THE SURGICAL ASPECTS OF DIABETES.

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Thesis submitted for the M. D. Edinburgh

by

James McDonald, M.B. C.M., Edin.1892.

(Late) Assistant Surgeon to Lugar Iron Works and Cumnock Hospital.

Medical Officer, Belford Union.

Medical Officer, Belford Workhouse.

Public Vaccinator Belford Union.

Medical Officer to Post Office.

Police Surgeon.

Certifying Factory Surgeon Belford Union.

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It is a surgical maxim instilled into the minds of every student as soon as he enters the surgical wards, that the urine should be examined in all cases which are to undergo operation.

This examination may reveal the presence of unsuspected constitutional disease, which will render the operation in its results dangerous to the patient. On the other hand it may guide the surgeon, if possible, to delay the operation until by treatment the patient is in a more fit condition.

If albumen or sugar be found in the urine not only will the operation be more dangerous on this account, but the subsequent repair of the tissues will be delayed. The presence of sugar in the urine is always of serious importance to the surgeon and raises the question as to whether it is advisable to operate at all.

In any case it makes it imperative to investigate the history of the patient previous to the incidence of the malady for which operation is required.

If the surgical condition has arisen in a person suffering from diabetes, the surgeon will only advise operation when the case is urgent and necessary to the saving of life, such as strangulated hernia, dangerous haemorrhage or in injuries.
If, however, he is satisfied that the sugar has appeared in the urine subsequent to the surgical lesion his attitude will be considerably modified, and he will agree to operations of expediency such as radical cure of hernia, varicose veins, etc., and he will be fairly justified in expecting a favourable result. The fact of sugar in the urine, however, will make the surgeon specially particular that everything is done with strict antiseptic precautions. He will make the patient's skin as surgically clean as possible be most careful with his instruments, his hands and those of his assistants. The hygiene of the patient's surroundings must also be looked into.

The operation done will be the one involving least shock. The tissues in diabetes are of low vitality and must be handled with very great care. Incisions should be as small as possible, compatible with efficiency, so as to have little raw surface.

The tissues of diabetes are very susceptible to the invasion of bacteria and if sepsis occurs it spreads very rapidly and is very likely to cause local death and general blood poisoning, and the danger of operation is the possible development of coma. The patient survives the operation and appears to be fairly well but drowsy. This drowsiness increases and coma supervenes and patient dies.
There is also the danger of post anaesthetic acidosis which is not uncommon in these cases.

In the present thesis I propose to discuss the question in its various bearings, and endeavour to throw some light on the subject by giving the results of some personal observations.
The Effect of Diabetes in the Tissues.

Since Willis, more than two centuries ago, marked that the urine in certain cases characterised by polyuria and thirst, tasted sweet, a vast amount of investigation into the causation of diabetes has been made, and many different pathological conditions have been found occurring in this disease. The abnormality in diabetes has been ascribed to many and varied causes, by different observers, but it cannot be said that any single cause has yet been definitely found. It is probable that there is no single cause, that diabetes is not a pathological entity, but rather a group of symptoms which may be produced by different morbid conditions.

Garrod says, "Whilst it is true in some cases, the nervous system is evidently at fault, and that in others the thyroid, the pituitary or the pancreas is the seat of disease, in the great majority of cases which we encounter in practice, we can find no indication of of the underlying lesion on which the metabolic derangement depends. The various tests of pancreatic efficiency give uncertain or negative replies. No ductless gland is evidently the seat of the disease, no intestinal disturbance points to an origin in the alimentary tract and no carotid vascular
changes help us to classify the case. There is to all appearance a sheer wanton metabolic insanity. Nevertheless, we can hardly doubt that in such cases there is an underlying morbid cause at work if only we could find it out. I, for one, do not believe that the chemical processes in the body go awry of themselves."

The influence of heredity cannot be doubted, for one meets with the disease in different members of the same family. It is also a fact that it is met with in husband and wife. Conjugal diabetes has suggested the theory that it is contagious and due to some specific microbe, such has not been found, however, by any observer.

Glycosuria has followed injuries and more especially those involving shock, contusions of the abdomen and thorax, and also severe muscular strain.

Violent mental, emotion, overwork or worry in business or family affairs is the only apparent cause one can very frequently find.

In women the climacteric disturbance is frequently coincident with the onset of the disease, so also do we find it following pregnancy and parturition, but here it is usually ephemeral and passes off with the exciting cause.

Some cases are set up by inflammatory conditions
more especially associated with the formation of pus in some part of the body. It has been found in abdominal abscess following appendicitis also in abscess of the gall bladder.

Dr. Imlach\(^2\) reported a case in 1885 in which the removal of the uterine appendages, the right Fallopian tube being distended with pus, cured a glycosuria of 5 months standing. The patient was prepared for the operation by diet and medicinal treatment. His patient made an excellent recovery and was out of bed on the ninth day.

Various acute infectious diseases, such as erysipelas, enteric fever, diphtheria, influenza, malaria and syphilis has been cited as antecedents of the disease, so also the injection of certain poisonous drugs, such as morphia, curara, alcohol, chloroform, etc.

The morbid conditions found in the organs of the body and in the blood, while they do not enable us to specify a cause for the disease, throw some light on the various sequelae of the disease.

In the brain different lesions have been found, cysts and haemorrhages and softening. Tumours also have been found in the medulla.

Neuritis is a painful and not infrequent incident in diabetes. It may be multiple or may affect one
nerve only. It occurs in elderly diabetics and may cause paralysis. Lesions of the Sympathetic nervous system have been found and notably of the semilunar ganglia.

