At radical nephrectomy with segmental resection of the caval wall including the cancerous lesion, the 150 g tumour specimen showed extensive necrosis. The patient is well without signs of recurrence or metastasis.

Neither patient had side-effects after MC except for temporary anorexia, leucocytosis, and raised serum lactic dehydrogenase shortly after the infusion. Antitumour effects were clearly demonstrated radiologically and histologically. In the first patient pain from bone metastasis was well controlled. Our preliminary results suggest that arterial infusion of MC microcapsules is effective in cancer patients with an invasive primary lesion or a localised metastatic lesion where selective catheterisation is possible.

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RAPID APPEARANCE OF Hb_sAg AFTER INOCULATION

SIR,—The incubation period of hepatitis B is generally thought to be several weeks. By sensitive techniques, hepatitis B surface antigen (HB_sAg) has been detected as early as six days after parenteral exposure.[1] We describe here a patient whose serum was positive for HB_sAg at least from the third day after accidental transfusion with blood containing HB_sAg.

The patient was a 63-year-old man who received several units of blood during open-heart surgery. One of the units contained blood positive for HB_sAg (titre 1/13 000 by Finnish Red Cross radioimmunoassay[2] and hepatitis B e antigen (HB_eAg).

APPEARANCE OF HEPATITIS B ANTIGENS AND ANTIBODIES

Days after transfusion	HB_sAg titre	HB_eAg*	Anti-HB_e*	Anti-HB_s†
−3	..	..	..	..
0	..	..	..	..
3	210	—	—	±
8	900	—	—	±
11	2300	—	—	±
18	23000	+	—	±
31	160000	+	—	—
44				
51–78	..	..	..	..
111	5	—	—	—

*By agar gel diffusion;
†By 'Ausab' radioimmunoassay (Abbott).
Patient had raised transaminases without symptoms on day 44 and clinical hepatitis between days 51 and 78.

The donor was within the incubation period of hepatitis B; his serum had been negative for HB_sAg at the previous blood donation about five months earlier and hepatitis B developed in him and in the recipient of the blood at the same time. The heart operation had no immediate complications and the patient recovered normally. Several serum-specimens were taken within four weeks of the transfusion. The first, taken on the third day, was already positive for HB_sAg (see table). The titre of HB_sAg increased rapidly up to day 18 (average increase 37% per day) after which the increase was somewhat slower (16% per day between days 18 and 31). The patient had clinical hepatitis between days 51 and 78 from which period no blood specimens were unfortunately available. Anti-HB_s was detected (weakly positive results by 'Ausab') in the first four samples. The other transfused blood units may have contain anti-HB_s. The patient recovered from the disease, but h serum was still positive for HB_sAg one month after recover

This case-report shows that HB_sAg may be continuousl present in serum after massive inoculation with hepatitis-B virt and that the titre of HB_sAg may increase rapidly during th first week after exposure. On the other hand, no symptoms wer observed during the first four weeks when the HB_sAg titre in creased rapidly, whereas the patient developed hepatitis abou one month later. This strongly favours the view that mecha isms other than a direct cytolytic effect of virus replication a mainly responsible for liver damage during hepatitis B.

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SCHIZOPHRENIA AND DEGRADATION OF ENDORPHINS IN CEREBROSPINAL FLUID

SIR,—The role of endorphins in mental disorders is beir studied extensively. Several investigators have attempted relate endorphin levels in cerebrospinal fluid (CSF) to the disorders.[1-6] Dupont et al.[6] reported that a CSF compone was more rapidly inactivated in chronic schizophrenics than controls. This component, designated as enkephalin-lil material, was measured by ^3H-naloxone displacement and it c migrated with methionine(met)-enkephalin or leucine-enk phalin after gel permeation chromatography. The authors su gested that decreased levels of enkephalin-like material in CS of schizophrenics are at least partly due to enhanced inactiv tion of endorphins *in situ*.

In order to extend their observation to opioid peptides *p se* we have investigated degradation in CSF of schizophreni and controls of synthetic β-endorphin, which is stable to pr teolytic degradation and met-enkephalin, a susceptible pe tide.[7,8]

We investigated 9 patients with chronic or chronic relapsir schizophrenic psychoses. All had been on maintenance thera with neuroleptic drugs and had been admitted because th were at least partly resistant to these drugs. Non-psychiatr patients with various neurological diseases served as control CSF samples were obtained by lumbar puncture and immed ately frozen. Samples were stored at −20°C.

