since other organisms may overgrow the streptococci if the swabs are not plated at once. Tubes containing uncoagulated sterile ox serum were inoculated with the same group of organisms to see if it was likely that E.O.S.S. might support growth. All the tubes gave visible growth except that containing *Str. pyogenes*, subculture of which showed that only slow multiplication had occurred.

Conclusions

In our opinion the use of plain cotton-wool swabs for the isolation of bacteria should no longer be considered as good practice. Charcoal-coated cotton-wool and alginate-wool swabs are good enough only when such swabs are plated within a few hours of collection. Serum-coated swabs preserve the viability of *Str. pyogenes* but permit overgrowth by other organisms. We therefore recommend that swabs soaked in 20% bovine albumin (fraction V) and sterilized by steam should be used. Bovine albumin is believed to be free from antiviral properties and is a constituent of virus transport and tissue culture maintenance media.

It was noted that serum swabs were absorbent, whereas albumin swabs were non-absorbent, but whether absorbency is an advantage or a disadvantage is not known.

We propose to continue to use charcoal-coated swabs with Stuart's (1956) transport medium for material suspected of containing *Neisseria gonorrhoeae*; gonorrhoea is not common enough in the Winchester area for a test of the ability of albumin swabs to preserve the gonococcus to be carried out. Though the use of charcoal swabs with Stuart's medium for all bacteriological swabs is an effective procedure it is too cumbersome for routine use.

We are grateful to Armour Pharmaceutical Co. Ltd. for supplying a solution of bovine albumin (fraction V) without azide, and to Stayne Laboratories Ltd. and Medical Wire and Equipment Co. (Bath) Ltd. for supplies of serum swabs sterilized by ethylene oxide.

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Preliminary Communications

Potential of Insulin Action in Normal Subjects by a Pituitary Polypeptide

British Medical Journal, 1969, 3, 451-452

Summary: A pituitary polypeptide, acceleratory polypeptide-growth hormone (ACG), has been found to increase the sensitivity of fasting normal people to intravenous insulin. It is suggested that diminished activity of this polypeptide—or increased activity of another polypeptide, inhibitory polypeptide-growth hormone (ING)—may have a role in the genesis of diabetes mellitus.

INTRODUCTION

The hypoglycaemic effect of a pituitary polypeptide (ACG)¹ produced by hydrolysis of growth hormone in diabetes mellitus has been reported (Armstrong *et al.*, 1969). It was suggested that the mechanism of this effect was the reversal of resistance to the endogenous insulin present in the patients studied.

This communication reports the influence of ACG on the degree and duration of hypoglycaemia induced in six normal volunteers by the intravenous injection of insulin.

PRESENT INVESTIGATION

Techniques.—The preparation of ACG and the blood glucose estimations (reported to the nearest 5 mg./100 ml.) were undertaken as described in the earlier report. The six volunteers, all of whom gave informed consent, ranged in age from 19 to 32 years. None was diabetic or had a family history of diabetes. All were of normal weight.

¹ ACG stands for acceleratory polypeptide-growth hormone. Experimentally it is capable of accelerating glucose uptake and fatty acid synthesis in various tissues. It may be derived by either non-specific or specific hydrolysis of purified growth hormones.

Experimental.—All volunteers were fasted for 18 hours previous to the study. This duration of fasting was chosen to induce the regulatory mechanisms which maintain blood glucose levels in the face of a pronounced depletion of liver glycogen. Among these mechanisms the anti-insulin effect of growth hormone might be expected to play a part, since fasting has been shown to increase growth hormone levels in plasma (Glick *et al.*, 1965). The blood glucose level was followed for 30 minutes to establish a baseline, and at zero time in the first experiment (series A, see Table) soluble insulin 0.025 unit/kg. body weight (1.4 to 1.6 units) was administered intravenously. Blood glucose was then measured at 10-minute intervals for 90 minutes. In a second experiment (series B, see Table), following an identical technique, insulin and ACG 1 mg. were administered simultaneously and blood glucose was followed for 90 minutes. The low dose of insulin was chosen, as it was thought that the standard dose of 0.1 unit/kg. + ACG might produce severe hypoglycaemia.

RESULTS

The Table shows the fall in blood glucose levels after insulin alone (A) and insulin with ACG (B) in each experiment, together with the mean changes. The Chart depicts the mean changes in blood glucose levels for the whole group.

