. In both conditions there is cirrhosis due to poisons arriving by the umbilical vein; the difference between the pericellular cirrhosis of hereditary syphilis and the mixed (monolobular and multilobular) cirrhosis of so-called congenital obliteration of the bile ducts must depend on a difference in the poisons in the two diseases. This is at one with Thomson's statistical proof that syphilis plays no part in the antecedents of so-called congenital obliteration of the bile ducts.

4. The fact that in some instances several cases of this rare disease have occurred in the same family. Hypertrophic biliary cirrhosis not infrequently occurs in several members of the same family, and has been thought by Boix to be a waterborne disease. Against the view that so-called congenital obliteration of the bile ducts is in reality a form of congenital cirrhosis, it might with reason be objected that the poison that sets up the change must pass through the mother, and that she should show evidence of its influence. It must be admitted that there are at present no data to provide a satisfactory answer to this argument. As bearing on this point, however, it may be mentioned that the extremely fatal biliary cirrhosis in Brahmin infants around Calcutta, which is also a family disease, has been thought to depend on the mother's milk. The mothers restrict themselves to a dry diet and take a decoction of black pepper. If this is the causal factor, it evidently affects the nurslings more than the nurses, and might justify the suggestion that in fœtal life the infant's liver may be more susceptible than the mother to poisons tending to produce cirrhosis; while again the effects of syphilis may be, and usually are, much more manifest in the infant than in the syphilised mother.

To sum up, it seems reasonable to believe that the disease is primarily started by poisons derived from the mother, and conveyed to the liver of the fœtus, and that a mixed cirrhosis and cholangitis are thus set up. The cholangitis accounts for the jaundice, and by descending to the larger extrahepatic bile ducts induces an obliterative cholangitis analogous to obliterating appendicitis.

In some cases, especially those fatal early in life, the latter change has not been affected, and cirrhosis alone is found. Possibly in some instances this change never occurs, and in this way some of the cases of cirrhosis in very early life are accounted for. Again in exceptional instances the obliterative cholangitis might possibly be delayed, and come on much later; such an event might bring Treves's case, already referred to, into line with the others.

It is possible that there are several conditions at present included under the title congenital obliteration of the ducts, and that some, such as D. Ross's case, are due to constriction of the duct by localised peritonitis, and deserve the title better than those cases that are intimately associated with cirrhosis.

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ON THE TREATMENT OF GLYCOSURIA AND DIABETES MELLITUS WITH SODIUM SALICYLATE.

By R. T. WILLIAMSON, M.D.Lond., F.R.C.P.,

Physician to the Ancoats Hospital, Manchester, and Assistant Lecturer on Medicine, Owens College.

It is somewhat difficult to form a correct estimate of the action of drugs on the sugar excretion in cases of glycosuria and diabetes for many reasons. The occurrence of complications, such as phthisis for example, in the course of the disease may cause a diminution of the sugar excretion, which at first sight appears to be the result of medical treatment. Also in the treatment of diabetes the diet is usually restricted, more or less, at the same time that various drugs are prescribed; hence it is often impossible to say how much of the improvement which follows is due to the action of the restricted diet and how much to the drug. In the most severe form of diabetes it is usually easy to demonstrate that no drug has much influence on the sugar excretion. But in the milder forms of diabetes or persistent glycosuria, considerable care is necessary in forming an opinion as to the action of various drugs. Before attributing a diminution of sugar excretion to the action of any drug, it is important to show that such a diminution is not due to a restricted diet or to the occurrence of some complication. Also in certain rare cases of the mild form of diabetes there are periods in which the sugar excretion decreases markedly, quite apart from treatment (cases of intermittent diabetes).

Recently I have endeavoured to ascertain whether sodium salicylate has any definite influence on the sugar excretion in glycosuria and diabetes. This drug was strongly recommended in diabetes long ago by Professor W. Ebstein, of Göttingen, and though a number of papers have been since published both in English and foreign medical literature, recording its supposed favourable action, still it does not appear to be now prescribed very extensively for diabetes in England. About ten years ago I gave it a careful trial in several severe cases of diabetes, but only in small doses. I was never able to detect any definite diminution of the sugar excretion under its use in these cases; but often the patients stated that they felt better when taking the sodium salicylate than when other drugs were given. Recently I have prescribed the drug in large doses in a number of consecutive cases of diabetes and glycosuria, and in 20 of these I have been able to keep records of the sugar excretion from time to time. But I wish especially to record the observations which I have made in 1 case that has been under my care, both as an in-patient and an out-patient at the Ancoats Hospitals for twelve months. Altogether the patient was in the hospital for twenty-two weeks, and during this period I estimated daily the sugar excretion.