β-endorphin or met-enkephalin (Organon International B Oss, Netherlands) were added to the CSF samples. Samples incubation medium were taken at the start and at the end incubation (5 h, 37°C) and were subsequently heated for 1 min at 95°C. The amounts of β-endorphin and met-enkephal were assessed by radioimmunoassay.[9] A decrease in immun

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INCUBATION OF β-ENDORPHIN AND MET-ENKEPHALIN WITH CSF OF SCHIZOPHRENICS (N=9) AND CONTROLS (N=9)

Peptide	Patients	Amount +SEM (ng/ml)	
		At 0 h	At 5 h
-endorphin	Controls	6·1±0·5	5·5±0·8
	Schizophrenics	6·9±0·4	7·2±0·3
Met-enkephalin	Controls	26·8±0·8	26·0±1·0
	Schizophrenics	27·2±0·7	25·0±0·7

reactivity can be considered a reliable measure for degradation because in both radioimmunoassays known degradation products of β-endorphin—e.g.,α-endorphin, γ-endorphin,[10] and their des-tyrosine analogues (unpublished observation)—and met-enkephalin do not cross-react to a significant extent.[9]

There was no significant decrease of β-endorphin and met-enkephalin after 5 h incubation with CSF obtained from schizophrenics or from controls (Student paired *t*-test). β-endorphin and related peptide fragments are thus not degraded by proteolytic enzyme activities in CSF in vitro and the metabolism of these peptides in CSF of schizophrenics and controls is thus no different. We conclude that inactivation of endogenous enkephalin-like material in CSF of schizophrenics, as reported by Dupont et al.,[6] is unlikely to be due to degradation of β-endorphin related peptides. In our opinion the enkephalin-like material is distinct from known endorphins and may be subject to other metabolic mechanisms in CSF. Our studies indicate that altered levels of β-endorphin and possibly other endorphins in CSF of schizophrenic patients reflect changes in cellular metabolism and release rather than altered enzymatic activity in CSF.

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CONTINUOUS SUBCUTANEOUS INSULIN INFUSION OR SUBCUTANEOUS INSULIN INJECTIONS

SIR,—Continuous subcutaneous infusion (CSI) is a new means of administering insulin for the control of diabetes, and two papers[1,2] and an editorial (June 16) in *The Lancet* have discussed whether better control might help to prevent the complications of diabetics. Whilst CSI may eventually lead to miniaturisation and an implantable infusion device, we suggest that other more conventional approaches have not yet been overtaken. Several features of the studies may have biased the results in favour of CSI. The subcutaneous injection regimens employed[1,2] were often not those held in highest regard (e.g., twice daily soluble insulin) and no special attempt to optimise them was described. The order of the two observational days was not randomised, so conventional treatment was always studied first, on the first day in hospital, without time for equilibrium to the emotional, exertional and dietary changes to be achieved.

The distinction between the basal insulin replacement and meal insulin requirements provided by the infusion regimes can also be provided by combinations of long-acting and short-acting subcutaneous insulins. Thus we have, over a mean period of 10 months, treated 22 insulin-dependent diabetics with a regimen based on 'Ultratard MC' insulin providing the basal replacement and twice daily 'Actrapid MC' insulin to cover meals.[3] With control monitored by monthly series of four blood-samples per day, taken at home with an 'Autolet'[4] and transported by collector bottles[5] for laboratory assay, we obtained a mean plasma glucose concentration of all, and therefore unselected, blood samples before breakfast, before lunch, before the evening meal and before bed of 6·1, 5·8, 7·3, and 7·2 mmol/l, respectively (unpublished). Similar results were obtained with home monitoring by Sönksen and Walford and colleagues.[6,7] These concentrations were comparable to, or slightly higher than, the published results of insulin infusions under ideal conditions in hospital,[1,2,8–10] and there is little experience of the degree of control obtained with the infusion regimens long-term.

If patients were being treated with the currently available portable infusion pumps, the introduction of a rational regimen of intermittent injections of subcutaneous insulin might be hailed as an advance, liberating the patients from the need to carry a pump. CSI may have advantages, but their demonstration will depend on future comparison with optimal intermittent subcutaneous insulin injection regimens to find out which provides less variability of blood glucose on a day-to-day basis, lower hæmoglobin A_{1c} easier control during infections and stress, and the greater acceptability to patients.

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LABORATORY TEST FOR KIDNEY FUNCTION—UREA OR CREATININE?

SIR,—The two most common tests of impaired kidney function are the measurement of urea and creatinine in plasma. There seems to be a widespread opinion nowadays that the test for plasma creatinine is the more efficient but we are not aware of experimental data which support this view. We have measured both substances in 280 routine blood-specimens and analysed the results by the standard methods of multiple regression.[1] We excluded those samples whose measured values deviated by more than three standard errors from the values calculated from the regression equation and repeated this manœuvre until no further cases could be excluded.

By this means 10 out of 134 samples from women and 10 out of 146 samples from men were excluded. All 10 women had raised urea and creatinine; 8 of the men had raised urea and creatinine, and in 2 men only one value was high.

On the remaining 260 specimens the regression equations were as follows, for men (n=136):

creatinine (μmol/l)=33·32+8·95 urea (mmol/l) +0·25 age(yr)
with SE±21·1 (r=0·83);

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