The heart is frequently sound and free from valvular disease, but occasionally fatty degeneration and hypertrophy is proved. There is a change in the blood. The amount of sugar is increased as is also the amount of fat and the alkalinity is reduced by the presence of various acids in it: \( \text{B} \cdot \text{Oxybutyric} \), diacetic and acetone.

The lungs are frequently the seat of disease, diabetics are prone to be affected by pneumonia, bronchitis, pleurisy and tubercular disease. It not infrequently happens that one of these diseases closes the scene.

The liver is frequently fatty and sometimes cirrhotic but there is no definite or constant change met with in the liver. This may be said of the kidneys. Probably the most important morbid change is found in the pancreas. It is frequently atrophied and sometimes the seat of cystic degeneration. In twenty-four postmortem examinations Williamson found the pancreas diseased in 16.3

Claude Bernard in 1848 by asserting that the source of the sugar circulating in the blood and nourishing
the tissues was the liver, was the first to throw light on the pathology of diabetes. That the liver is a sugar forming gland has been much disputed and been subjected to much, but today it holds the field and is generally accepted.

Bernard discovered that the liver stored carbohydrates in the form of glycogen and he believed that this was converted into sugar by a diastetic ferment in the blood and given out to the tissues according to their needs. He further demonstrated that by puncture of the floor of the fourth ventricle of the brain an excessive production of sugar was caused and that this excess was passed out of the body in the urine. It, therefore, seemed a plausible theory that structural disease in the fourth ventricle was the cause of diabetes, various other nerve lesions have been experimentally made and have been found to cause glycosuria but it is only of a transitory character.

In 1887 Lancereaux made an important contribution to the pathology of the disease when he shewed that frequently in diabetes the pancreas was diseased. Experiments on dogs by Von Mering and Minkowski demonstrated that, after extirpation of the pancreas they suffered from the classical symptoms of diabetes glycosuria polyuria, thirst, hunger and wasting.
On the other hand obstruction of the pancreatic duct by ligature had not this effect which showed that it was some element in the pancreas but not in the juice which it pours into the intestine that affected the sugar metabolism in the body.

It was also found that if a part of the gland was left and it retained its vitality the sugar excretion was not increased.

Diabetes has followed the extirpation of the pancreas in man.

The pancreatic hypothesis of diabetes is a great advance in the pathology of the disease, but it does not cover the whole ground, because we know that in some cases of diabetes, there is no apparent disease of this gland. When disease of the pancreas is present, however, we are justified in concluding that it is the cause, just as we say granular kidney is the cause of the symptoms in Bright's disease.

Saundby says that if the liver possesses in health not only the power of sugar formation but also some mechanism by which it adopts the amount manufactured and passed into the circulation to the current demands of the organism and keeps the percentage of sugar in the blood at a constant point, we have only to assume a disorder of this mechanism or regulating
function to explain the occurrence of diabetes."

Now it is quite conceivable that this regulating mechanism may be put out of fear by injury to the nervous system, by poisons, or by the want of the influence of the pancreatic "element" in the circulation. The presence in the tissues of an excessive amount of sugar, and the changes set up in the tissues thereby, renders them very liable to the invasion of bacteria. We know that diabetics are specially prone to certain forms of skin disease. Not only have we skin disease as the expression of the constitution condition but we may have and very frequently do have local affections caused directly by the irritation of the sugar in the urine. These skin affections in diabetes are usually very persistent and refractory to treatment. The local lesions affect the orifices of the urinary passages; the external genitals and the neighbouring skin. In men you find the meatus a deep red. The prepuce and the glans may become acutely inflamed and this may spread over the penis and scrotum and thighs. A gradual thickening of the prepuce may occur which causes phimosis with attendant difficulty of passing urine. It is in the females that the local lesions are as a rule most severe because of the larger surface affected. It spreads over the whole of the vulva and into the
vagina over the labia majora to the skin adjacent in
the perineum and over the contiguous parts of the thighs.

These conditions cause intense itching. The
scratching breaks the surface and microorganisms gain
entrance and find a very suitable soil.

Diabetics are very liable to boils and carbuncles.
Many of the other forms of skin diseases are found in
diabetes such as eczema, erythema, herpes, lichen,
psoriasis urticaria, acne and while these diseases
have not special characters in diabetes, they are found
when associated with this disease to respond slowly
to treatment. Eczema in diabetes is apt to become
very severe and to have all its characters accentuated.

Then we may have the supervention of gangrene as
the result of a very trifling cause. It may occur
in patches or it may spread over a large surface,
causing serious destruction.

Hyde has described what he calls eczema diabeticorum which affects the genital organs of both sexes,
but more particularly it is found in women. It is
accompanied with severe pruritus and an enormous
tumefaction of the genito urinary canal and sur-
rounding parts.

There is a variety of Xanthoma which occurs in
association with diabetes. The lesions are dis-
tinguished from ordinary Xanthoma by the presence of a raised red area around the yellow spots. It appears first in the flexor aspects of the limbs then in abdomen and lower part of back and may appear in the penis. Its appearance has been the first warning that the patient was suffering from diabetes.

In diabetes the nutrition of all the tissues is profoundly affected and this malnutrition is exhibited in the many and varied lesions connected with the disease. In elderly diabetics we frequently get gangrene of the extremities. This usually starts at some part where there has been pressure or where there has been some slight injury. Sometimes, however, no traumatic origin can be ascribed and this is spoken of as spontaneous gangrene. Usually there are premonitory signs of it in the form of tingling and burning sensations, or it may be that it announces itself by intense paroxysmal pain which the patient can hardly endure.

