Immune ascitic anti-Ippy†	+	+	+
Monoclonal antibodies‡			
2093–0004	+	+	+
2054–0006	+	+	+
2074–0007	+	+	+
2129–0018	+	+	+

*Convalescent serum from Lassa fever patient (provided by CDC), at working dilution 1/32.

†Mouse immune ascitic fluid against Ippy virus prepared in Bangui (no 272), at working dilution = 1/40.

‡Lassa/Mozambique nucleocapsid specificity, 2093–0004 at working dilution 1/20; Lassa/Mozambique nucleocapsid specificity, 2054–0006 at working dilution 1/40; Lassa glycoprotein specificity, 2074–0007 at working dilution 1/20; Lassa/Mozambique nucleocapsid specificity, 2129–0018 at working dilution 1/20.

Dr Swanepoel (March 16, p 639) has reported that the prototype strain of Ippy virus, isolated in 1970, is a member of the Lassa fever complex. The "Ippy" strains studied by us and by Swanepoel et al seem to differ in that our strain reacted with monoclonal antibody 2093–0004 while Swaneopoel's did not (2093–0004 is the same as 5293–4). These two strains are identified as Ippy viruses by classical tests but the use of monoclonal antibodies may now be revealing some differences in epitopes.

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GLYCOSYLATED HAEMOGLOBIN IN IRON-DEFICIENCY ANAEMIA

SIR,—A 1984 *Lancet* editorial[1] discusses interference with the measurement of glycosylated haemoglobin used to evaluate long-term control of blood glucose,[2] and cites as an example Brooks and colleagues' report of the increased glycosylation of haemoglobin, as measured by a cation-exchange microcolumn method, in association with iron-deficiency anaemia.[3] However, the ion exchange chromatography method yields peaks that are likely to be contaminated by non-glycosylated haemoglobin.[4] Affinity chromatography is thought to be more specific,[5] and this is the method we have used[6] in our attempt to confirm Brooks' findings.

We selected fourteen patients with iron-deficiency anaemia. Four patients were male and ten female, ages ranged from 27 to 89 years (mean 54 y), and all were non-diabetic. The mean glycosylated Hb (6·9±0·9% SD) was not significantly different from normal (7·0±1·7%). Only one iron-deficient patient had a value (8·8%) just above the normal range of 5·3–8·6%. All patients responded to oral iron with significant increases in haematological indices but their mean glycosylated haemoglobin values did not change (6·5±1·7%).

The high levels of HbA_1 reported with cation-exchange chromatography may be due to post-translational modifications of haemoglobin other than glycosylation in iron deficiency, the and glucose residues on globin chains only.

We conclude that when affinity gel separation is used iron-deficiency anaemia does not produce high glycosylated haemoglobin values.

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REGRESSION OF METASTATIC VIPOMA WITH SOMATOSTATIN ANALOGUE SMS 201–995

SIR,—Vasoactive intestinal polypeptide (VIP) has been implicated as the cause of the severe watery diarrhoea of Verner-Morrison syndrome and raised blood levels are found in patients with bronchogenic carcinoma, phaeochromocytoma, ganglioneuroblastoma, and pancreatic tumours. Early diagnosis and resection of a vipoma may be curative, but inoperable or metastatic vipomas are difficult to treat. Intravenous somatostatin suppresses VIP secretion[1] but its plasma half-life is only 1·1–3·0 min.[2] SMS 201–995 is a synthetic octapeptide with a longer half-life which can be given by subcutaneous injection; it also suppresses VIP levels. We report a case of a metastatic vipoma treated with SMS 201–995 50 μg once daily.

