The method of Shouldice⁵ depends on preliminary repair by overlapping of the transversalis fascia forming the posterior wall of the inguinal canal followed by a modified Bassini type of repair.

It should be possible to determine the method that most effectively prevents recurrences, but carefully controlled clinical trials are difficult to construct because of the number of variables. Patients vary in age, sex, obesity, and activity. Hernias are indirect or direct, small or large, recent or long standing, with firm or diffuse margins to the defect. Surgeons vary in experience, meticulousness, enthusiasm for carrying out large numbers of hernia repairs, and desire to follow up their patients for long periods and adjust the technique in response to the results. The most variable aspect of all is operative method: some techniques named for an originator would be disowned by him because the user has neither watched him nor even bothered to read his original description. In addition, none of us can resist adding our own little "improvements."

Methods of reporting the results also vary. Some patients are personally followed up for long periods, others for short periods with indirect assessment of recurrence. Recurrence is usually given as an overall percentage but may be calculated after separating the original hernias into different groups and using statistical corrections.⁶

Nevertheless, certain trends emerge. Pooled series show high recurrence rates,^{7 8} individual series show low rates,^{5 9} but recurrences probably are less frequent than formerly. Before the second world war a surgeon was proud to report recurrence rates of around 5%¹⁰ and a review of 13 major studies in 1954 showed recurrence rates as high as 15%.¹¹ At present large series are reported with recurrence rates below 1%.^{5 9 12 13} We should remember, however, that the surgeons who report most of the results do not perform most of the hernia repairs. The writers of papers are enthusiasts, committed to promulgating a method and striving to achieve better results than others. Most of the herniorrhaphies in Britain and elsewhere⁶ are performed by surgeons in training. Sir Cecil Wakeley made a statement on cost effectiveness before that term became fashionable when he stated¹⁰ in 1940: "A surgeon can do more for the community by operating on hernia cases and seeing that his recurrence-rate is low than he can by operating on cases of malignant disease."

One common argument is that the type of repair should be tailored to the type of hernia,⁶ so that difficult, diffuse hernias may require extra precautions to facilitate closure of the defect.^{14 15} At the Shouldice Clinic in Toronto a single method is used, almost unvaried, for all types of hernia, and Glassow⁵ from there has now reviewed his enormous experience and outstanding results in a Hunterian lecture at the Royal College of Surgeons of England.

In my experience outstanding and thoughtful surgeons who devise a new technique attribute their success to the method. They are too modest. Their colleagues know that it is not the particular method that brings success but the enthusiasm for perfection and the painstaking skill with which it is accomplished. If I develop a hernia I shall not worry about the method of repair but about who is carrying it out.

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Causes of insulin dependent diabetes

Ancient Indian physicians believed that diabetes was a heterogenous disease caused by genetic and environmental factors. The evidence is that they were correct, and we now recognise two major types of diabetes which are genetically distinct—insulin dependent diabetes and non-insulin dependent diabetes.¹

Genetic influences are powerful in insulin dependent diabetes, especially through genes in the HLA region (HLA-B8, B15, DR3, and DR4). But juvenile onset insulin dependent diabetes is at least partly determined by non-genetic factors. Studies of identical twins have shown that half of those with insulin dependent diabetes have an unaffected cotwin; and as identical twins share the same genes such differences must be due to environmental agents.² Even insulin dependent twins with an unaffected cotwin, however, are genetically susceptible to the disease, so it is unlikely that environmental agents are entirely responsible for insulin dependent diabetes.³

If insulin dependent diabetes is caused by both genetic and environmental factors and they interact then they probably do so through the immune system. Unlike non-insulin dependent diabetes, the immune system has been implicated in the pathogenesis of insulin dependent diabetes: the HLA region is the same genetic region which controls the immune response in mice, and the disease is associated with autoimmune diseases such as hypothyroidism.^{1 4} In addition, recently diagnosed insulin dependent diabetics have mononuclear inflammatory cells permeating and surrounding their islet cells and islet cell antibodies.^{5 6} This insulinitis might be produced either by destruction of islet cells or by an autoimmune reaction; both could be caused by an environmental agent acting in a genetically susceptible host.