In these necroses, atheroma of the arteries is perhaps the main cause. This is also usually some degree of neuritis present. This, evidenced by the loss of sensation in the feet. But the main cause is the starvation of the parts caused by the narrowing of the lumen of the vessels and by thrombosis and this degeneration of the vascular system is
largely caused by the diabetic condition. Gangrenous conditions always become septic and the altered conditions of the tissues combined with their low vitality and the presence of septic organisms brings about in a very short time a condition of extreme gravity.

Cataract is frequently met with in elderly diabetics and is also a manifestation of the faulty nutrition — more rarely retinitis is found in this disease.

References.

5. Mervins, Hyde, Diseases of Skin 1900, p. 231.
While sugar is the most prominent and constant abnormal substance found in the urine in diabetes, there are other three substances which may appear in greater or lesser amounts. The appearance of these substances in the urine namely $\text{B.oxybutyric acid}$, acid, diacetic acid and acetone is an indication that the patient is in the very serious condition of acid auto intoxication which causes coma, which condition is fatal. These three substances which have been called the acetone bodies are closely related to one another and are all derived from the same mother substance and merely represent different stages of oxidation. Acetone is a colourless liquid with a characteristic odour which has been likened to that of hay or apples. It is derived from the unstable diacetic acid which in turn is derived from $\text{B.oxybutyric acid}$. The chemical changes involved are as follows:

Boxy butyric acid yields diacetic acid by oxidation and this in turn is converted into acetone by the loss of CO. (3)

$$\begin{align*}
\text{CH}_3\text{CH(OH)CH}_2\text{COOH} &= \text{B.oxybutyric acid.} \\
\text{CH}_3\text{COCH}_2\text{COOH} &= \text{Diacetic acid.} \\
\text{CH}_3\text{COCH}_3 &= \text{Acetone.}
\end{align*}$$

Whenever these bodies therefore appear in the urine we must assume some perversion of oxidation.
A healthy man who is living on a diet of meat and fat will excrete these bodies in small quantities.

The actual mode of operation of these acids seems to be that they become neutralised by the alkali present in the body and that if they are present in excess there is risk that the alkalescence of the organism may be so reduced that it is unable to carry on its functions in an efficient manner. That this is so in animals is proved by the fact that if sodium carbonate is injected acid poisoning does not occur.

These acetone bodies have no specific toxic properties but their deleterious effect is due to the power they possess of drawing alkali from the organism for it is found in coma the alkalescence of the blood is greatly reduced.

In a diabetic person the oxidative processes are perverted and especially is the power of fat katabolism lessened. He is thus always on the brink of acid auto intoxication and a very trivial injury, a fatiguing journey, violent emotion or the incidence of any inflammatory disease may cause a lethal amount of acids to accumulate in his blood and so precipitate coma. It is unfortunate for the surgical patient suffering from diabetes that this tendency to accumulate acid in his body is increased by the
necessity for giving him an anaesthetic. The condition which may be set up by the anaesthetic has been given many different names such as "delayed Chloroform Poisoning," "Acetonuria," "Acidosis," "Aciduria," "Acetonaemia," or acid poisoning. We will discuss this danger of post anaesthetic acidosis in some detail. It is characterised by the presence of acetone in the urine and it may be detected by the smell of acetone in the breath.

Peters in 1857 first recognised acetone as a pathological constituent of urine and he found it present in a case of diabetes.

Kaulish in the same year reported a series of cases of diabetes in which he found it present in the urine and attributed the onset of coma to acetonæmia.

Kussmaul in 1874 disputed this theory and later Gerhardt showed that acetonuria was associated with the presence of diacetic acid and B. oxybutyric acid in the urine of diabetic patients and that probably they were responsible for the onset of coma.

Acetonuria has been found not only in diabetes and after general anaesthesia but also in fevers, broncho pneumonia, starvation, pernicious vomiting of pregnancy, Phosphorous poisoning, sepsis, chronic morphinism, shock due to injury and mountain sickness.
It has also been produced experimentally by the administration of Phloridzen and by extirpation of the pancreas. In many of these conditions, however, toxic symptoms are absent.

In 1904 Brockett, Stone and Low definitely proved that the symptoms of delayed chloroform poisoning were due to an acid intoxication.

Of course the great majority of patients recover from general anaesthesia with no symptoms at all of acidosis, in a few the symptoms are obvious and urgent and in a small minority a fatal issue results.

The symptoms of delayed chloroform poisoning may not come on for 12 hours even for 2 or three days. The patient may begin suddenly to vomit and this vomiting is usually copious, frequent and intractable. He is extremely restless and may become delirious. His pupils are dilated. The delirium subsides sometimes quickly and the patient becomes unconscious and gradually comatose. The characteristic smell of acetone will pervade the room of the patient when in this condition.

As coma supervenes the face of the patient becomes dusky. The respiration, sighing and irregular. Dyspnœa and air hunger are also sometimes present.

In 1894 Guthrie published a paper entitled "Some Fatal after Effects of Chloroform in Children." He
recorded nine fatal cases and his description of the symptoms and of the pathological changes found after death resemble those described by continental observers. His conclusions were not accepted and death was attributed to such causes as carbolic poisoning and fatty embolism.

In 1903 he published a second paper entitled "The Fatal Effects of Chloroform in Children Suffering from a Peculiar Condition of fatty liver." He described four cases as the cause of death which he attributed to the action of chloroform on a morbidly fatty liver, such a condition as we may readily have in diabetes.

Since then it has been shown, that not alone the administration of chloroform is the cause but it may occur after ether or even after ethyl chloride.

Post mortem findings in cases of post anaesthetic acidosis reveals a state of intense fatty degeneration and this is markedly so in the liver. It has been often found to be fawn coloured and in some cases it has been described as canary yellow.

