Numerous papers are published about diabetes, but for years on end important aspects of it are rarely mentioned. It is widely believed that all diabetics should be on a diet, should regularly test their urine, should attend a diabetic clinic, and that initially they should be admitted to be 'stabilised' (at least if they are thought to need insulin). My object in this article is to examine some of these activities in an attempt to distinguish those based on evidence from those based on theory, the value of which is unproven.

**DIET**

No treatment rests on a more secure basis than does dietary restriction for the obese, maturity onset diabetic. If such a patient reduces her carbohydrate intake from about 400 to 100 g daily, her glycosuria and associated symptoms will usually disappear. She will also lose weight, which is desirable, and if she achieves and maintains normal weight even her glucose tolerance test may return to normal; so by all ordinary tests she ceases to be a diabetic.

Most obese diabetics fail to diet strictly. No doubt they stop taking sugar in tea and eating sweets—for everyone knows that sugar is poison to diabetics—and they may cut down on white bread. But they carry on eating 'starch reduced' or brown bread, ryvita, or dry biscuits far in excess of the recommended 100 g carbohydrate or so daily. No amount of urging by their doctor will persuade them to restrict their diet indefinitely. The problem of what is to be done with such people is almost ignored in most textbooks.

The patients themselves often plead for tablets, or even injections, instead of diet. Many have been prescribed anorectic drugs, which may temporarily help them to follow their diet, but in the long run there is no evidence that such drugs are effective, and many are addictive. The biguanide drugs are widely recommended, since they are said to diminish appetite as well as to reduce glycosuria. Perhaps they improve the situation sometimes, but they do not reduce most obese diabetics to normal weight. The sulphonylurea drugs diminish glycosuria, but if taken as a substitute for dietary restriction, cause the obese to become more obese. If insulin is given there is likely to be a spectacular increase in weight.
When obese diabetics refuse to diet properly, is it best to let them continue with a persistent heavy glycosuria that at least rids them of part of their excess food intake? Subsequent pruritus vulvae may encourage a more strict adherence to diet. The theoretical objection to this plan is that the persistent hyperglycaemia will make degenerative complications more likely. But there appears to be no convincing evidence that this is so. It seems, therefore, that less obesity with glycosuria is probably preferable to more obesity without glycosuria.

Another approach to this problem is to find means of persuading patients to restrict their diet permanently. The most promising method seems to be the self-help clubs, such as the Weight Watchers. Many people, both diabetics and non-diabetics, who have failed to follow dietary advice given by doctors or dieticians, have been persuaded by the example and encouragement of fellow sufferers to get their weight down and keep it down, and to abolish their glycosuria. Unfortunately, many of the most obese will not, or say they cannot, afford to join these organisations.

In insulin-dependent diabetics the need for strict dieting is by no means so obvious and whatever the dietary advice given many such diabetics eat an almost unrestricted diet. Tunbridge and Wetherill (1970) found that less than one-third of the patients attending their diabetic clinic were keeping within 10 per cent of their prescribed diet—a similar proportion to a previous survey in 1948. There was no apparent relationship between the strictness of diet and the degree of diabetic control. Provided that diabetics on insulin or on sulphonylurea compounds avoid hypoglycaemia, obesity, pruritus, and thirst, why should attempts be made to make them diet more strictly? One possible answer is that, although feeling well in spite of persistent heavy glycosuria, they may feel better with less sugar in the urine. This suggests that the patient’s symptoms alone are an insufficient guide, but it does not prove that precise diets laying down the exact quantity of carbohydrates at each meal should be advocated. Many diabetics who do not consciously keep their carbohydrate intake constant have a high proportion of sugar-free urine tests. No doubt many of them do have a steady carbohydrate intake, as do many of the ordinary middle-aged population who lead well-regulated lives. If, then, a diabetic on insulin shows sugar in, say, half or less of his urine tests, is not obese, and has a steady weight, has few or no hypoglycaemic attacks, and feels well without strict dieting, the only justification for chiding him is that the continuance of lax dieting will lead to degenerative complications.