Case 1.—The patient was a woman, aged 70, who had suffered from diabetic symptoms for 10 years. The case may be described as one of mild diabetes mellitus or persistent glycosuria. There were no complications, and the amount of sugar excreted was fairly stationary apart from medical treatment. I first saw her in 1894. At that time the urine had a specific gravity of 1032, and contained 24 grains of sugar to the ounce. In 1899 the specific gravity was 1034 and the amount of sugar 25 grains to the ounce before treatment was

commenced. The amount of urine was from 70 to 80 ounces daily. The case was in every way suitable for making observations respecting the action of drugs on the sugar excretion. The period of observation was sufficiently long, and the diet was kept unaltered in order that there might be no possibility of the action of diet being attributed to the influence of drugs. There was nothing peculiar with respect to the previous history or symptoms of the patient. There was no family history of the disease; the symptoms of diabetes had appeared after the patient had been subjected to much anxiety and overwork in connection with the nursing of her husband for three years through a long illness which proved fatal.

On examination of the patient nothing abnormal could be detected in the chest or abdomen. The knee-jerks were present. The radial arteries were atheromatous. The patient was well nourished, but not obese. The urine gave no reaction with perchloride of iron, and contained no albumen.

The patient was admitted into the Ancoats Hospital on November 15th, 1899, and for twelve weeks the amount of urine and the sugar excretion were estimated daily. She was allowed the ordinary hospital diet, with the exception of sugar, rice pudding, and potatoes; these three articles she had not taken for several years; so that her diet remained practically the same as it was before she came under treatment. She was allowed ordinary white bread and milk in unlimited quantities. (At a later period, as will be recorded, potatoes were allowed daily.)

It is not possible to publish the figures showing the daily sugar excretion during this period of twelve weeks, owing to limited space, but the results were as follows:

First Period.—For eleven days no medicine was given except chloroform water (one tablespoonful three times a day). During this period the condition was as fellows: Daily amount of urine, 60 to 86 ounces; specific gravity, 1034 to 1037; amount of sugar, 24 grs. to 30 grs. per ounce; total sugar excretion, 1,584 grs. to 2,400 grs. daily.

Second Period.—For fifteen days the action of heroin or uranium nitrate was tried. The doses were small, and only a slight diminution of the urine and of the sugar excretion was observed during this period. The daily amount of urine was from 56 to 64 ounces; specific gravity, 1030 to 1038; amount of sugar, 22 grs. to 32 grs. to the ounce, and the daily excretion 1,150 grs. to 1,680 grs.

Third Period. Sodium salicylate was now commenced (on the twenty-sixth day after the patient was admitted into the hospital). At first 10 grs. were given three times a day, then four, five, and six times a day; afterwards 15 grs. were given four times a day. The sugar excretion steadily diminished, and at the end of twenty-seven days the amount of urine was 43 ounces, the specific gravity 1029, the sugar excretion 10 grs. to the ounce, or 430 grs. in the twenty-four hours.

Fourth Period.—In addition to the 15 grs. of sodium salicylate four times a day, salicylate of bismuth was now given as a powder for seventeen days, in 4-grain doses, at first twice a day, then three times a day. This was followed by a slight diminution of the sugar excretion, and at the end of the period the amount of urine was from 47 to 59 ounces, the specific gravity 1022 to 1028, the sugar from 5 grs. to 8 grs to the ounce, and the daily quantity excreted from 295 grs. to 456 grs.

Fifth Period.—During the last fifteen days the patient was in the hospital 15 grs. of sodium salicylate were given five times a day. For five days the sodium salicylate was given alone, but during the last nine days a powder containing 10 grs. of bismuth salicylate was given in addition three times a day. The sugar excretion gradually diminished. The condition during the last ten days was as follows:

Amount of Urine.	Specific Gravity.	Grains of Sugar per Ounce.	Daily Excretion of Sugar in Grains.
54	1022	6	324
53	1019	2	106
49	1024	3	147
53	1023	1	53
47	1020	Less than 1 gr.	—
53	1020	1	53
49	1019	Less than 1 gr.	—
43	1021	,, ,,	—
47	1022	,, ,,	—

At the end of this period the amount of sugar was too small to estimate by the fermentation test. On boiling with Fehling's solution there was only a slight greenish turbidity, which occurred as the test tube cooled. There was no thirst, the patient's general condition was good; she felt quite well, and had gained 5 lbs. in weight. No complication had developed.