The kidneys also are in a state of fatty degeneration. The heart is usually pale and the muscle fibres shew fatty degeneration. In 1904 Stiles and McDonald excised pieces of liver under ether, some days later they gave chloroform and excised more liver. Sections of the first portion shewed little fat.
Sections of the second portion were markedly fatty. They also showed that fatty changes which appeared some hours after chloroform did not disappear for some days or weeks. Thus it is seen that chloroform and to a less extent ether are capable of producing fatty changes.

We have seen that acetone is derived from diacetic acid which is in turn derived from glyoxybutyric acid. These are fatty acids. From what are they derived? From carbohydrate, Proteid or Fat.

In diabetes it has been noticed that the sudden withdrawal of carbohydrate has been followed by symptoms of coma and a marked increase of the ferric chloride reaction in the urine and by resuming carbohydrate food these symptoms diminish or disappear. It is thus obvious that carbohydrate is not the source. It was long held that Proteid was the source. It has since been shown that this is extremely improbable because their excretion is not accompanied by a corresponding increase in the excretion of nitrogen and sulphur.

Since acetone is derived from a fatty acid, fat is the most probable source. Further, by giving a diet of fat alone you can produce acetonuria and cause the fatty acids to appear in the urine in great quantities.
In diabetes the patient has diminished power of carrying out the process of fat Katabolism, the seat of which is in the liver and in diabetes you find the liver is always richest in fat and in those cases where death has followed an acute acid toxaemia, the liver has always been found markedly fatty. It is because then of the inadequacy of the liver to deal with fat that the anaesthetic acts the part of the "last straw" and brings about the serious condition of acidosis.

The determining factor in delayed chloroform poisoning is not the duration of the anaesthetic but the inadequacy of the metabolic processes before the operation. For the result of a drug is the reaction it produces between itself and the organism and just as you may alter the result by changing the drug, so you may alter the result by changing the organism.

Treatment before Operation.

We must prevent as far as possible the formation of these fatty acids. Since it is probable that fat is the substance from which the acetone bodies are formed it would seem to be a logical proceeding to reduce the amount of fat in the food of the diabetic. But fat is of paramount importance in maintaining the nutrition of the diabetic. It is as Von Noorden has said the 'sheet anchor' in all
severe cases, and he says we are not justified in excluding fats from the diet. He exemplifies the fact that if butter is thoroughly well washed in cold water, the lower fatty acids are removed and it is they that exercise the greatest influence in the acetonuria. If this is done he thinks as much butter may be given as the body needs for its nutrition.

The administration of carbohydrates to a diabetic person in ordinary circumstances is what we are careful to avoid because we know that it increases the glycosuria but when acidosis is present it is the best and most efficient means at our disposal for combating the condition.

It has been suggested that large doses of glucose should be given Another and efficient means at our disposal is the administration of alkalies in order to disintoxicate the poisonous acids and prevent their accumulation in the blood. The alkalies administered combine with the poisonous acids and so prevent these acids combining with the fixed alkali bases of the body and they thus act as disintoxicants. Then the compound formed between the medicinal alkali and the poisonous acid is more readily eliminated by the kidneys than is the free acid itself. In a sense, the alkali acts as scavenger to the body by keeping it rid of the poison.
In any case of diabetes with a positive ferric chloride reaction, which indicates the presence of acidosis, bicarbonate of soda should be given in doses 3 or 4 times a day. This may be the means of averting coma. If by this means we can render the urine alkaline we know we are giving sufficient alkali. It may not of course be found that the patient's stomach will stand enough alkali to make the urine alkaline.

Before operating on a diabetic person it is best if the surgical condition is not urgent to treat the patient for some time with alkali, and also to give him a course of glucose.

It is also of prime importance to make sure his bowels are being regularly cleared out, for nothing is so striking in the post mortem findings in coma as the colon full of "hardened impacted faeces." If coma itself is present or signs of it, there is no time to delay in treatment. The excretions should be stimulated, the stomach should be washed out with alkaline solution 3 i sod bic. to the pint. It should also be injected subcutaneously In coma, however, it is not as a rule found the improvement effected by treatment on the above lines is other than temporary. The old proverb that "Prevention is better than cure" is abundantly true in connection with this condition and so all the
available means at our disposal should be used in order to prevent this most fatal condition arising for it is the sleep of death.

In the gangrene which occurs in diabetes one of the chief factors at work is the arterio-sclerosis which is nearly always present. In senile gangrene arterio-sclerosis is always present.

Smith and Durham who have collected 63 cases and analysed them found arterio-sclerosis mentioned as being present in 24, it was present in each of my cases another characteristic is that the gangrene is found in elderly patients and it is them that sclerosis of the vessels would be most likely to be found. S. and D. found the majority of their cases between 50 to 70 years.

The gangrene in most cases has an inflammatory beginning. The tissues are being nourished by barley by the abnormal blood and are a ready soil for the septic organism. Usually a history of a slight traumatism is to be got, it may merely be a tight boot, long standing or walking causing blisters on feet, or a blow causing slight abrasion, or as in one of my cases the suppuration of a hand corn. The conclusions they come to in regard to operation may be summarised thus:

1. Care in the antiseptic routine.
2. The site of amputation must avoid including infected tissues in the stump and also to make sure the arterial supply to the stings will be sufficient.

3. Avoid osteoplastic operations such as Pirogoffs, or Stokes-Gritti.

4. The flesh ought to be treated with extraordinary delicacy.

In inflammatory conditions about the feet in diabetics it is

1. Of great importance to rest the part completely and to elevate the limb.

2. To keep the part dry and warm and well dusted with iodiform and boracic acid powder and covered with cotton wool.

3. To regulate the diet and pay strict attention to the regular movement of the bowels.

4. To give the drugs the best of which is morphia.
Dangers of Surgical Treatment in Diabetes.

1. Danger due to development of Sepsis.

In the preantiseptic era of surgery the results of surgical operations in diabetes were so invariably bad that the presence of sugar in the urine was considered a barrier to operation.