Fall in Blood Glucose (mg./100 ml.) From Fasting Level

Volunteer:	1 (F. 24)		2 (F. 19)		3 (F. 21)		4 (M. 32)		5 (M. 20)		6 (F. 23)		Mean	
	A	B	A	B	A	B	A	B	A	B	A	B	A	B
10 min.	5	10	5	0	0	10	0	15	5	0	10	15	4.2	8.3
20 min.	20	35	15	35	10	20	25	40	20	25	30	20	20.0	29.2
30 min.	25	45	30	40	20	35	25	45	5	30	30	40	22.5	39.1
40 min.	25	30	0	30	20	25	25	40	5	30	20	10	15.8	27.5
50 min.	10	20	0	5	0	20	10	15	0	20	5	0	4.1	13.3
60 min.	0	20	0	0	0	10	0	15	5	15	5	0	1.7	10.0
70 min.	0	10	0	0	0	5	0	10	0	15	0	0	0	8.3
80 min.	0	10	0	0	0	5	0	10	0	15	0	0	0	6.7
90 min.	0	0	0	0	0	5	0	5	0	0	0	0	0	1.7

In all instances the fall in blood glucose induced by this small dose of insulin was significantly increased by the administration of ACG, the change at 30 minutes being significant at less than the 1% level, and in five of the six volunteers the duration of the hypoglycaemia was prolonged. The difference in means at 70 and 80 minutes is significant at the 5% level.

DISCUSSION

The results show that under the conditions of the experiment—that is, prolonged fasting—ACG induces increased sensitivity of normal people to small amounts of intravenous insulin as measured by changes in blood glucose.

These conditions were deliberately chosen, since it was postulated that with the increased blood growth hormone levels induced by fasting ING² activity might be increased. Thus it would be expected that the effect of ACG in reversing ING inhibition might be more readily shown.

The results suggest that ING:ACG relationships may have a physiological role in glucose homeostasis, insulin acting to promote glucose entry into muscle and fat, and facilitating

glycogen synthesis in these tissues and the liver, while the relative ING:ACG activity exerts a modulating effect on the utilization of intracellular glucose and hence on insulin sensitivity. Variations in insulin sensitivity have been invoked as factors in determining glucose tolerance and the genesis of diabetes (Cerasi and Luft, 1967; Martin *et al.*, 1968), and the increased plasma insulin response after oral glucose in many diabetics may be a reflection of ING-induced resistance to insulin in these patients. The evidence reported here, suggesting the presence of ING in normal humans, makes more cogent the hypothesis that increased activity of this polypeptide or diminished activity of ACG might have a role in the genesis of diabetes mellitus, acting in the manner of the addition and compensation factors suggested by Cerasi and Luft (1967).

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²ING stands for inhibitory polypeptide-growth hormone. Experimentally it is capable of inhibiting glucose uptake and fatty acid synthesis and indirectly causing the mobilization of unesterified fatty acids from peripheral fat. It may be derived by either non-specific or specific hydrolysis of purified growth hormones.

Medical Memoranda

Renal Allograft with Massive Perirenal Accumulation of Lymph

British Medical Journal, 1969, 3, 452-453

Complications of renal transplantation are protean (Starzl, 1964; Calne, 1967). Those associated with mechanical problems are of particular surgical interest. Recently we encountered a patient with chronic renal allograft rejection and the nephrotic syndrome (Harlan *et al.*, 1967). In addition a large amount of lymph had accumulated at the region of the transplant to produce the iliac compression syndrome. Review of the literature shows that this complication has received only passing mention (Hume, 1968).

CASE HISTORY

A 17-year-old Negro youth of blood group B, Rh-positive, presented in March 1966 with chronic glomerulonephritis and the nephrotic syndrome. By December 1967 he had to be supported by dialysis. Bilateral nephrectomy on 7 March 1968 confirmed the diagnosis.

On 1 May 1968 a kidney from a 40-year-old male Negro of blood group O, Rh-positive, who had died of brain injury, was transplanted into the patient. Lymphocyte antigen typing showed a C minus matching grade between donor and recipient (Lee *et al.*, 1967). The other donor kidney, weighing 290 g., was macroscopically and microscopically normal. Immunosuppression with prednisolone and azathioprine was given at the usual dosage (Straffon *et al.*, 1967). Local irradiation to the graft, with a total dosage of 900 rads, was given.

The immediate postoperative course was marked by acute tubular necrosis of two weeks' duration and urinary infection with *Escherichia coli*, which promptly subsided with kanamycin therapy. Renal function progressively improved during the first month—the serum creatinine decreased to 1.6 mg./100 ml., the 24-hour urine protein excretion stayed around 1 g., and the blood pressure fell to 160/80.

During the fifth postoperative week urinary infection with *E. coli* recurred. Proteinuria increased to 20 g./24 hours. An intravenous urogram showed enlargement of the graft. A renal arteriogram showed that the distal arterial branches were spaced far apart, a finding suggestive of intrarenal swelling or interstitial oedema (Hall and Holley, 1969). The urinary infection again responded to kanamycin treatment. Prednisolone was increased to 400 mg. daily for five days, and a course of actinomycin D was given because of