The patient went home, and discontinued the salicylate. She returned as an out-patient a month later. The specific gravity of the urine had risen to 1031, and the sugar to 25 grs. to the ounce. She was given 20 grs. of sodium salicylate four times a day; she returned twenty-seven days later (having taken the medicine regularly); the urine had then a specific gravity of 1023, and contained only 3 grs. of sugar to the ounce.

The patient then left Manchester, and discontinued treatment. In August, 1900, she returned as an in-patient at the Ancoats Hospital. She remained in the hospital for ten weeks, and I estimated the sugar excretion daily during this period. The patient was allowed the ordinary hospital diet, with the exception of sugar and rice pudding, which she had not taken for years. In order that there might be no possibility of any diminution of sugar excretion being attributed to altered diet, I allowed her to take potatoes in the same quantity as non-diabetic patients in the hospital, though she had not taken them for several years. She was allowed, also, ordinary white bread and milk in unlimited quantities.

After she had been in the hospital eight days she was given simply one teaspoonful of peppermint water three times a day, for three days, in order to estimate the sugar excretion apart from the action of any drug. The result was as follows:

Amount of Urine.	Specific Gravity.	Grains of Sugar per Ounce.	Daily Excretion of Sugar in Grains.
68	1033	23	1,564 } Taking peppermint water 5j three times a day.
68	1030	22	1,496
70	1032	22	1,540

Sodium salicylate was now given, 20 grs. three times a day, and afterwards four times a day. The sugar excretion diminished steadily. At the end of twenty-four days the sugar excretion was only 2 grs. to the ounce, and the total for the twenty-four hours only 48 grs. (see table, A.). The sodium salicylate was then discontinued and chloroform water given (mv four times a day). The sugar excretion at once increased, and in six days had reached 18 grs. to the ounce, and 1,152 grs. for the twenty-four hours (see table, B.). Sodium salicylate was again given (20 grs. four times a day). The sugar excretion rapidly diminished, and in six days was only 1 gr. to the ounce and 39 grs. for the twenty-four hours (see table, C.). During the next fourteen days, until the patient left the hospital, salicylates were given four times a day (the dose being some days 20 grs., on other days 15 grs.). The sugar excretion continued to diminish; during the last nine days the patient was in the hospital the sugar excretion never reached 1 gr. to the ounce. On testing with Fehling's solution, only a very slight reduction was obtained (a greenish turbidity some time after boiling).

On leaving the hospital, the patient felt quite well; there was no thirst, the appetite and general condition were good; her weight was 11 st. (on admission it was 10 st. 10 lbs.). The specific gravity of the urine was 1015, and only the slightest trace of sugar was present—less than 1 gr. to the ounce. No complications had developed.

	Daily Amount of Urine.	Specific Gravity.	Grains of Sugar per Ounce.	Grains of Sugar Excreted Daily.
A	34	1027	2	68 } Sodium salicylate gr.20 four times a day.
	22	1026	2	44
	24	1022	2	48
B	33	1026	4	132 } No medicine except chloroform water mv four times a day.
	62	1020	8	496
	58	1021	9	522
	64	1022	12	768
	62	1026	16	992
	64	1027	18	1,152
C	67	1026	16	1,072 } Sodium salicylate gr.20 four times a day. *
	65	1029	15	975
	66	1022	6	396
	45	1026	7	315
	38	1028	7	266
	39	1021	1	39
	48*	1023	1	48
D	59	1016	—	} Last three days in hospital.
	42	1017	—	
	67	1015	—	

* Sugar excretion steadily diminished during the next twelve days under salicylates. For the last nine days only a trace of sugar was present—less than 1 grain per ounce.

The results just recorded appear to me to prove conclusively that in this case sodium salicylate had a definite influence in greatly diminishing the sugar excretion. The other conditions, diet, etc., being kept the same, when the drug was given in large doses (75 to 80 grs. daily) the sugar excretion greatly diminished; when the drug was discontinued the sugar excretion at once increased rapidly; when the drug was again given the sugar excretion greatly diminished.

