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THE CLINICAL APPROACH TO THE PROBLEM OF DIABETES MELLITUS*

BY

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A few years ago Dr. James B. Conant (1947), in delivering the Terry Lectures at Yale University, pointed out "that scientific advance depends on two main factors—first, the development of new concepts, and secondly the development of new techniques—and that these two processes are mutually interdependent and complementary one to the other." In similar vein Dr. R. D. Lawrence (1954) said that "the twin sisters clinical observation and laboratory experiment had walked in the field of diabetes very closely hand in hand, to their mutual stimulation; the latter method is more exact, the former often more initially suggestive." It seems that, with the increasing complexity of research which is developing along so many lines, the place of the clinician can be considered in the terms of Conant's "new concept, new technique" hypothesis for the benefit of both practitioner and research worker. I feel that at times there is a tendency for clinicians, when attempting a scientific approach to a problem, to forget the role of clinical observation or to relegate it to a minor place.

The Planned Study of Disease

Shortly before his death Sir James Spence (1954) pointed out that there were two main kinds of clinical science, one of which is in danger of being overwhelmed by the other. He defined clinical science as the planned study of disease or of the phenomena of disease in human beings who are sick. He stated that this was a practical craft and that the clinical scientist must possess a particular skill necessary for collecting and systematizing knowledge. The first branch of clinical science is "the planned observation of the sequence of phenomena in that number of sick people which allows us to know the common course of the disease under study from its beginning to its end and to estimate and know the variations from that course." He suggested that this had immense practical value, for a knowledge of disease in this way forms the basis of exact therapy in professional practice. He termed this branch of clinical science "clinical cartography," because it helps the practitioner to know where he is and where he should go.

The second branch of clinical science he termed "clinical phenomenology." This seeks to study the isolated phenomena of disease, and its aim is to explain the mechanism and clinical significance of such states. He paid tribute to the enormous value of the work performed by the late Sir Thomas Lewis in establishing the precise and rational study of clinical phenomena. However, he qualified this in saying that by the mere force of his personality Lewis had so influenced clinical research in Great Britain in recent years that the study of clinical phenomena had overshadowed other kinds of clinical research, and, although the advantages derived from this influence had been great, there were grounds for believing that its dominance had led to the neglect of the planned study of disease. It could be inferred from his statements that the influence of William Osler and James Mackenzie was waning and that a return to the long-term study of the individual patient could still yield fruitful results.

The Concept of Pancreatic Involvement

The successful isolation of insulin in 1921 by Banting and Best produced a revolutionary change in medical thought and outlook on the problem of diabetes which has persisted to the present day, and it is appropriate to consider the effects of this discovery upon the cartography of the complaint. The concept of pancreatic involvement in diabetes which developed through a half-century recalls the distinguished contributions of Langerhans, von Mehring, Minkowski, Laguesse, Opie, and many others, whose observations led by induction to the conclusion that the pancreas was in some way intimately concerned with this complaint. At the time of the isolation of insulin the clinical concept of diabetes was of a static nature. Woodyatt (1909) stated "that diabetes is a disease in which the body has lost in part its ability to utilize sugar. Sugar arrives at the point where it should burn, but, failing to do so and accumulating in the blood, creates a hyperglycaemia. Disregarding accessory factors, which may play a part, we can say that ultimately the failure of sugar combustion in diabetes mellitus depends upon the lack of a something derived from the pancreas."

Within the next decade, clinical medicine came into the debt of Allen, whose scientific approach had produced a rational basis for treatment which greatly improved the outlook of many patients. In 1919 he said that "the status of the islands of Langerhans as an internal secretory organ and as the seat of the specific diabetic disturbance is now as firmly established as any fact in physiology or pathology" (Allen *et al.*, 1919). As a result of his experiments he was convinced that diabetes arose solely from pancreatic dysfunction, and considered it to be "a unified entity rather than a disjointed symptom complex."

*The Banting Lecture delivered at the University of Toronto on May 16, 1955.