There has been an endless debate about the relationship of strict dieting, good control, and degenerative complications. The most important factor behind such complications is the duration of the diabetes. The complications
do not occur in the early years after diabetes develops in the young, but few who have had diabetes for over 25 years are free of them all. There is no consistent relationship between poor control and early complications, so the most that can be claimed of good control is that it minimises complications, but even this has not been proved. No controlled series, similar in all respects except in degree of control, have been studied over many years. Conclusions have been reached by comparing groups of patients who may have differed in many ways.

If a diabetic on insulin takes unaccustomed exercise he will be liable to hypoglycaemia. He should, therefore, take extra carbohydrate before or during the exercise. But how much extra carbohydrate should be advised? The patient can only be told to learn from experience. This problem is given little attention in the textbooks. Joslin et al. (1959) cites Richardson, the diabetic tennis champion, who 'said he took 10 five-gram lumps of sugar in 2½ hours of strenuous play and more or less according to whether he played singles or doubles'. According to Oakley et al. (1968) 'most diabetics soon learn how much extra carbohydrate they need to take before or during games or gardening. It may be none or it may be 60 or 70 g'.

The advocate of strict dieting gives diet sheets prescribing so many grammes of carbohydrate at each meal, perhaps along with scales and measures, yet he has to allow quite inexact variations of this intake according to exercise. One more complication is the varying composition of foods. According to Joslin et al. (1959) 30 g of bread contains from 15 to 18 g of carbohydrate. And most made-up dishes vary more than this.

The simplest dietary advice for the diabetic starting insulin is along the following lines: carry on eating as usual, but never miss or be late for meals. Keep the amount of starchy or sweet food at each meal steady (except when about to take extra exercise). Always have a snack mid-morning, mid-afternoon and before bed, in addition to the three main meals. If, after some months the patient's weight is steady and he is not obese, he feels well and has no troublesome insulin reactions, and if about half his urine tests are sugar-free, this regime can continue. If he is becoming too fat, having heavy glycosuria, or, perhaps, if his insulin requirements are steadily rising, he should be urged to restrict his diet. Whatever disagreements there are about the treatment of diabetics, one thing is certain, the treatment must be tailored to the individual.

**STABILISATION**

When diabetics are first started on insulin, their insulin requirements frequently fall after the initial phase. According to Oakley et al. (1968):
‘When the patient begins to pass sugar-free specimens there is often a dramatic increase in the responsiveness to insulin therapy . . . And the dose of insulin must be reduced.

‘When the diabetes has been stabilised if the patient has been in hospital or living an unusually inactive life, it is essential to reduce the dose of insulin by at least 20 per cent when normal activities are resumed . . . It is not uncommon during the weeks or months immediately succeeding initial stabilisation for the dose of insulin to fall gradually to a very low level, in fact some patients come off insulin altogether for periods varying from weeks to months, and rarely for years.’

In the light of all this, what does ‘stabilisation’ mean? It would seem to imply a steady state persisting indefinitely, yet it is used especially in relation to newly-developed diabetes in the young, who may be admitted to hospital for ‘stabilisation’. But these are the very cases who do not subsequently remain ‘stable’, and need to reduce their insulin dose steadily. After discharge from hospital some have severe hypoglycaemia and are perhaps re-admitted for ‘re-stabilisation’. Many diabetics on insulin are ‘stable’, it is true, but this state of stability is particularly characteristic of those who have been on insulin for a long time; some may have unchanged insulin requirements for year after year.

‘Stabilisation’ is, then, an improper concept, especially in relation to newly diagnosed diabetes, and the word should be expunged from the language of medicine. Patients should not be given the impression that they are ‘stable’; rather should they be told that their insulin requirements may vary and that they should learn how to make the necessary adjustments—to control, not to stabilise, their malady.

**ADMISSION OF DIABETICS TO HOSPITAL**

Severe diabetic ketosis is the clearest of indications for immediate admission to hospital. There are often good grounds for admitting diabetics with some such complicating maladies as pneumonia, carbuncle, or gangrene. But many physicians regularly admit newly diagnosed young diabetics. This kind of admission is also recommended in two recent British textbooks on diabetes. Oakley et al. (1968) say: ‘In the absence of severe ketosis (most patients) . . . can often be safely treated without admission to hospital, although a short stay in hospital is ideal as it provides opportunity not only for stabilisation of the diabetes but also for teaching diet, urine testing and the techniques of insulin injection’.

