Adhesives and Closure of Wounds

SIR,—I read with great interest your leading article (17 January, p. 129), and the papers on closure of wounds by Mr. G. T. Watts (21 February, p. 501) and adhesive and closure of wounds by Mr. J. Capperault (28 March, p. 819).

There are two types of skin wounds: those in which no tissue has been lost and those in which tissue has been lost. When the skin wound in which no tissue has been lost is closed with cyanoacrylate tissue adhesive in place of suture the following advantages are apparent: more rapid repair, and better cosmetic results.

However, cyanoacrylate monomers polymerize on the tissue, and the tissue reaction is more extensive than those caused by implanted suture materials or wires. The higher homologues which evoke less tissue reaction degrade very slowly.¹ Since fragmented and undegraded polymer films are occasionally encapsulated by a pocket of connective tissue oncogenic study of a longer period is necessary.²

The definite disadvantage of the skin closure with the higher homologues such as normal or isobutyl cyanoacrylate is the fact that the maximum wound strength is attained later than for sutured wounds, owing to the presence of polymer fragments between the approximated tissue which prevents the proliferation of the microcirculatory vessels and fibroblasts bridging the wounded surfaces.³ Therefore, when tissue adhesive is used for closure of large wounds, such as laparotomy incisions, separation of the wound is usual within a few days after surgery, while primary healing without separation is seen in small wounds, such as closure of plastic incision of the face, in which no tension is required to approximate the tissue.

Further investigation is needed in the selection of the location of the wound, nature of the wound, method of application, and tissue adhesives. These studies, in addition to the basic study of the fate of the implanted polymer, must be done before unlimited clinical use.—We are, etc.,

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Fibrin Degradation Products

SIR,—With regard to the paper by Dr. A. H. Henderson and others on fibrin degradation products in pre-eclamptic toxæmia and eclampsia (5 September, p. 545) my own results (1 January, p. 175) happily, and despite the differing technique of estimation, are broadly in agreement.

The outstanding problem of toxæmia is to relate the low grade intravascular coagulation to the hypertension. Is the syndrome primarily the result of intravascular coagulation

resulting from placental degeneration with fibrin deposition in the glomeruli producing renal hypertension? Or does hypertension of any type in association with the marked physiological inhibition of fibrinolysis that is characteristic of pregnancy engender via vascular damage a low-grade intravascular coagulation, particularly in the kidney? Or is it that both intravascular coagulation and renin production are centred on placental ischaemia?

I have some results from the study of radioactive fibrinogen catabolism in pregnant rabbits in which "toxæmia" has been induced by placental ischaemia by the method of Berger.¹ In the twelve hours after induction of ischaemia a variable degree of intravascular coagulation occurs in all animals. If during this phase fibrinolysis is further inhibited death may occur from pulmonary embolism or on occasion renal cortical necrosis may be induced. Though accumulation of radioactive fibrin can be demonstrated in the kidneys light microscope changes are limited to a little fibrin in the glomerular capillaries. If the placental ischaemia is mild and the fetuses remain in situ, hypertension follows and this has been shown by Berger and Cavanagh² to be the result of renin production from the placenta. If, however, placental abruption occurs, a shock syndrome ensues. In either event there is more fibrin at the site of placental damage than in the kidney, and I therefore agree with Dr. Henderson and colleagues that the fibrin degradation products come mainly from this source.

In support of the second possibility there are several methods of inducing hypertension in which there is secondary accumulation of fibrin in the glomeruli.³ These include the administration of salt and D.O.C.A. to rats, or of renin in addition to D.O.C.A., or of progesterone to pregnant rats, or progesterone in high dosage to rabbits. In all of these methods it is my experience so far that hypertension precedes intravascular coagulation. Possibly hypertension distends the glomerular capillaries so that there is exudation of fibrin and when pregnancy is superimposed, with loss of fibrinolysis, fibrin stasis actually occurs within the capillaries. This type of response would certainly explain the early occurrence of toxæmia in patients with pre-existing renal disease. However, it has been reported that in the sheep hypertension is not essential to the development of glomerular abnormalities.⁴

The theory that uteroplacental ischaemia¹ causes both intravascular coagulation and renin production would seem best to fit the facts, but it is curious that, as in the rabbit experiments, there is said not to be in the human situation a spectrum of changes from abruptio placentae to toxæmia.—I am, etc.,

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Diabetes and the Driving Licence

SIR,—Dr. P. J. O'Connell (12 September, p. 646) wonders what advice to give to diabetics when they apply for a driving licence and advises them not to disclose to their motor insurers that they suffer from diabetes, unless specifically asked.

Diabetics controlled by diet alone run no risk of hypoglycaemia and, provided they are otherwise fit, do not have to declare their diabetes. However, questions 6 (a) (ii): "Do you suffer from sudden attacks of disabling giddiness or fainting?" and question 6 (e): "Are you suffering from any other disease or disability which could be likely to cause the driving of a motor vehicle by you to be a source of danger to the public?" require special consideration by diabetics.

The Medical Advisory Committee of the British Diabetic Association considers that the insulin- or tablet-taking diabetic may answer "no" to these questions, provided the diabetic condition is stable and well controlled; the symptoms of hypoglycaemia are readily recognized; and sugar or glucose is always carried in the car or on the person, and is taken promptly should hypoglycaemic symptoms occur.

Of 300,000 known diabetics in the United Kingdom, about half are potential car-drivers, and their medical fitness to drive is the concern of medical practitioners. The great majority are well-controlled and, provided they take their meals regularly and do not engage in unaccustomed physical exertion, are not subject to attacks of hypoglycaemia. They are perfectly capable of maintaining proper control of a motor car. There remains a small number of patients, chiefly young diabetics in whom control is difficult and who often suffer from hypoglycaemia and its consequences, and some whose hypoglycaemia comes on suddenly, and those who do not readily recognize the symptoms. These diabetics cannot answer "no" to the questions 6 (a) (ii) and 6 (e) and should not drive a motor-car.

The Medical Advisory Committee of the British Diabetic Association therefore asks all medical practitioners looking after diabetics taking insulin or tablets and driving motor-cars, or riding motor or pedal cycles, to make certain that their patients understand the possible causes of hypoglycaemia and how they can be prevented. The Committee especially hopes that, if a doctor knows that a patient does not recognize the onset of symptoms of hypoglycaemia, he will advise his patient not to apply for a driving licence. Further, diabetics on insulin or tablets should not be employed driving public vehicles (buses or coaches), and they should certainly not drive passenger trains or pilot air-liners. The diabetic who develops complications such as cataracts, retinopathy, or severe vascular disease should be advised to give up driving.

Finally, Dr. O'Connell advises his diabetics not to disclose to their motor insurance societies that they suffer from diabetes, unless specifically asked. The British Diabetic Association advises all diabetics, irrespective of the treatment they are receiving, to declare their diabetes to the insurance company. If a diabetic decides not to tell the insurance company about his diabetes and has an accident it is possible that some insurance companies will repudiate the