This non possumus attitude is not now warranted for even operations of expediency are now performed with favourable results.

The antiseptic and aseptic methods have shown the operation in diabetes of much of its danger so that no clan of surgical patient owes more to the modern system of surgical procedure than the diabetic.

The liability of the tissues to sepsis is specially strong in diabetes, being of low vitality. The micro-organism finds in them a suitable nidus. Diabetic tissues form a poor soil for healing processes and this is brought about by various causes.

Kléen says that the excessive quantity of sugar circulating in the blood prevents the blood giving up to the tissues its water with the result that there is dryness or dessication of the tissues which affects their nutrition and functional power.

In addition to hyperglycaemia there are acid toxins circulating in the blood chiefly B. Butyric acid, diacetic acid and free fatty acids, and these also
impair the vitality and reparative power of the organism as well as constitute in themselves a grave danger which the physician no less than the surgeon must never ignore in his management of a case of diabetes.

The blood vessels are very prone to atheromatous disease and this is very notable in the smaller vessels which condition further tends to malnutrition.

Neuritis is a very common complaint in diabetes. It may only affect one more or it may be multiple. When the nervous influences on the tissues are cut off or impaired it is well known that there is a great tendency to suppuration to necrosis and this is well exemplified in gangrene and perforating ulcer of the foot which is frequently found in diabetic subjects.

These conditions of the blood, the blood vessels and the nerves as well as the commonly weakened action of the heart are the various causes of the vulnerability of the tissues to septic injection and their poor healing activity.

In addition the diabetic is often of neurotic temperament and not infrequently adds alcoholism to his other defects, also the diabetic who comes under the care of the surgeon is most frequently handicapped by age.

It thus happens that even a slight wound may easily
become a serious matter for the diabetic and it is not often that operation wounds heal by first intention. Some surgeons have stated that if after an operation the wound for no apparent cause becomes septic or  sloughs the urine should be examined for sugar. It should be remembered in this connection that sometimes sepsis may cause glycosuria which is most usually temporary and disappears on the recovery from septic state. Cases of carbuncle and cellulitis and many other suppurative processes exemplify this. The following cases are instances which have occurred in my practice.
Case 1. George M. 52, Grocer. A heavy flabby man addicted to over-indulgence in liquor was seen on 26th May 1909. On the nape of the neck there was a carbuncle about the diameter of a tea cup. There were numerous "points" some of which had burst and were pus, and the base and surrounding tissues were hard and deeply inflamed. The patient was suffering much pain and was not able to obtain sleep. His temperature was 103-6 and pulse 100, regular and full. Tongue was dry and red. He had intense thirst and was passing about 5 pints of water in the 24 hours. Bowels constipated. The urine was acid. Sp. 1028 and contained sugar, no albumen. The patient was given 5 grains Calomel followed up by Seidlitzs powder and had bowels well moved.

The carbuncle was cleansed with Corrosive sublimate solution and dressed with boracic poultices, 2 grains quin 25 grs. Dover' every 4 hours. He was fed on milk, eggs and beef tea and given of whiskey every 4 hours.

The carbuncle disintegrated and much slough came away in the course of the next 7 days. The temperature was normal on the sixth day and the patient's condition from being a grave one was now much improved. The sugar was found in the urine up to the 10th day.
The healing was not complete for six weeks. Since then although I have examined the urine several times there is no glycosuria.

Robert, M. aged 30, a woodman, always been healthy, felt an itching on the under side of the scrotum which he attributed to the friction of a new pair of trousers. There was nothing to be seen but what he called a hot spot. I saw him 4 days after he first felt the itching, January 10th 1912 and by this time he was very flushed in the face, thirsty and had a temperature of 104°. His pulse was 120. The scrotum was swollen and deeply red, the integuments of the penis were swollen and oedematosus, the whole presenting the appearance of erysipelas of a phlegmas character. The skin on the under part of scrotum sloughed and on the under side of the penis.

The bowels were constipated.

The urine was acid Sp. g. 1026, no albumen, sugar was present in considerable quantity on the fourth day of his illness and I was able to detect it for 7 days afterwards. It got less each day and had disappeared by the time the feverish state had passed off and healing had commenced. The man is perfectly well now but complains of nervousness. The healing of the wounds was slow.

Case 2.

Mrs. G.W., 61, wife of an innkeeper, stout and
florid, a temperate very busy woman was first seen in June 1911. "She has been dressing a sore place on the sole of the foot for several weeks with boric ointment but it was not healing." On examination I found a round, pinched out hole with thickened cuticle surrounding it. It was tender and there was slight discharge from it. On examining the urine it was found to be acid Sp. g. 1030, sugar and trace of albumen present. She was passing 6 pts in 24 hours and did not complain much of thirst. She had lost considerably in weight and was feeling her work "a burden." The ulcer was situated beneath the metatarso phalangeal joint of the little toe. The part was dressed antiseptically and patient laid up and diet regulated and she was given opium. The surrounding inflammation abated so also the discharge and a piece of necrosed bone was removed. There is now no inflammation nor discharge and it is practically healed. There is still sugar in urine. The knee jerks are absent. This patient has lived a very active life she has suffered much from varicose veins, she is of a sanguine temperament and does not worry much over any thing. Restriction to strict diet reduces the sugar in the urine but does not remove it.