The remarkable happenings of the past thirty years are still too close to view in true historical perspective. Nevertheless I think it is true that the cartography of diabetes has not kept pace with the growth of knowledge of diabetes in other spheres. The concept of a primary pancreatic failure as the reason for the development of human diabetes rests on a less secure foundation than it did, but, so far, clinical observation has failed to suggest a satisfactory alternative. The influence of various endocrine dyscrasias in varying the pattern of clinical diabetes has been established; the effect of alterations in dietary habits upon insulin requirements has been recognized, with relaxation of the rigidity of carbohydrate restriction; the existence of pre-diabetic states has been suggested; the unified concept of the aetiology of diabetes has been challenged; the influence of obesity has been recognized; the importance of heredity has been stressed; the occurrence of separate clinical types of diabetes as suggested by Lancereaux has received experimental support; while the grim realization that long-standing diabetes predisposes to premature arteriosclerosis has posed a problem second only to that of the causation of diabetes.

The unified concept of the pancreatic origin of diabetes led to the idea that the complaint when once established was incurable and that a patient was maintained in health only by living within the limits of his decreased insulin output, although this has never been proved, or by using insulin as a means of substitution therapy. Yet Banting (1929) himself quoted an early case which is of much interest. In August, 1922, a woman of 65 suffered an infected gangrene of the leg with ketosis and a high blood-sugar content. After five days of insulin therapy her urine was sugar-free, her leg was amputated, and she improved rapidly. After four months insulin was discontinued and eight months after the operation she was living satisfactorily on a free diet. For three years thereafter she remained well, dying eventually of pneumonia in 1926.

Fluctuation in Severity

In 1930 I first observed the apparent fluctuation in severity of diabetes (as estimated by variation in insulin dosage) in patients suffering with infections of various types (Downie, 1930). I also noticed disturbances in the carbohydrate utilization (as shown by variations in glucose-tolerance tests) in patients with no diabetic heredity during the course of infections (Long and Downie, 1932). With subsidence of the infections such patients showed normal glucose-tolerance curves, which remained normal over periods of some years. Since then this problem has interested me and has stimulated thoughts that perhaps there are some forms of diabetes which are not permanent throughout life but which remit or disappear under influences as yet unrecognized. The relationship of obesity to the development of frank clinical diabetes presents a similar problem. In 1939 Newburgh and Conn showed that in many patients of this type reduction of body weight to normal limits resulted not only in the disappearance of symptoms but in a reversion of the glucose-tolerance curve to normal and in some instances to an apparent cure, as the curves remained normal over many years of observation.

This raises a question whether in the cartography of diabetes reversibility of the disorder can occur apart from the conditions already recognized—namely, infections, endocrine disorders, pregnancy, and obesity. Obviously the clinical assessment must rely in the last instance on the figures obtained by a glucose-tolerance test performed under standard conditions, with due regard to previous diet and the estimation of true blood sugar. This problem has attracted relatively little interest, which is all the more surprising in view of the suggestions of Haist and Best (1941), who indicated possible ways in which clinical trials could be undertaken in an attempt to protect the beta cells of the pancreas from damage. Watson (1942, 1949) described a series of patients whom he had observed over a period of years in which diabetes seemed to subside and to recur under the influence of dietary restriction and excess.

Influence of Diet

It so happens that there has come within my experience a group of patients which not only supports the findings of Watson but which poses additional questions. As a clinician I find it perplexing to be confronted with problems in cartography to which there is, as yet, no answer. To illustrate this I propose to consider briefly some case histories taken from my experience since the war.

The first two cases are concerned with the effects of obesity. In the first case, an apparent remission occurred with a fall in weight, only to be followed two years later by a recurrence

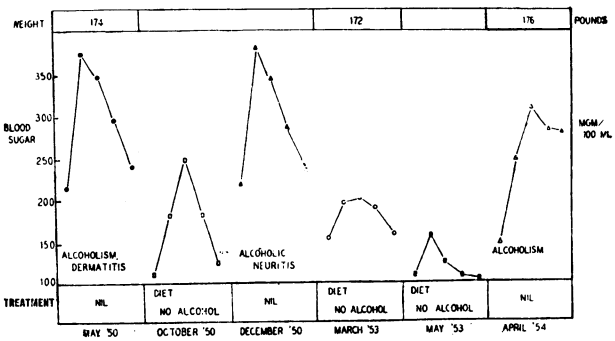

FIG. 1.—Man aged 51. No family history.