In 19 other cases of diabetes or persistent glycosuria I have recently given sodium salicylate; but as only one of these cases was in hospital the results are much less reliable than those in the case just recorded; also in most of these cases the diet was restricted at the same time that the sodium salicylate was given. They may be divided into four groups.

I. In 7 cases the diet had been restricted before the patients came under my treatment. Potatoes, rice, and saccharine articles had not been taken for some time, but all were taking bread or some bread substitute loaded with starch. The diet was kept the same, and sodium salicylate (which had not been taken previously) was prescribed. All were cases of the mild form of diabetes or (as some writers would describe them) cases of persistent glycosuria. The amount of urine in these 7 cases was normal, or not much above the normal quantity. The number of grains of sugar per ounce of urine in the 7 cases at the commencement of the sodium salicylate treatment, and the results of the treatment were as follows: Case II.—Sugar, 13 grs.; in ten days a trace only, less than 1 gr. Case III.—Sugar, 16 grs.; in one month slightest trace only, much less than 1 gr. to ounce; drug discontinued; sugar rose in six days to 6 grs. to ounce; drug commenced again; sugar steadily decreased,

in ten days only slight trace present, less than 1 gr. to ounce. Case IV.—Sugar, 15 grs. to ounce; diet kept unchanged; after six weeks of sodium salicylate treatment sugar only 3 grs. to the ounce; in 20 days later no reaction for sugar by usual methods of testing, but by special methods the slightest trace detected. Case V.—Sugar, 30 grs. to ounce; in sixteen days 6½ grs. to ounce. Case VI.—Specific gravity 1033; sugar, considerable amount; in three weeks a trace only; a month later sugar absent. Case VII.—Sugar excretion had been arrested by restricted diet; a few weeks later sugar returned (24 grs. to the ounce); diet kept the same, and sodium salicylate given; in two weeks only slightest trace of sugar present; shortly afterwards it disappeared; six months later sugar again appeared in the urine, but disappeared when sodium salicylate was given. Case VIII.—Sugar, 2 grs. to the ounce; reduced to slightest trace (not detected by Fehling's solution) under sodium salicylate. I am prepared to admit that in the Cases II to VIII the results were not quite conclusive, since the patients were not in hospital; but I think it probable that in most, if not in all, of these cases the sodium salicylate had a favourable action.

II. In one case, specific gravity of urine 1033; sugar 29 grs. to the ounce. No treatment previously. Diet was restricted (potatoes, rice, and food containing sugar forbidden, but bread allowed) and sodium salicylate given. The sugar steadily diminished, and in seven weeks had entirely disappeared. The restricted diet has been continued. During the last ten months the urine has been examined every week. Seven months ago the sodium salicylate was discontinued. On three occasions a trace of sugar has returned, but has disappeared when sodium salicylate has been taken.

III. In 5 severe cases of diabetes (the age of the patients being 19, 31, 35, 38, and 40 respectively) there was only a very slight diminution of the sugar excretion, but the symptoms were relieved and the patients gained weight and felt better when taking the drug. In these 5 cases the diet, though restricted, was the same as that taken before the drug was commenced. In 4 cases of medium severity the drug appeared to produce no definite effects, but it was not possible to watch these cases very carefully.

IV. In one case the patient was losing flesh when he commenced the sodium salicylate. After taking it for five days he stated that the diuresis was greater. The drug was at once discontinued, and other treatment given. Death occurred nineteen days later. In another case sodium salicylate was given for seven days, at first in doses of 15 grs. three times, afterwards four times, a day. It was then discontinued. Six days after the drug was discontinued death occurred.

Limited space forbids an account of these cases, but I fail to see any reason for attributing the fatal termination to the administration of the drug for the short periods named. It is not advisable to give sodium salicylate if serious complications are present, or if the patient appears to be losing ground rapidly, because the drug has a bad reputation with many practitioners, and a fatal termination during the salicylate treatment is liable to be attributed the drug.

I may point out in conclusion that I do not regard sodium salicylate as a specific for diabetes. It does not usually produce any marked diminution of the sugar excretion in the severe forms of the disease; also it has little influence in some of the mild cases. But in certain mild cases of diabetes or persistent glycosuria, as in the first case recorded in this article, it has a decided action in very markedly diminishing the sugar excretion.

