Insulin Sensitivity in Schizophrenia

SIR,—We read with interest the paper by Dr. R. Hunter and his colleagues (21 February, p. 465) concerning impaired glucose tolerance: a late effect of insulin shock treatment in schizophrenic patients. We have been interested in this problem and investigated the insulin tolerance test in chronic schizophrenic patients.

Four men and four women, age range 43 to 76, were all tested with a standard dose of insulin 0.1 units/kg. All had received insulin shock treatment from 13 to 25 years previously, the maximum dose used ranging from 120 to 300 units of insulin a day. Using the dextran-charcoal radio immunoassay insulin antibodies were not detected in any patient and the fasting plasma insulin was normal between 2.0 and 10.4 $\mu\text{g./ml.}$ One patient, an obese 69-year-old woman, had unrecognized diabetes with a fasting blood sugar of 332 mg./100 ml. The insulin sensitivity of the eight patients expressed as the logarithmic rate constant of blood glucose fall ranged from 0.24 to 5.3; mean 3.3. This compares with a mean insulin tolerance to 6.23 ± 1.87 (S.D.) in 51 normal individuals and 3.9 ± 1.42 in 27 non-obese maturity onset diabetics studied in the same way.

This finding of a slight degree of insulin insensitivity in schizophrenics long after insulin therapy and in the absence of detectable insulin antibodies supports the conclusions reached by Dr. Hunter and his colleagues that there may be a diminished tissue responsiveness to insulin associated with impaired glucose tolerance in these patients.—We are, etc.,

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Postural Hypotension after Guanacine

SIR,—We would like to record a side-effect which we have observed during a clinical trial of guanacine (Leron; Bayer 1464) in the treatment of hypertension. Guanacine is a sympatholytic agent with limited post-ganglionic sympathetic blocking action¹ and prolonged catecholamine depletion.

An Indian male, aged 45 years, suffering from diabetes mellitus, hypertension, and a previous right hemiparesis was given guanacine in place of guanethidine because giddiness, particularly on standing during hot weather, had developed. After 6 months on 135 mg. daily of guanacine severe postural hypotension ensued. The blood pressure was 130/100 lying down and unrecordable on standing. This lasted for 3 weeks despite measures to restore the B.P. with Effortil ((1-3-hydroxyphenyl)-1-hydroxy-2-ethylamino-ethane HCl) and fludrocortisone.

We have culled from the literature 13 cases of persistent orthostatic hypotension after treatment with guanacine.²⁻⁴ It appears from our case and the literature that the hypotensive phenomenon was so severe that the patients could no longer stand and had to stay in bed. As no other drug was given in our trial guanacine was clearly the cause of the postural hypotension.

A detailed report of our clinical experiences with guanacine in the treatment of hypertension will be published in a separate report.⁵—We are, etc.,

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Silicone and Hydrocortisone

SIR,—I read the article by Dr. Mary Corbett and others (3 January, p. 24) with interest. It confirms several of my own observations, most importantly that the silicone oil is innocuous and there is satisfactory absence of side-effects. It gives a better response in joints that have bony congruity; clearly it acts as a simple lubricant and cannot overcome a block to movement produced by large bony excrescences. In the osteoarthritic knee I now confine its use to patello-femoral osteoarthritis, where it can help by floating the sesamoid off the femoral condyle.

In a large series of treated joints I have found that the average length of time the material takes to absorb is four months. It is interesting that at three months the silicone shows the best figures for improvement in the rheumatoid joint. In order to improve on the length of time that the silicone oil is retained and to avoid pooling in recent months I have been working with an oil of much higher centistoke value. The results will be published in due course.

Hydrocortisone is therapeutically one of the most potent of the intra-articular injections, and if there is nothing to choose between hydrocortisone and silicone then the latter may well prove to be the safer material, for we have yet to see a neuropathic joint as a consequence of silicone injection.—I am, etc.,

London W.1.

B. HELAL.

Electrocardiography and the Family Doctor

SIR,—Dr. D. Short's article (28 February, p. 560) causes me considerable concern, in the first instance because of the apparent yet perturbing logic of his argument and thesis, and in the second instance because I feel almost guilty in defending my own vulnerable position as a family doctor who possesses an E.C.G. machine and is under the impression that it is a useful aid to diagnosis in general practice.

The title of his article should have been "The Diagnosis of Ischaemic Heart Disease and the Family Doctor," as it is with this that 99.9% of the article is concerned.

Having declared my own vested interest, I find his views depressing and disturbing. Dr. Short may agree that the E.C.G. machine is after all only an aid, albeit an important one, to diagnosis, but no more nor less, and a carefully taken history and examination still remain a vital part of the eventual assessment. The family doctor may well be less able than an experienced cardiologist in interpreting the cardiogram, but surely this is more than compensated for by the deeper and wider knowledge of the patient, his environment, and family history?

The cardiologists can't have it all their own way and insist that the cardiogram is only an aid to diagnosis when family doctors request a hospital-based E.C.G. service, and yet when the family doctor provides his own E.C.G. machine warn him that it can be a misleading and therefore dangerous diagnostic tool.

The cardiograms in our practice are reported independently by two partners, with a full clinical history available, and if there is any important disagreement we ask for help from a consultant colleague. With all the controversy surrounding the management of the acute coronary infarct it is a bold man who would put his neck on the block. I am prepared to be persuaded by rational, statistically based argument that what Dr. Short advocates is correct, but at the moment I fear that his arguments are neither rational nor statistically based. In this rural practice of three partners my own impression, unsubstantiated statistically, is that the acquisition of an E.C.G. machine has improved our clinical efficiency. It may be that in the long term its greatest use in general practice is in the prompt diagnosis of cardiac arrhythmias.—I am, etc.,

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IAN CAPSTICK.

Spasm and Secretion in Asthma

SIR,—I was rather surprised by the letter of Dr. E. F. G. James (7 March, p. 631) where he stated "... when isoprenaline is applied to the bronchial mucosa it inhibits bronchial secretions, whereas salbutamol enhances them." This is, indeed, a very important statement if it be true, but it is not clear to me whether it is a statement of opinion or whether there is some published work on this subject. If there is, I would be very interested to have the reference, otherwise I view the statement with some scepticism. He goes on to suggest that some of the deaths attributed to pressurized aerosols may be due to plugging of bronchi with mucus from the local action of isoprenaline on bronchial secretion.

I understand that there was no significant increase in deaths associated with the use of these aerosols in America and that the chief one used contained a mixture of isoprenaline and phenylephrine.

The final paragraph of the letter suggests that some asthmatics do not have excessive mucus and they will be helped by isoprenaline—which, he states, inhibits bronchial secretion. I am afraid I do not understand this, or the site or nature of this secretion.—I am, etc.,

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