Case 4. W.C. Farmer, aged 52 consulted me first in 1896. He looked well and strong and was a very active man.
For many years he had in addition to his own farm, been manager of other two farms. He had had a considerable amount of "financial" strain he complained of excessive thirst and was passing a large quantity of water and was losing flesh. He told me he believed he had diabetes as the previous year on being examined for life insurance he had been rejected and was then told it was because he had diabetes. His urine was clear, acid, no albumen, Sp. G. 1030 and contained 3 grains of sugar in an ounce. He passed 8 pints in 24 hours. He was advised about diet and gave him a pill containing $\frac{1}{2}$ grain of opium and $\frac{1}{2}$ ext. belladonna. One to be taken after breakfast and one in the evening. He kept fairly well and was able to do his work during the next 3 years. In 1899 he began to complain of pains in the feet and legs below the knees and also about the angles of the scapula. These pains disturbed him at night and kept him awake. He was also less able to walk about, but continued to ride. His knee reflexed was present but not so good as when I first saw him.

In December of 1899 I was called to him as one of his toes on the left foot had turned 'black.' I found the toe gangrenous and laid him up and dressed his foot with iodoform gangue and kept it warm. He was carefully dieted. The toe became quite mortified and was removed. During the next 6 months the same
thing occurred to all the toes on that foot except the big toe but the disease did not spread into the foot. In June 1900 the big toe of the right foot became gangrenous and the disease spread into the foot and involved the metatarsal bones and joints.

Consent was given to remove the foot. He refused to have an amputation of it should mean losing any part of the leg. There was pulsation in the anterior and posterior tibial arteries and the tissues about the ankles were in fair condition so a Symes amputation was performed. The foot had been carefully dressed antiseptically for a considerable time and did not need any special preparation before operating. The operation was done in the patient's bedroom a colleague of mine gave the anaesthetic which was chloroform and I did the amputation. It was the first occasion in which I had done this operation and it occupied considerable time. The patient stood it well and did not lose much blood. The stump did not heal by first intention it discharged a good deal and a very small margin of skin at one part of the flap became gangrenous. This did not, however, spread. It was all healed up in two months and he could bear a little weight on the stump. I got a boot for him and he could walk about his farm and even ride on horseback. His general condition im-
proved and he began to resume a good deal of his former work.

In 1904 in returning from a lamb sale he felt out of sorts. He had not been careful with his diet for some time and this day he had had a good deal of fatigue and a railway journey. I saw him next day. He was in bed and had an exhausted look. His hands and arms were swollen and brawny and of a deep red colour. He did not complain of any pain but was drowsy. He gradually sank into coma and died. The smell of acetone in the breath was noted. This man belonged to the fair ruddy type and he had lived an active life. He had had a great deal of worry of a financial kind. The duration of the disease was known to have been nine years. It was risky amputating at the ankles instead of the seat of election, but he got a very good stump and certainly a much more serviceable leg than if it had been removed higher up.

He recovered from the anaesthetic with nothing more than the usual disturbance one finds in a normal person. Some months after the above patient's death his widow Mrs. I. C. aged 62, consulted me about an "itching in the forebody." I found she was passing urine in excessive quantity and on examining it found its Sp. G. to be 1028 and containing both sugar and a trace of albumen.
The external genitals were swollen and red and bore evidence of scratching. She had lost weight very much in the last year, she was dieted and given opium pills and a daily purge of saepctile of soda. She was advised to bathe the parts each time after micturition with carbolic lotion and to apply ointment at night. She lived 37 years after her husband, gradually failing in strength. She died in a comatose state and her urine gave the acetone reaction with ferric chloride before death. This is the only instance of con-
geguae diabetes that has occurred in my practice. She had a very trying time during her husband's long illness and after his death she had a great deal of worry in connection with farming and financial affairs. It is interesting to glance at the history of their descendants, 3 sons and 3 daughters reached adult life. The eldest son is obese and dull and easy going. He is the father of 5 children living and of 3 dead. The surviving children are quite healthy. One child died from hydrocephalus one was an anencephalic monster and one was prematurely born. The second son has a family of 4 children living, 2 dead. The eldest, a boy, is crippled from an attack of poliomyelitis and the youngest, a girl, has cleft palate and harelip. The other two a boy and girl are quite healthy.
The two dead were twins, and they died from icterus neonatorum. It is very interesting to note that all the children of this family have suffered from icterus at birth and the severity of the disease has increased with each successive pregnancy. The third son died of enteric fever.

The eldest daughter has 3 healthy children. The second daughter has been married 10 years, suffers from neurasthenia and has no children. The third daughter has been married 8 years, suffers from "Rheumatism" and has no children.

Case 5

Mrs. B. aged 64. Had been in poor health for some years. She said she had not been well for 6 years and thought the cause was anxiety and over-work during her husband's illness which ended fatally. She had for some time noticed she was drinking a large quantity of water. She had developed cataract in the right eye and some months before I saw her pains in the feet had come on and she was troubled with cramp in the legs. When I was called to her in August 1905 she had a painful ulcer between the fourth and fifth toes in the right foot, it was deep and coated with black slough and around it there was a good deal of inflammation. The feet and legs felt cold and were thin and wasted. The knee jerks could not be elicited. The urine was acid Sp. G. 1023 contained sugar and a
trace of albumen and the quantity was greatly increased. She was dieted and given 5 minims of Sig. Morph. Hydroch. 3 times a day and the foot dressed with iodoform gange and kept warm.

The disease spread, all of the toes became gangrenous also the anterior half of the foot. There was also a gangrenous patch on the heel. The patient was suffering a great deal of pain and was desirous of having the foot removed. Her arteries were hard and pulsation could not be felt in the foot.