The drug is not suitable in all cases of diabetes. It requires to be very carefully watched, and fairly large doses are usually necessary to produce decided results. The natural sodium salicylate is more satisfactory than the common artificial preparation. It is best to commence with 10 grs. three and then four times a day, and to increase slowly up to 15 grs. four or five times a day, watching carefully for any toxic symptoms. I have usually given it in peppermint water, or in equal quantities of peppermint water and ordinary drinking water.

In severe forms of diabetes, though sodium salicylate does not usually cause much change in the sugar excretion, still the patients sometimes gain weight and improve in general condition whilst taking the drug.

AN EASY OPERATION FOR CONGENITAL PTOSIS.

By FREELAND FERGUS, M.D.,
Surgeon to the Glasgow Eye Infirmary.

ANY operation undertaken for the rectification of this abnormality must fulfil two conditions: it must be efficient for the purpose, and it must be so arranged as to leave no cicatrices or scars.

I was recently asked if I could operate on a case of ptosis without leaving much disfigurement, and I employed the following method, which I found easy of execution, and which gave results satisfactory alike to myself and to my young patient's friends:

A horizontal linear incision was made in the eyebrow along its whole extent, this situation being selected because the hairs of the eyebrow afterwards completely hide the scar. No other skin incision is required. With a few strokes of a scalpel the skin was completely separated from the underlying tendon and fascia of the occipito-frontalis muscle; the separation was carried to about a distance of nearly 2 inches above the horizontal wound. In the opposite direction the skin and fascia and portions of the muscular structure were separated from the orbicularis muscle and from the tarsus; the division being carried almost to the free margin of the eyelid.

The next step was to mark out a vertical band of the tendon and fascia of the occipito-frontalis, about three-quarters of an inch broad and 2 inches long. This was dissected up from all underlying structures, and when the dissection was complete its only attachment was to the occipito-frontalis at the part furthest away from the skin incision. The end of the band was drawn down into the upper eyelid, and its margin was secured by catgut sutures as near the margin of the lid as possible. The wound in the skin was closed and covered with a sterilised dressing. The wound healed rapidly and well.

The photographs from which the figures are taken were made about a month after the operation. No. 1 shows the patient after one eyelid had been done. On the right side it

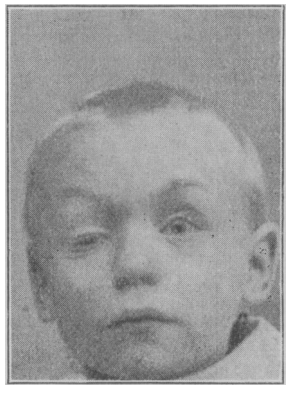

Fig. 1.

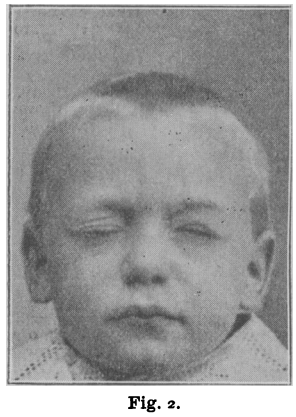

Fig. 2.

will be observed that the ptosis is still present, while on the other it has been overcome by the operation. Figure 2 represents the patient with both eyes shut, from which it appears that there was no difficulty in closing the palpebral fissure.

An operation such as the above seems to present many advantages over those commonly employed. Till now the operation which gave the best results in my hands was unquestionably that of Professor Panas, but his operation, although thoroughly removing the ptosis, always left, so far as my operations were concerned, very ugly cicatrices. I have never tried Pagenstecher's modification of Dransart's operation, as it has always seemed to me to be an extremely crude proceeding to trust to inflammation set up by subcutaneous sutures. Moreover, such sutures will not set up the desired inflammatory reaction if they are perfectly sterile when introduced, and surely modern surgical knowledge entirely forbids the introduction of any sutures which are not thoroughly aseptic.

Since performing the operation I have glanced at the literature of ptosis operations, and the only proceeding at all like the one above described which I have been able to find is that of Kunn[1] described by him in 1893. The differences between the method described above and his are very considerable.

REFERENCE.
1 *Wien. med. Wochenschrift.*

PRESENTATION.—Dr. Livingstone has been presented with a case of instruments bearing the following inscription on a silver plate: "Presented to Dr. Hillhouse Livingstone of Stanhope by the Members of his Ambulance Class at Frosterley, March, 1901."