which was not associated with any gain in weight. Diabetes has persisted, and the patient has required insulin for the past six years. The second case is one of response to reduction in weight with a relapse after carelessness with diet. Symptoms recurred and subsided with weight reduction, and at least for a time diabetes has again passed into a stage of remission. The next example may possibly be related to the effects of infection. It is that of a man whose mother suffered from diabetes, and who developed typical symptoms at the age of 55 years in 1947. With dietary restriction he remained well, and in 1953 showed a normal glucose-tolerance curve after a period of unrestricted diet. Within the past year, following a virus pneumonia, he again developed symptoms, and his curve has reverted to a diabetic pattern.

The next case history is that of a man of 51 with no known family history of diabetes whose diabetic state seems to come and go with his alcoholic habits (Fig. 1). He has been an extremely heavy drinker for many years, but on two occasions within the past five years his diabetic condition seems to have subsided with periods of abstinence.

It is tempting to try to relate these findings to some cause; obesity, alcohol, or infection, and to infer that, in some way, dietary or alcoholic indiscretion or infection has played a part in determining a relapse in a susceptible subject. However, the next two examples do not permit such an explanation, as recurrence has developed for no apparent reason.

The first history is that of a man aged 56 in 1937 who was diagnosed as suffering from diabetes prior to an operation for thyrotoxicosis while in London. His father was a diabetic. He was stabilized on insulin and remained on this treatment for 10 years, when the use of insulin was discontinued because of severe and repeated hypoglycaemic attacks. He remained well, and glucose-tolerance tests were normal for the next six years. Then for no demonstrable reason he relapsed with typical symptoms of uncontrolled diabetes and has resumed insulin therapy.

The second history is that of a man of 40, not overweight, whose father and an uncle suffered from diabetes. He developed typical symptoms of the complaint in 1948. Within three months of dietary treatment and with no appreciable alteration in weight, his glucose-tolerance test gave a normal response. It remained so for at least the next five years, when he again developed symptoms with a typical recurrence of diabetes. No obvious reason has been ascertained for this.

It is, of course, the privilege of patients to rationalize their experiences and to attribute the development of diabetes to this or that event which, in their minds, is directly related in time to the development of symptoms. Be this as it may, a sudden shock or nervous stress is often mentioned as a precipitating factor.

A man of 50, whose father and mother suffered with diabetes, was found to have symptomless diabetes when examined for life assurance (Fig. 2). With dietary treatment his glucose-tolerance reverted to normal and remained so for over two years. Then, following a period of severe financial stress, he developed symptoms for the first time in his life and again showed a typical diabetic response to a glucose-tolerance test. Another man, aged 62 years, with no family history of diabetes, developed

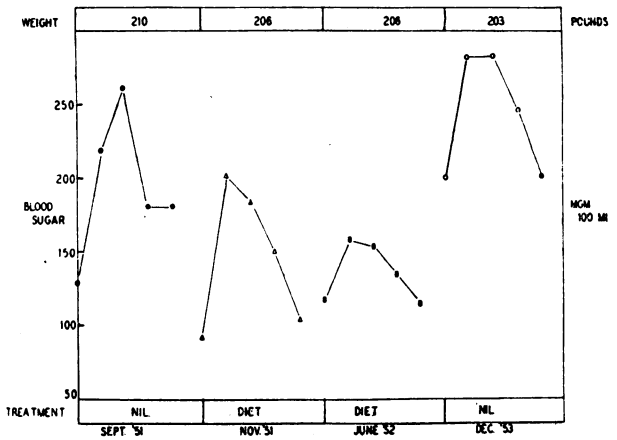

FIG. 2.—Man aged 50. Family history, two cases.