Patient was put under chloroform and the leg amputated at the seat of election. It was then seen that the arteries were very atheromatous and almost occluded. Patient stood operation well but on the third day there was some brownish discharge from the wound and the flaps were deep red in colour. Next day the flaps were bluish and the discharge was profuse and smelling.

Patient did not complain of pain but lost strength from day to day and on the 10th day after the operation she became comatose and died.

In this case there was no doubt a long standing diabetes. The disease evidently following the strain of nursing her husband and in addition working for the maintenance of her family. The atheroma of the vessels and the neuritis present made the case a
not very hopeful one, but the condition was rather hopeless and the operation offered at least a chance. It saved the patient, I think, a good deal of suffering and she of course was made aware of the risks and accepted them. She also suffered from pruritus.

Case 16.

Wm. T. Gardener, aged 70. Had always been a healthy man and lived regular life. When I saw him his complaint was a pestering corn in the little toe of the left foot. He had had the corn for many years but it had only begun to pain him very much a few days before I saw him, which was on 7th June 1909. On this day the toe was swollen and the skin in the dorsum of the foot was tender and red and a serious discharge was exuding from the seat of the corn. Next day the foot and leg were much swollen and tender to touch. The knee reflexes were present. The pulse was 80 and the temp. 99°.

The urine was acid, no albumen, 5 grains sugar per ounce, Sp. G. 1020, quantity 4 pints.

The sore was dusted with iodoform and the whole foot wrapped in cotton wool. Patient was given Pil opii gr. ½ Belladonna gr. ½ l.d.s.

The little toe became black over the distal phalange and loosened and separated five weeks from the commencement of symptoms. The sugar had varied in amount and the other symptoms had greatly benefited by the
rest in bed and diabetic treatment. Eventually the wound granulated and healed.

I was inclined to think this was a glycosuria set up by the septic condition of the foot but his subsequent history shows that the glycosuria still continues reduced in amount and so far there has been no more gangrenous symptoms I have been informed. But he has become blind from cataract.

Mrs. E. H. aged 50. Family history. Mother died of heart disease at 54 years, father died of asthma at 49 years, two sisters died about 50 years of age of heart disease.

She is the mother of six children all alive. Had once suffered from gall stone colic.

On the 28th January she went to bed after having a light supper feeling quite well. She awoke in the middle of the night with severe pain in right hypochondrium reaching up to shoulder on same side and across the abdomen. She vomited bilious fluid at intervals the retching being severe.

When I saw her on the morning of the 29th January the pain had abated there was rigidity over right side of abdomen and tenderness over gall bladder. Temperature was 97.8 pulse 80, regular but weak. She had an exhausted look. Her bowels had been moved by a dose of cascara taken the previous evening.
The urine was high coloured and contained urates and was scanty. On the following day vomiting had ceased, temperature 97.8 and pulse 80 and regular. She had been perspiring during the night. Urine had a deposit of urates and was high coloured. There was a decided tenderness on right side as low down as level of umbilicus.

Next day conditions unchanged except that a decided lump could be felt which was diagnosed as a distended gall bladder. Operation was decided upon. Next day, patient was conveyed by motor car to a private hospital in Newcastle on Tyne a distance of 50 miles. She stood this journey well. Just before the operation her urine was examined and was found free from albumen but containing sugar. There had been at no time previously any signs of diabetes about the patient. No thirst, no polyuria, no wasting. She was anaesthetised with chloroform and kept under with ether. A traumatic incision was made exposing the gall bladder and the right lobe of the liver.

The gall bladder was greatly disturbed and was darkened in colour and at the fundus showed indication of commencing gangrene. It was incised and the contents which were viscid and glairy looking drained off. Calculi were found one of which was impacted in the cystic duct.
The liver was distinctly cirrhotic. The Surgeon, Professor Rutherford Morison on seeing it at once asked if this lady was alcoholic. I was able to say with certainty that she was not. The pancreas was not examined. The wound healed well and in four weeks time she was able to go about. The sugar disappeared from the urine while she was in hospital, but it has reappeared since coming home and was first noticed after an acute period of anxiety concerning a son who was very ill. She is passing about 4 pints in 24 hours.

Her urine has never exceeded Sp. G. 1020 is acid, clear, contains no albumen but it contains sugar. There is no polyuria nor thirst. The bowels are constipated.

The points in this case are. Sugar first found after pus formation in gall bladder. After the emotion of the imminence of operation which was considerable. The disappearance and the reappearance after anxiety about son.

The frequency of gall stones in diabetics. The cirrhosis of liver. Will there be cirrhosis of pancreas also?

It should also be noted that this lady has been passing through the disturbance of climacteric which is itself said to be a cause of diabetes.
I have noticed that her hair has very rapidly become grey this last year and now it is quite white.

The glycosuria has no effect whatever in the healing of the wound which was perfect, leaving a beautiful scar. She had no ill effects from the anaesthetic.

Case 8.

Miss H. G. Aged 49, was seen first in December 1909. She was then suffering from menorrhagia and was very anaemic and had swelling of feet and legs. Urine was pUle and contained a trace of albumen but no sugar. She was given an iron tonic and advised about diet, etc. I did not see her again for a year when she complained of swelling over lower part of abdomen. On examining I found a large round swelling just above the pubis and passing down into the pelvis. The bleeding was very frequent and copious and anaemia very pronounced.

A uterine fibroid was diagnosed and operation advised. The urine was examined and was found to be acid, Sp. G. 1015, trace of albumen and sugar was present.

