in more concentrated form are more successful than dextran 40.

Whether any advantage over the use of bupivacaine alone would justify the known risks of potentially dangerous reactions to dextran remains a matter for future debate when more information is available.

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Reference

1 Loder RE. A local-anaesthetic solution with longer action. Lancet 1960;2:346–7.

Mr Kingsnorth replies as follows:
I agree with Dr Loder that the local anaesthetic agent we used after mixing with dextran 40 was a 0.25% bupivacaine solution in 5% dextran 40. The theoretical possibility that it is the large number of big molecules which retard absorption was explored by Aberg *et al.*, a reference cited in our paper. Tritiated mepivacaine remains in guinea-pig intradermal injection sites almost 50% longer when mixed with 3% dextran and almost twice as long when mixed with 6% dextran. However, in test subjects having tooth anaesthesia neither 3% nor 6% dextran prolongs the duration of soft-tissue anaesthesia.

The logical conclusion is that although the macromolecules of dextran may form a complex with the local anaesthetic molecules (and this is only a supposition) and result in their slow removal from the tissues, it cannot be inferred from this that the local anaesthesia lasts any longer: indeed, Aberg showed that it does not. That a macromolecule of 150 000 should be more effective than one of 40 000 molecular weight in slowing absorption is again conjecture and again implies nothing about local anaesthetic action.

We did not add adrenaline to our solution because of the increased risk of haematoma formation after wound closure (open wounds were infiltrated in the patients quoted in Dr Loder's original papers, presumably after haemostasis had been obtained), a factor known to increase the incidence of recurrent hernias.

In reply to Dr Hughes, reference to the *British Pharmacopoeia* shows that dextran 40 is a 10% solution w/v with average molecular weight 40 000. Dr Loder's studies in 1960 and 1962 used consecutive patients with retrospective controls, and wound infiltration was used after general anaesthesia. To describe them as 'successful' using present-day scientific standards would be hard to justify. I see no value in repeating these studies but rather in carrying out controlled double-blind ones such as ours. I am unaware of other trials being carried out and so are the manufacturers of Marcain (bupivacaine).

We had no allergic reactions to dextran solutions —the only recorded cases have occurred after its intravenous use.

Tinel sign in brachial plexus lesions

Dr Antonio Landi and Mr Stephen Copeland (*Annals*, November 1979, vol. 61, p. 470) are to be congratulated for their description of a clinical examination which 'has not yet been surpassed in accuracy by the more sophisticated investigations of the late 20th century'. The clinical examination of lesions at the cervical spine leaves much to be desired. In our experience modern technology cannot substitute for a good clinical examination.

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Ultrasound in biliary tract and pancreatic disease

We share the enthusiasm of Mr N V Addison (*Annals*, November 1979, vol. 61, p. 448) for grey scale ultrasonography in biliary and pancreatic disease *(1,2)* but disagree regarding some of the aspects of diagnosis and management.

The author suggests that endoscopic retrograde choledochopancreatography (ERCP) is contraindicated (and dangerous) in acute pancreatitis. On the contrary, this investigation is becoming more widely used in patients with pancreatitis whose clinical features suggest gallstone impaction; emergency endoscopic sphincterotomy provides an excellent alternative to urgent surgical drainage *(3)*.

Oral cholecystography is preferable to intravenous cholangiography for the investigation of patients after an attack of pancreatitis. It is safer, less uncomfortable for the patient, gives better opacification of the gallbladder, and usually demonstrates the common duct as well after a fatty meal. Oral cholecystography may indeed be unsuccessful in patients with suspected acute cholecystitis because of failure of absorption of the contrast agent; intravenous cholangiography is then diagnostic if the duct can be seen in the absence of gallbladder opacification. Alternatively, the oral contrast medium may be administered rectally.

In the presence of jaundice tomography of the gallbladder after intravenous infusion of contrast is very accurate in the diagnosis of acute cholecystitis and depends on the demonstration of increased thickness of the gallbladder wall *(4)*.

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References

1 Vallon AG, Lees WR, Cotton PB. Grey-scale ultrasonography in cholestatic jaundice. Gut 1979; 20:51–4.
2 Lees WR, Vallon AG, Denyer ME, Vahl SP, Cotton PB. Prospective study of ultrasonography in chronic pancreatic disease. Br Med J 1979;i: 162–4.