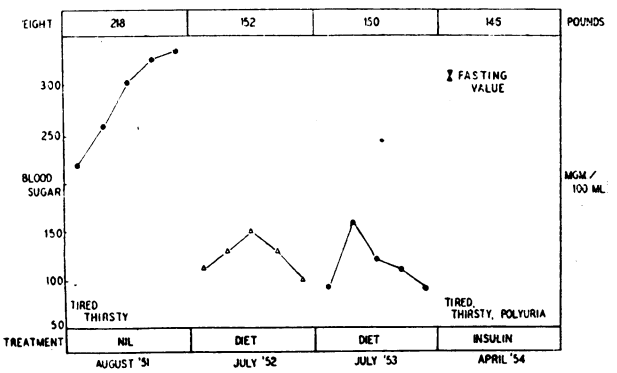

FIG. 3.—Man aged 62. No family history.

symptoms in 1951 (Fig. 3). He had been overweight for many years, and with reduction of his weight to normal his diabetes passed into a remission for over two years. Then, following a long period of intense domestic stress, he again developed symptoms and now requires insulin for satisfactory control.

All these patients have been maintained on a mild carbohydrate restriction (between 150 and 200 g. daily) throughout the period of observation except for one week prior to the performance of glucose-tolerance tests, when their intake has been raised to approximately 300 g. daily. It seems significant that the majority of these patients have shown clear evidence of a strong hereditary influence. This surely poses a question of considerable clinical importance in these days of diabetic detection drives, for if attempts are made to discover the diabetic patient in his earliest symptomless phase, what advice is to be given by the physician ?

A Technique of Value

Before proceeding into this controversial field I wish to refer to a technique which may well prove to be of value to the physician confronted with such problems. Fajans and Conn (1954) have used a cortisone-reinforced glucose-tolerance test for detecting disturbances of carbohydrate metabolism that are not revealed by the standard tests in common use. I have recently applied this technique, not to the problem of detecting unknown diabetes, but to patients who have developed diabetes and have subsequently passed into remission. A few examples are considered briefly.

A child of 6 years, with a family history of diabetes in a maternal grandmother and a paternal grandfather, developed diabetes in 1950 and showed a typical response to a glucose-tolerance test (Fig. 4). She was controlled with dietary restriction and insulin, and responded quite satisfactorily to treatment throughout 1951 and 1952. Blood-sugar estimations during this period were usually within normal limits. Early in 1953, with some trepidation, I discontinued insulin and maintained a strict dietary regime. Her blood-sugar values remained within normal limits and her urine tests showed no sugar. Fascinated by this, I performed a glucose-tolerance test after a week of completely unrestricted diet. The result was normal. Dietary treatment was resumed, and six months later another test was performed after unrestricted diet, and it was normal. A year later it was still normal, and a cortisone-reinforced test was also normal.

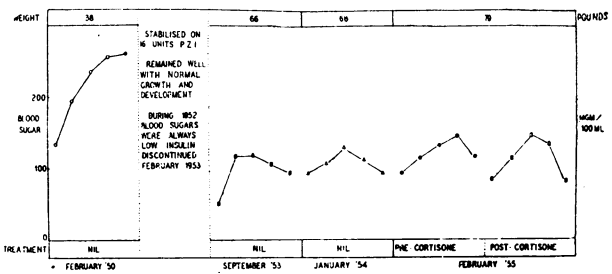

FIG. 4.—Girl aged 6. Family history, two cases.

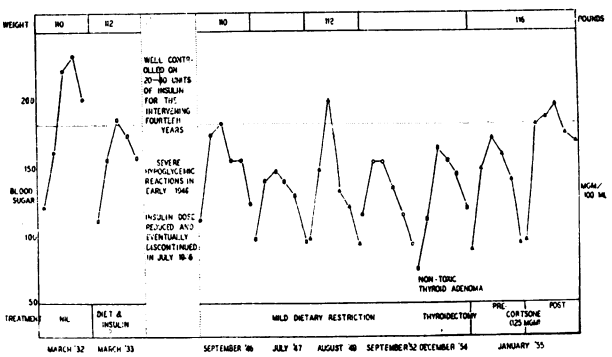

FIG. 5.—Woman aged 27. Family history, one case.

This child is unique in my experience. She is normal in height and weight for her age (10 years), but I am still fearful that when she approaches puberty diabetes will recur. Had I a preparation of purified growth hormone I would be tempted to use it prior to a glucose-tolerance test in an attempt to resolve my doubts.