On Xmas day 1910 she was given chloroform and the abdomen opened by mesial incision. The tumour was quite the size of a child's head and a cyst of the right ovary about the size of a duck's egg was found. The tumour, uterus and appendages were removed. The tumour was undergoing mucoid degeneration which accounted for its rapid increase in size. The wound healed by first intention and a good scar resulted.
After the operation the patient slept for some hours and appeared to be going on normally the first day. Twelve hours after the operation she became very restless and commenced to vomit and complained of pain in the epigastrium. The vomiting was very frequent and very distressing and continued for 3 days. During this time she was at times quite oblivious of the surroundings. Her pulse was weak and quick. Her urine gave the ferric chloride reaction and the room quite distinctly smelt of it.

On the 4th day the vomiting ceased and the patient felt better, was quite conscious and was given milk and barley water and some tea. Her urine examined on the 10th day contained no sugar nor has it appeared on any occasion since, although a regular series of examinations has taken place. On January 9th of this year I saw her. She told me she was "better than she had ever felt," and her appearance certainly justified her.

This case interested me because it is the only one of post anaesthetic acidosis I have had at least the only severe one. It is also interesting because of the glycosuria. The glycosuria came on with the increase of bulk in the tumour and it entirely disappeared after the tumour was removed. Although the retching was severe the scar obtained was a good one and there was no delay in the healing.
Case 9. Mrs. J. R. aged 42, seen first on 12th October 1911. She was thin, pale and cachetic looking. In the right breast there was a typical schirrus cancer and the glands in the auricle were enlarged. She had first noticed the swelling 6 months before but had not been in good health for a year. She complained of thirst, her tongue was dry and red and she had also noticed an increase in the quantity of urine. There was evidence of consolidation in the right apex of lung. Heart sounds were weak but no murmurs were heard. She was very constipated. Her urine amounted to 6 pints in 24 hours, Sp. G. 1026, acid, no albumen, 4 grs. sugar

I advised operation and she went to Newcastle Infirmary for this purpose. After dieting and rest in bed for a week she had the breast and glands removed. She never rallied from the operation but sank and died in coma about 10 hrs. after.

Case 10. T.P. 26, single man, farm labourer. Had severe attack of pleurisy four years ago from which time his mother told me he had not been strong. When I saw him on 15th November 1911 he was suffering severe pain on the left side of head and the mastoid region was tender on pressure, temperature 103, pulse 120. He was rather deaf. There was no discharge from the ear nor history of discharge.
On 15th pain was severe, he had been delirious during the night, very thirsty and had passed large quantity of water. Pressure over the jugular vein elicited pain, temperature 104, pulse very rapid. Pupils were equal and contracted to light and accommodation. Diagnosis of mastoid abscess with probable thrombosis of lethal sinus was made and operation was advised. His urine was examined and found to contain sugar and trace of albumen.

He was operated on at home in his bedroom and first the jugular vein was dissected out and tied, though it was found to contain fluid blood.

The mastoid was cut down on and opened by means of chisel. The bone was very hard. A quantity of greenish, foul smelling pus escaped. The part was dressed with iodoform gauze and the flaps brought together. The patient stood the operation well and next day seemed much better, temperature 100, pulse 86. On the third day he complained of pain in the right thoracic region and he had a cough. Temperature 101 pulse 90. His urine on this day contained sugar. He had the typical signs of pneumonia from which he gradually sank and died comatose on the 6th day of onset. His urine reduced Fehling's solution to the end. The mastoid wound showed no sign of healing and discharged freely. This patient died, I think, from septic absorption and I also think the glycosuria
was the result of sepsis for this man had none of the symptoms of diabetes previous to the mastoid suppuration. He had been as his mother said a delicate man but always able to take a fair share in the work of the farm. Otitis media is said to be common in diabetics, the infection spreading up from the naso pharynx which is in a state of chronic catarrh. Its onset is sudden. The pain of great intensity and persistence even although the discharge may be free from the external meatus. The discharge is often bloody and there is a great tendency to mastoiditis and necrosis. (Eulenstein) quoted by Phillips, Lancet, May 17th 1902, p. 1390.
In diabetic cataract the result of operation is usually good. There is no greater tendency of inflammation of the orbital contents, if antiseptic precautions are taken, than in cases of cataract that are not diabetic in origin. The following is the only case that has occurred in my experience where the result was not quite satisfactory.

Mrs. S. aged 70 was in fair health for her age and completely blind from cataract in both eyes.

Her urine contained sugar though the amount was small. On 12th January of this year Professor Wardale of Newcastle-on-Tyne operated on the left eye. The anaesthetic used was eucaine and an iridectomy was performed as well as extraction of the lens. The patient was not excited in any way and stood the operation well. Twelve hours after the operation she became very sick and vomited a great deal, first the vomit was gastric juice and bile, it was accompanied with a great deal of retching and pain in the epigastrium. The vomiting continued for 26 hours and latterly was coffee coloured and contained ascered blood. The urine was rather scanty and gave the ferric chloride reaction and had the characteristic smell of acetone. She was given carbonate of soda 3/ to the pint ad lib to drink and she drank it freely. Her bowels were made to act freely by saline and she had oatmeal
gruel made with milk. A mustard plaster over the epigastrium relieved the pain and seemed to help the vomiting. She made a tardy recovery and was not able to leave her bed for 3 weeks. The operation wound healed quite normally and despite the retching the operation was quite successful in giving the old lady sight.

This was a case of acid auto-intoxication. Was it caused by the local anaesthetic eucaine? or by the diabetic condition? I think there is no doubt that but for the operation the toxic symptoms would not have arisen. They were serious and but for the free administration of alkali and carbohydrate in the form of oatmeal would positively have ended in coma. For diabetic acidosis Von Noorden advocates the free use of oatmeal gruel.
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