And Malins (1968) says: ‘Ideally, every patient who has to start insulin
treatment should be admitted to hospital so that dosage can be worked out at leisure and repeated lessons in injection technique given'.

The fallacy of 'stabilisation' has just been discussed. The education of the diabetic may no doubt justify sessions at the hospital where instruction can be given. But why is it 'ideal' to keep a diabetic in a hospital ward for days or weeks? The ideal management would seem, on the contrary, to be to treat the diabetic from the start while going about his normal activities. If he clearly needs insulin, what is a better time to start than then and there, on his first attendance at hospital? He should be urged to give himself the first injection; I have known few above the age of 12 who did not do so. If he has not already stopped work or school, he should be encouraged to carry on next day as usual; if he has stopped, he should be urged to go back within a few days. This is the policy I have adopted for the last 25 years, with some patients as young as 3 or 4 (when the parents have given the insulin initially). On no single occasion has there been any serious difficulty.

**Urine Testing, Blood Sugar Estimation, and Dose Adjustment**

Diabetics on insulin are usually advised to test regularly for sugar at least the second urine specimen before breakfast and an evening specimen. Probably few test consistently throughout their lives, though most do so occasionally. Some do not test because they claim that they can assess the degree of glycosuria from their feelings. This claim sometimes appears to be soundly based. If most tests reveal no more than a trace of sugar the situation is satisfactory. But if all the urine specimens are loaded with sugar, action is indicated, even if the patient claims that he feels well.

It would seem self-evident that if diabetics are advised to test their urine, they should also be advised to adjust their dose of insulin, or of a sulphonylurea drug. Yet many intelligent longstanding diabetics have insisted to me that they have never been told to change their insulin dose. Instead, the only course they are allowed when glycosuria becomes heavy is to make another appointment at the clinic, when the doctor will order a change in dose. This seems indefensible and encourages the feeling of invalidism, a feeling to be deplored in diabetics. The right policy is, surely, to encourage from the start the idea that diabetics should learn to look after themselves; they have their malady for life, and if they don't look after themselves no one else will, because no one else can.

The reasonable advice about insulin dosage would seem to be: pass as little sugar as possible provided you get no reactions. Probably no diabetic on insulin has consistently negative tests 2 or 3 hours after his heaviest meal.
And some get bad hypoglycaemia although most of their tests are positive. Perhaps, on average, the best that can be achieved is to maintain about half the tests negative.

Diabetic departments vary in their advice to patients on testing for ketone bodies in the urine. All, some, or none may be advised to do so. Many patients whom I have acquired from other areas have produced a list of regular ketone tests, but have insisted that they have been given no instructions as to what action they should take if the tests are positive. Some of those physicians who advise ketone testing tell their patients that regular positive tests are a ground for increasing the insulin dose; others recommend that if the tests are regularly positive an immediate telephone appointment should be made with the clinic.

When a physician is seeing a diabetic he has to guide him the patient’s weight, symptoms, tongue appearance, current urine test for sugar, a list of urine tests for sugar done at home (though these may sometimes be viewed with caution) and, if he wishes, a recent blood sugar estimation. From all these it may be evident that the insulin dose should be increased. It seems difficult indeed to lay down criteria to indicate by how much the insulin dose should be influenced by the urinary ketone test. To add to the difficulty, the test may be positive in degrees from slight to very strong and vary from hour to hour.

If the expert has a problem in interpreting urinary ketone tests, how can the patient be satisfactorily guided by them? It is surely impossible to lay down instructions to the patient as to how much to increase his insulin dose on account of positive ketone tests, when he already has instructions about varying the dose according to the degree of glycosuria. And a positive ketone test is not necessarily of serious significance. One may suspect that the regular performance of these tests adds to the patient’s anxieties without doing him any good.

In most diabetic departments each patient, at least if having insulin, has a blood sugar estimation at each visit. In some departments the current blood sugar level is known to the physician when he sees the patient; in others this figure is not known till after the patient has left.