In 1933 I saw in consultation a woman aged 27 (Fig. 5) whose father had suffered from diabetes. Two years previously she had felt tired, thirsty, and had lost weight, and glucose-tolerance tests revealed diabetes. She was stabilized with diet and insulin, and remained satisfactorily controlled for the next 14 years. She then came under my observation because of repeated hypoglycaemic attacks which had developed only within the previous six months. Within the next six months I discontinued insulin, and with dietary treatment alone her blood sugar values were always normal. Accordingly I carried out a glucose-tolerance test after a short period of unrestricted feeding, and it was normal. For the past eight years she has followed a mild dietary restriction with a daily intake of carbohydrate of approximately 175–200 g. Her glucose-tolerance tests have been normal, even in 1952, when she had a severe throat infection which lasted several weeks and failed to respond to antibiotic therapy. Within the past few months a glucose-tolerance test was quite normal, but with cortisone reinforcement a typical diabetic curve was found.

Fajans and Conn investigated six mild obese diabetic patients, all of whom, after weight reduction, exhibited normal glucose-tolerance curves. With cortisone reinforcement they all showed a significant deviation from their previous response. I have recently had the opportunity of observing two obese patients whose symptoms subsided with weight reduction and whose glucose-tolerance curves returned to a normal pattern. Each subsequently showed a reversion of the curve to a diabetic pattern, although there was no alteration in weight or other determining factor. These two men were studied with cortisone-reinforced glucose-tolerance tests and gave different responses.

The first example is that of a man aged 36 with no known family history of diabetes (Fig. 6). He has always been heavy, and on discharge from the Army in 1946 weighed 280 lb.

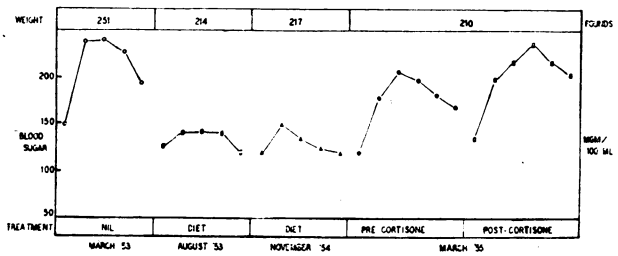

FIG. 6.—Man aged 36. No family history.

(127 kg.). His height was 6 ft. 4 in. (193 cm.). In 1952 he developed definite symptoms of diabetes, and a glucose-tolerance test gave a typical response. His weight was 252 lb. (114.3 kg.). Within four months of dietary restriction his weight fell to 210 lb. (95.3 kg.) and his glucose-tolerance test was then normal. He remained on a mild carbohydrate restriction, and 15 months later the curve was still normal. A further review early this year showed that the curve had changed to a diabetic pattern. A cortisone-reinforced test was then performed and showed identical figures.

The second example is that of a man aged 44 whose father had suffered from diabetes. In 1953 he began to feel tired, and a glucose-tolerance test gave a typical diabetic response. His weight was 205 lb. (93 kg.) and his height 5 ft. 6 in. (167.3 cm.). With dietary treatment his weight fell to 176 lb. (79.8 kg.) in a period of seven months and the glucose-tolerance test returned to normal. Subsequently, for no obvious reason, it changed to a diabetic pattern, and with cortisone reinforcement a significantly greater deviation from normal was observed.

Finally, in 1952 I treated a girl of 12 years who presented with acute symptoms of diabetes which had been present for a few weeks. She was easily controlled with insulin and diet, and has remained well ever since. There was no family history of

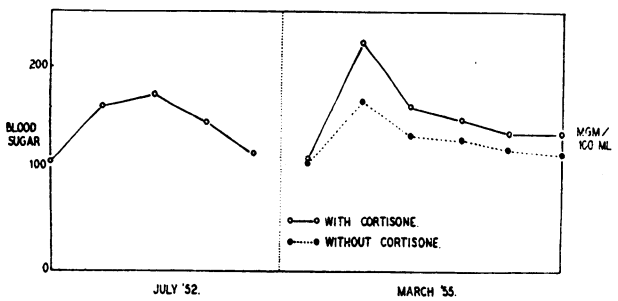

FIG. 7.—Girl aged 12. Identical twin sister has diabetes, controlled by 110 units of insulin daily.

diabetes, but the patient was an identical twin. Her sister was given a glucose-tolerance test in 1952 and showed a normal response (Fig. 7). This twin has remained well and symptomless ever since, and a recent test was still normal, but a subsequent cortisone-reinforced curve showed a significant deviation.