A 75-year-old woman with a 9 year history of watery diarrhoea associated with hypokalaemia and mild hyperglycaemia was found to have raised plasma VIP levels. The primary tumour was removed

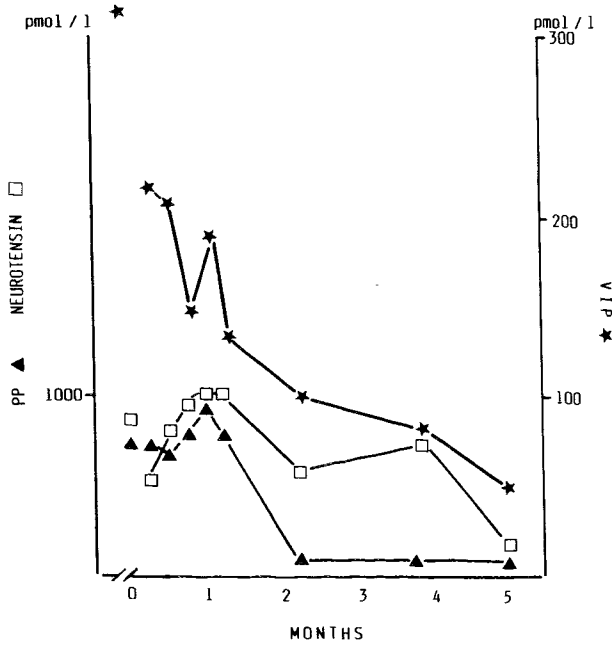

Fig 1—Neurotensin, VIP and PP levels before and during treatment with SMS 201–995.

Normal ranges: VIP <30 pmol/l; PP <300 pmol/l; neurotensin <200 pmo

By June, 1984, she was having watery diarrhoea up to fifteen times a day passing 1·5–2 litres, had lost weight, and was hypokalaemic despite potassium supplementation. Her plasma VIP, pancreatic polypeptide (PP), and neurotensin concentration were raised; gastrin and glucagon levels were normal.

SMS 201–995 50 μg once daily produced a progressive improvement in hormone levels (fig 1) accompanied by a weight gain of 5 kg, reduced stool frequency and volume, and correction of hypokalaemia. There have been no side-effects, apart from a reduced insulin requirement. Computerised axial tomography (CT) after 5 months of treatment revealed a reduction in the number, size, and contrast enhancement of the liver secondaries (fig 2).