When diabetes first develops, frequent blood sugar estimations may be useful, though even this is debatable. But the value of a blood sugar estimation every 3 or 6 months at routine hospital visits is by no means apparent. In the best controlled diabetic on insulin there is likely to be a fivefold or more variation between the minimum and maximum blood sugar levels, say between 50 and 250 mg/100 ml. Unless at each visit the blood is always taken at the same time after food or insulin great differences in blood sugar levels may be found, which are not necessarily significant. If a patient has persistent heavy glycosuria, no blood sugar estimation is needed to decide that his
insulin dose should be increased. If, on the other hand, he has slight and infrequent glycosuria and feels well, what action should be taken if his blood sugar level is thought to be higher or lower than ideal? A single blood sugar estimation alone does not provide grounds for varying the insulin dose.

There may be the further difficulty that the doctor does not know the blood sugar level until after the patient has left the hospital. When he learns this figure a day or two later, he may, if he thinks it too high or too low, write or phone the patient or the patient’s GP to advise a change in treatment. Since he has already advised the patient at his visit, the task of deciding how much the advice should be changed by the blood sugar level is difficult indeed.

There remains the special case of the diabetic with a low renal threshold. For him, urine tests are almost valueless because he nearly always has glycosuria. It might be argued that such people should regularly have blood sugar estimations twice a day—and with the dextrostix the patient could do this himself—but I have never heard of this course being recommended. A blood sugar estimation at each 3- or 6-monthly visit to the clinic is of little more apparent value in the patient with a low renal threshold than in other diabetics. If he feels well, with no change in weight and no thirst, a single unexpectedly high (or low) blood sugar figure does not justify a change in insulin dose.

Another way of studying this problem is to inquire what is the object in treating diabetes. The most obvious disorder of the diabetic is his impaired ability to metabolise glucose, which is consequently poured out in his urine. The aim of treatment is to minimise this glucose loss. The means of assessing the loss is urine testing, not blood sugar estimation.

It may therefore be concluded that occasional routine blood sugar estimations do not help in treating diabetics, and they may be misleading.

diabetic ketosis
Throughout the literature on diabetes there are repeated references to diabetic coma, or precoma, as well as to diabetic ketosis. Old people with uncontrolled diabetes are not uncommonly comatose, but there may be other factors such as cerebrovascular disease, in addition to the metabolic disturbance, which are responsible for coma. Frank diabetic coma in young or middle-aged diabetics is very rare. Even people who are desperately ill with diabetic ketosis are usually not unconscious, though their cerebration may be markedly impaired.

This widespread use of the term ‘diabetic coma’ has given the impression to many doctors that if the patient is not comatose the situation cannot be all that bad. I have seen many patients whose admission to hospital was not
requested because they were fully conscious, although vomiting and clearly unwell. When admitted later, they have been very ill indeed, though still not comatose. The situation would be improved if the term diabetic coma was replaced by ‘diabetic ketosis’. It is true that the occasional patient is seen who is very ill and drowsy, or even comatose, with extreme hyperglycaemia, dehydration, and hyperosmolarity, without being ketotic. Such cases have come to be described as examples of ‘non-ketotic hyperosmolar coma’. But, once more, the term ‘coma’ is over-used, as it is applied both to those who are, and are not comatose; ‘non-ketotic hyperosmolar state’ would be better.

The constant use of ‘diabetic coma’ also seems to encourage diagnostic errors. The unconscious diabetic brought to hospital is usually hypoglycaemic. He may be sent to the ward with the label ‘diabetic in coma’. The house physician may then receive an urgent summons to see the new case of ‘diabetic coma’. And he, perhaps raw and inexperienced but conscious of having heard numerous references to ‘diabetic coma’, may ask himself the absurd question: ‘Is it a hyper or a hypo?’ If he then catheterises the patient and finds the urine loaded with sugar he may make the grotesque error of giving insulin. There may be a legitimate diagnostic problem between hypoglycaemia and head injury, stroke, epilepsy, or poisoning; there is none between hypoglycaemia and diabetic ketosis, for no conditions can be more dissimilar.