Four of these patients who have been investigated with cortisone-reinforced glucose-tolerance tests have had clinical diabetes. In none of them has there been any other clinical abnormality at the time of onset, and in all of them diabetes seems to have subsided for an appreciable period. In the four diabetic patients, cortisone reinforcement failed to alter the pattern of the curve in two, while a significant change was observed in the other two. The fifth patient, an identical twin of a diabetic sister with no clinical evidence of diabetes, shows a significant response to cortisone stimulation.

A Personal Concept

The concept I have developed from this experience is that of a dynamic fluctuating state rather than one of a static and permanent disease entity. For the present, reversibility of diabetes must remain a clinical dream. Yet, if we reflect upon the histological pattern of the diabetic pancreas and on the work of Wrenshall and his associates (1952), it must be realized that at least in some instances of diabetes of long standing the beta cells are obviously neither dead nor functionless. This leads to the speculation that they may be under the influence of some agent which, while allowing them to live as sick cells, is not lethal for them. One of the great experiences of my clinical life has been to witness the effect of cortisone therapy on the course of rheumatoid arthritis. Joints which, fixed, gnarled, and functionless, were regarded as the end-result of some noxious influence, have been transformed, causing a complete alteration in our concept of the nature of the complaint. With such thoughts in mind, I wonder whether something as yet unknown may some day be capable of reversing the changes in sick pancreatic cells.

It is permissible to speculate that in the cases just described an oscillatory state of bodily homoeostasis has been disturbed by factors beyond our present knowledge. The effect of carbohydrate restriction in compensating for this disturbance may represent no more than an adjustment to an altered equilibrum which may be influenced by any one of several stimuli that are not necessarily related to one another. The stimulus to the development of diabetes in a susceptible individual, like the clinical pattern of the disease at different periods of life, does not seem to be the same in all cases.

Heredity

In a significant number of the patients I have studied there is the common factor of heredity. I have formed the opinion that this is of greater frequency than can be disclosed by figures obtained from the interrogation of patients known to be suffering from the complaint. It is not uncommon to observe diabetes appearing in a child or a young adult whose family history is quite clear. Then, years later, the father or the mother or some other close relative will develop diabetes at an advanced age in life. A few years ago, Harris (1951) pointed out that various hypotheses have been put forward at different times to account for the observed familial distribution. Levit and Pessikova (1934) suggested that the condition could be regarded as an irregularly manifesting heterozygous character, while Pincus and White (1933) put forward the hypothesis that diabetics were homozygous for a relatively common recessive gene. Harris (1951) pointed out that there were certain features of the familial distribution which were inconsistent with both of these views. He suggested that sometimes the late onset and less severe forms of the disease may be heterozygous for a gene, which in homozygous form leads to the early onset and more severe type of case.

The problem of heredity is one of cartography, and is not likely to be solved by laboratory studies of phenomenology alone. My colleague and mentor, Sir MacFarlane Burnet (1953), has pointed out the importance of three new subsciences—population genetics, biochemical genetics, and microbial genetics—all of which are likely to have very great influence on the future as well as in the future. He gives as an illustration the condition of congenital amentia which arises because of the genetic absence of an enzyme (phenyl pyruvic oxidase) in body cells. This he regards as an example of biochemical genetics which he thinks may well emerge as the master biological science of the future.

He suggests that in the last analysis all the changes produced by genes are expressed through a series of chemical reactions and that there is a growing belief that in some way each gene controls no more and no less than a single type of enzymic action. If the generalization "one gene—one enzyme" is finally established it will represent one of those great simplifying concepts like the idea of evolution by natural selection or the quantum theory.