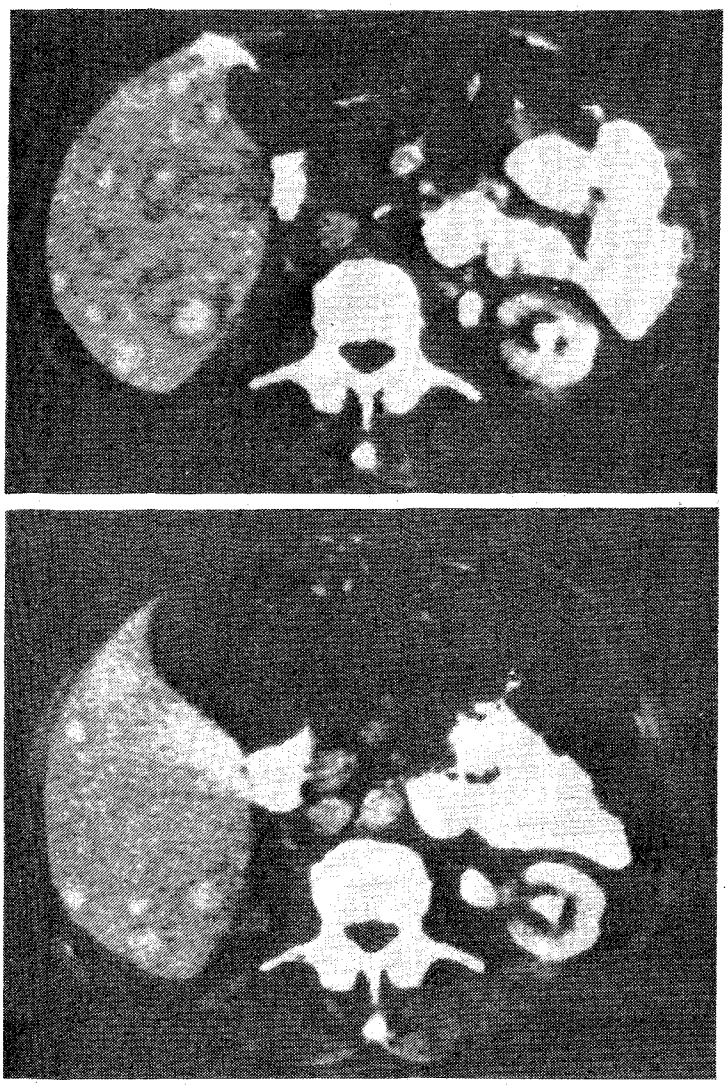

Fig 2—CT scan appearances before (upper) and after (lower) SMS 201–995 treatment.

Vipomas are often accompanied by increases in neurotensin-like immunoreactivity[3] and pancreatic polypeptide levels,[4] as in our patient. Her main problem was profound secretory diarrhoea, and, though the specific peptide responsible for this diarrhoea is not known, VIP remains the most likely candidate.

There has been considerable interest in the use of SMS 201–995 in the treatment of tumours and diarrhoea, and some success has been reported in the treatment of vipomas,[5-10] including one case of regression of liver secondaries[7] and a response without tumour regression.[9] Our patient has improved considerably on once-daily treatment (given at the same time as the insulin by the district nurse) with 50 μg of SMS 201–995, with improvement of symptoms, CT scan appearances, and VIP, PP, and neurotensin levels. The CT evidence supports a direct effect of SMS 201–995 on the tumour as a mechanism for the clinical and biochemical improvement. This patient's tumour has been successfully controlled with once-daily

We thank Dr R. Shentall of Sandoz for the supply of SMS 201–995. The peptide assays were done at the Royal Postgraduate Medical School, London, in Prof S. R. Bloom's department.

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RENAL FUNCTION IN DIABETICS

SIR,—*Lancet* correspondence about renal function in diabetics (Jan 5, p 53; Feb 23, p 466) prompted us to review our research. We have investigated the prevalence of diabetic nephropathy in a general diabetic population (G; n=843) and amongst diabetics attending a special eye clinic (R; n=115). Group G was drawn from a well-defined epidemiological population and group R consists of patients with serious diabetic retinopathy (maculopathy or preproliferative or proliferative changes, confirmed by an ophthalmologist). Both groups contained non-insulin-dependent and insulin-dependent diabetics.

Urine samples were tested for proteinuria ('Albustix') and patients were asked to submit a timed overnight urine collection to measure albumin excretion rate (AER). However, as the response rate for these samples was poor, a sample from a midstream urine specimen passed at the clinic was taken and the random urinary albumin/creatinine (albumin in mg/l, creatinine in mmol/l) ratio (RA/C) measured. A micro-ELISA technique[1] was used to measure urinary albumin and the Jaffe method for urinary creatinine. In our laboratory the upper limits of normal are 7·5 μg/min for AER and 1·9 for RA/C (mean +2SD after log transformation in 114 non-diabetic controls). Infected urine specimens are excluded.

As expected, the prevalence of proteinuria was greater in group R than in group G (see table). Microalbuminuria was also commoner. This is consistent with the similar microangiopathic basis of retinopathy and nephropathy.

Our findings confirm that macroalbuminuria and microalbuminuria are more common in diabetics with serious retinopathy. Since, according to our figures, 40% of patients with a treatable blinding condition will be missed, there is little case for using microalbuminuria to identify these diabetics with retinopathy. Also, its use to predict future retinopathy remains

ALBUMINURIA IN TWO DIABETIC POPULATIONS

	Group R	Group G
Albustix positive	18/115 (*15·7%*)	50/843 (*5·9%*)
Raised AER	33/55 (*60%*)	125/447 (*28·0%*)
Raised RA/C	44/68 (*65%*)	212/551 (*38·5%*)