THE DIABETIC CLINIC
It is widely assumed in Britain that all diabetics, at least if on insulin, should regularly attend a diabetic clinic. Some general practitioners treat diabetics without referral, but these are usually older patients suitable for dietary restriction alone, or dietary restriction and oral drugs. In so far as the patient who attends a clinic regularly sees a physician who has a special interest in diabetics and their problems, there are clear advantages in this policy. But in practice the situation is not always so satisfactory.

Many longstanding diabetics whom I have acquired from other areas have had two complaints about the diabetic clinics they have previously attended—that they are kept waiting (the standard hospital complaint), and that they do not always see the same doctor. They may even say: ‘I never saw the same doctor twice.’ In some clinics an attempt is made to keep patients to the same doctor; in others it is thought positively undesirable to do this. Whatever objections there are to keeping the same patient with the same doctor, most patients prefer it, and resent being fobbed off with Dr A when used to Dr B.

Another disadvantage of the clinic may sometimes be its distance from the patient’s home. This not only wastes time in travelling, but if he becomes
acutely ill he is likely to be admitted to a local hospital where the attending physician knows nothing of preceding treatment. This is frustrating for the physician and may make it difficult to give what he thinks is the best advice for the future. Patients should, therefore, attend a hospital into which emergency admissions are practicable. Physicians in charge of diabetic departments should not accept patients who come from long distances.

But perhaps the biggest problem arises when the patient who is attending a clinic suddenly becomes ill. From whom should he seek advice—the clinic or his own GP? No easy answer can be given to this question, since circumstances vary so greatly. But it can be most difficult for the GP when called to an ill diabetic who, though nominally his patient, is regularly looked after by someone else.

Is there, then, any objection to GPs looking after their own diabetics from the start? To conclude that they are all incapable of so doing is an insult. Perhaps the most important quality needed to look after diabetics is common sense, which GPs are as likely as anyone else to possess; and the GP’s knowledge of the patient’s home background can help. On the other hand, many GPs prefer to have further advice about diabetics. Probably the best routine plan is for the patient to attend the hospital initially and subsequently to attend his own GP, with occasional follow-up visits to the hospital.

Some of the most successful diabetics I have known have dealt with their own problems unaided for years. They have come under my care on account of some other malady and have then said that they soon tired of attending a clinic and that the only contact they have had with their GP has been to leave a note requesting repeat prescriptions. For year after year they have had no appreciable trouble. They have adjusted their dose of insulin when appropriate, and some have regularly taken more or less insulin at weekends, according to their activity. Such people have usually been intelligent, and have read all about diabetes.

**Discussion**

Despite the immensity of the research and published information about diabetes, only the following conclusions can be reached about treatment (leaving aside the oral drugs):

1. Calorie restriction is the supreme remedy for the obese maturity onset diabetic.
2. Insulin is the means of keeping most juvenile onset diabetics alive. Its dose must be adjusted to maintain the patient continually well, without becoming obese.
Many other widely accepted beliefs about diabetic treatment depend largely on theory, not on evidence, and are followed as a matter of habit. A rigid diet for active diabetics on insulin is of unproven value, is impracticable if varying exercise is taken, and, if advised, is rarely observed. Routine admission to initiate treatment is worse than unnecessary. Occasional blood sugar estimation and self testing for urinary ketones do not help to improve control. ‘Stabilisation’ is an almost meaningless word which should be expunged from the language of medicine. By a strange custom, there is constant reference in medical writing to ‘diabetic coma’, although very few of the patients so described are in fact comatose.

Many of the views advanced in this article cannot be strictly proven. But when a matter is in doubt the onus of proof should rest upon those who advocate, not those who oppose some course, especially when this has clear disadvantages. Admission for the initiation of treatment is costly, implies the cessation of work or school, and is disliked by most people. Those who advocate it should supply the proof; they should not just make an ex cathedra pronouncement that it is ‘ideal’. And those who advocate a blood sugar estimation when patients attend for a routine visit should explain just how this helps to improve the treatment, and those who instruct patients to test their urine for ketone bodies should demonstrate how this is beneficial.

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