What is the environmental agent responsible for insulin dependent diabetes? Attention has focused on viruses and toxins. "Wild" viruses exist which have a specific β cell

trophism and a diabetic potential in animals.⁴ Studies on animals, notably with diabetes induced by encephalomyocarditis virus, show that viruses can produce a type of disease like insulin dependent diabetes.⁷ There are four common human viruses which cause islet lesions in animals: Venezuelan encephalitis virus, Cocksackie B4, reovirus type 3, and rubella virus. In man viruses may damage the pancreas either generally, as with mumps pancreatitis, or specifically by damaging the islets. After fatal viral infections damage to the islet cells is found in about 10% of patients, and in laboratory experiments Cocksackie and mumps viruses have been shown to multiply in human pancreatic β cells and destroy them.^{4, 8}

A causal relation between viruses and diabetes was suggested last century, and numerous anecdotal reports have recorded the onset of diabetes days or months after viral infections.⁴ Diabetes is frequently found in patients with the congenital rubella syndrome, and these diabetics show an increased prevalence of HLA-DR3.⁹ There is little serological evidence at diagnosis, however, that insulin dependent diabetics have had a recent viral infection. One study, as yet unconfirmed, found that within three months of diagnosis insulin dependent diabetics had higher antibody titres to Cocksackie B4 than either those with insulin dependent diabetes of longer duration or normal people.¹⁰ In another study newly diagnosed insulin dependent diabetics with both HLA-B8 and B15 had higher antibody titres to Cocksackie B viruses than those who did not have these genes.¹¹ This accords with the hypothesis that there is a genetic and viral interaction producing the disease. The observation that insulin dependent diabetics are diagnosed most frequently in autumn and winter lends further weight to this theory.^{4, 11, 12} In one unusual case, where a young boy died from an overwhelming viral infection and diabetic ketoacidosis, Koch's postulates with regard to a viral aetiology of diabetes in man were nearly fulfilled.¹³ A Cocksackie B4 virus was isolated from his pancreas and it induced diabetes with islet cell damage in mice susceptible to diabetes. Evidence of this kind indicates a short interval between the viral infection and the onset of insulin dependent diabetes, against which there is strong evidence in favour of a long latent period before diagnosis when these diabetics show both immunological and metabolic changes.¹⁴ The explanation may be that a viral infection may precipitate the illness but that it need not necessarily initiate the islet cell damage.

Of toxins, only N-nitroso derivatives are known to induce diabetic ketoacidosis. Both alloxan and streptozocin cause diabetes in certain animals, and streptozocin produces insulinitis similar to that seen in insulin dependent diabetes—as well as breaking gene strands and inducing the appearance of type C viruses within surviving islet cells.¹⁵ The rat poison Vacor can cause diabetes in man, and after fatal poisoning necropsy has shown extensive β cell damage. Low concentrations of insulin and high concentrations of glucagon in these patients suggest that the poison has a specific cytotoxic effect on the β cells, and two patients were found to have islet cell surface antibodies.¹⁶ The only evidence that toxins are important in the aetiology of insulin dependent diabetes in general derives from a study in Iceland.¹⁷ As elsewhere, insulin dependent diabetics in Iceland are more likely to present during the autumn, and the time of diagnosis coincided with epidemics of viral disease in those under 14 years of age.

In contrast with other studies, however, there was a noticeable excess of male diabetics born in October, though these diabetics had the expected HLA genetic susceptibility. The authors speculated that this excess in October was due to parental consumption of smoked cured mutton. This meat,

traditionally eaten at Christmas and New Year, is rich in nitrites and N-nitroso compounds. Support for this tenuous hypothesis came from a study on mice which showed that when fed with the mutton they had male offspring with increased glucose and decreased insulin concentrations associated with β cell destruction¹⁸—though the islets showed no evidence of insulinitis typical of insulin dependent diabetes in man. No other food additive has been implicated in the pathogenesis of insulin dependent diabetes, though the low prevalence of insulin dependent diabetes in primitive societies may be worth noting.¹⁹ It is also relatively uncommon in Japan, where legal limits for food nitrite content are particularly strict. Because juvenile insulin dependent diabetes is more common in men than women the male susceptibility to the diabetogenic effect of smoked mutton, streptozocin, and possibly Vacor is of interest. But this does not favour toxins against viruses in the pathogenesis of insulin dependent diabetes, as men are also more liable than women to some viral infections.

So from the ancient belief that diabetes was caused by genetic and environmental factors we now have firm evidence that an environmental agent (or agents) is responsible in part for insulin dependent diabetes. Identification of this agent might lead to prophylactic treatment against the disease. But, while both viruses and toxins can probably cause diabetes in man, it remains unclear whether either is the environmental agent responsible for most insulin dependent diabetes.

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