This view, when considered with Harris's suggestions, can be related to the problem of human diabetes. Some few years ago Himsworth (1949) suggested that diabetes should be regarded as a symptom-complex rather than as a disease *sui generis*. Hyperglycaemia, the common factor in all diabetes, may not necessarily arise from the same cause in every diabetic patient. Likewise, no satisfactory attempt has yet been made to reconcile the arteriosclerotic complications of long-standing diabetes to genetic defects, yet no clinician of experience can deny that this is an inexplicable problem. For many years attempts have been made to relate it to adequate control of diabetes as measured by maintenance of normoglycaemia. It falls within the knowledge of us all that, although strict control of diabetes seems to offer some protection against vascular damage, it is by no means proved that it does so in all cases. This may be related to different types of diabetes arising from insulin deficiency or from disturbed utilization, or to genetic influences suspected but as yet unproved.

Conclusion

Medicine in the present day is becoming increasingly indebted to the developments of science and has need to keep pace with the ever-expanding frontiers of knowledge which are opening before it. Yet it seems that the sense of perspective, so necessary to any critical appraisal of new knowledge, must constantly be related to clinical experience. Cawadias (1953) has said that "the history of medicine has shown that, whenever medicine has strayed from clinical observation, the result has been chaos, stagnation, and disaster." This represents a serious challenge to those of us charged with the management of diabetic patients. The clinical concepts of diabetes at present rely heavily upon the interpretation of the glucose-tolerance test to determine the existence of this nebulous entity, diabetes, which, in the words of F. G. Young, cannot be precisely defined. The twin sisters clinical observation and laboratory experiment must obviously continue to walk together very closely, the one to produce concepts of reversible or curable diabetes, the other to produce techniques which, applied to the patient like Mackenzie's polygraph, will enable us to plot the course of the disease with ever greater accuracy. The need for long-term genetic studies of diabetes of fluctuating intensity must surely lead to greater understanding, while the development of new techniques which, for example, could provide an easy and rapid estimate of pancreatic, adrenal, and pituitary activity, would place into the hands of the physician weapons with which the problem of diabetes might be solved.

Surveying the development of our knowledge of this most fascinating and baffling subject, the secret of diabetes mellitus, we are reminded of the contributions of the great men who have passed. Of these Frederick Banting, whose memory we honour this day, must serve as an example of one who combined a concept with a new technique and so placed the world in his debt. The stimulus of his discovery is still felt throughout the world, while millions of patients owe their health and existence to him. It can be truly said that he has joined

> The choir invisible
> of those immortal dead who live again
> in minds made better by their presence, live
> in pulses stirred to generosity,
> in deeds of daring rectitude, in scorn
> for miserable aims that end with self,
> in thoughts sublime that pierce the night like stars
> and with their mild persistence urge man's search
> to vaster issues.

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THE DECREASE IN SURGICAL PAEDIATRIC DEATH RATE

BY

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The decrease in the mortality rate from various diseases and operations is an insidious process. Although one may suspect it, it is dramatically evident only when definite figures are produced. Coincident with a reduced death rate there is always a lowered morbidity rate, and in proportion to the latter there is an increased turnover in the bed state due to the shortened time in hospital. Unfortunately, other factors have crept in to counterbalance this, so that the waiting-list for the average hospital is greater to-day than it was 10–20 years ago; but a National Health Service, a Welfare State, and other personal economic factors have, in the meantime, come to play their part in the life of the private individual.

In the Royal Belfast Hospital for Sick Children the figures seemed striking enough to warrant publication. They probably represent merely what has happened in all similar institutions. The number of surgical beds in the unit has remained fairly constant. Two seven-year periods were selected, 1931–7 and 1947–53.

A series of those diseases which recur with sufficient frequency to warrant classification has been selected, and the results are shown in the Table and Fig. 1.

It may be argued that with some of these—for example, cleft-palate and pyloric stenosis—where the disease and the time for operation are under the control of the surgeon, the death rate should be nil. On the other hand, with others, such as burns, trauma, and the toxic conditions, the outlook must depend to a certain extent upon the insult the body has received in the way of shock and infection before treatment was started. Fig. 2 shows that there has been a steady decrease in the average age of death, largely due to the fact that surgery is being carried out for congenital abnormalities—that is, oesophageal atresia—which, in the first period, did not