duodenal relaxant may increase the incidence of duodenal ileus after pancreatitis.

As stated in your article the mortality in acute pancreatitis is approximately 25%. Twelve per cent of patients admitted with acute pancreatitis will develop one of the major complications, and when this occurs the mortality may rise to over 50%. Trapnell[2] described 70 patients who developed a major complication. Ten of these had prolonged duodenal ileus, four of them dying.

We have recently treated a 67-year-old man who was admitted to hospital with a pancreatic pseudocyst and copious vomiting two weeks after a severe attack of pancreatitis. He had an epigastric mass and signs of spreading peritonitis and was markedly alkalotic. He continued to deteriorate despite all efforts with conservative treatment, and laparotomy, drainage, and cystogastrostomy were performed. Postoperatively, treatment with glucagon was begun and was continued for seven days. Very high gastric aspirates consistent with duodenal ileus persisted for five days and began to improve only with metoclopramide treatment and after a Gastrografin swallow, which showed some contrast medium reaching the proximal jejunum. Unfortunately he developed further complications and died 10 days after admission. At postmortem the pancreas was completely necrotic. There was extensive abscess formation extending to the right subphrenic space, massive pulmonary embolism, and terminal haemorrhage into the stomach in the absence of demonstrable ulceration. The gall bladder and biliary tree were normal.

It is likely that the role of glucagon will prove to be in reducing the severity of the initial attack of pancreatitis. This should lead to a reduction in the incidence of major complications and therefore further reduce the overall mortality. Duodenal ileus is believed by Trapnell to be a consequence of continuing pancreatic destruction rather than a mechanical obstruction by a swollen and oedematous pancreatic head. If this is so then glucagon may reduce the frequency and severity of continuing pancreatic destruction and consequently of duodenal ileus. In the presence of established duodenal ileus, however, the relaxation of duodenal musculature that glucagon causes may worsen an already dangerous situation.—I am, etc.,

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1 Condon, J. R., Knight, M., and Day, J. L., *British Journal of Surgery*, 1973, **60**, 509.
2 Trapnell, J. E., *Annals of the Royal College of Surgeons of England*, 1971, **49**, 361.

Blood Carboxyhaemoglobin Levels in Smokers

SIR,—In view of the current interest in the carbon monoxide content of cigarette smoke,[1 2] the following results appear to be relevant. We have measured the rise in carboxyhaemoglobin (COHb) in smokers smoking a single low-, intermediate-, or high-nicotine cigarette. The cigarettes used were of familiar size, weight, and density and contained the same blends of tobacco. However, the filter efficiency for nicotine differed in such a way that if the cigarettes were smoked in a standard manner in a smoking machine the mainstream smoke contained 1, 2·1, and 2·4 mg of nicotine in the low-, intermediate-, and high-nicotine cigarettes respectively, but the carbon monoxide yield did not differ. The smokers smoked in their normal manner at rest and all except two of the cigarettes (both intermediate-nicotine) were smoked as the first cigarette of the day. COHb was measured from finger-prick samples before and immediately after smoking by the method of Commins and Lawther.[3] The puffing rate and the amount of nicotine delivered to the smoker from each cigarette were also estimated.

Blood COHb level, Puffing Rate, and Nicotine delivered to Smoker in Subjects smoking low-, high and intermediate-nicotine Cigarettes

	Low-nicotine			Intermediate-nicotine			High-nicotine			Analysis of Variance	
	Mean	S.D.	n	Mean	S.D.	n	Mean	S.D.	n	F	Significance
COHb %											
Before smoking	2·3	1·1	10	2·6	1·5	8	1·9	1·0	10		
After smoking	4·8*†	0·8	10	3·8*	2·0	8	3·1†	1·5	10		
Difference	2·5*†	1·1	10	1·2*	0·7	8	1·1†	1·0	10	3·9710	P<0·05
Puffs/min	1·8	0·7	10	2·2	1·2	8	1·6	0·6	9	1·1547	n.s.
Nicotine delivered to smoker (mg)	1·2†	0·3	12	1·3‡	0·5	8	1·8†‡	0·4	9	5·7538	P<0·01

Symbols refer to significant differences between types of cigarettes: * = low-v. intermediate-nicotine; † = low-v. high-nicotine; ‡ = high-v. intermediate-nicotine (*t* tests; P<0·01 in all cases).

The rise in COHb during smoking was greatest for the low- and least for the high-nicotine cigarettes (see table). This difference was accompanied by a tendency towards a lower puffing rate for the high-nicotine cigarettes, though more nicotine was in fact delivered. Thus the subjects were able to obtain a relatively high dose of nicotine with a smaller increase in COHb from the high- as compared with the low-nicotine cigarettes. The intermediate-nicotine cigarettes occupied an intermediate position. These findings appear to be in general agreement with those of the longer-term study by Russell *et al.*,[2] in which it was shown that cigarette consumption and COHb rise diminished in subjects changing from their usual brand to high-nicotine cigarettes over five-hour periods. In Russell's study, unlike the present one, the carbon monoxide yield of the different types of cigarette differed owing to differences in size and weight of the cigarettes. Nevertheless, our findings agree with the suggestion that a "safer" cigarette might be one which combined a low yield of carbon monoxide and tar with a relatively high yield of nicotine.

We have also followed some short-term changes in the blood level of COHb in a few subjects. The rise while smoking a single cigarette of the subject's own brand in four smokers with different initial levels of COHb is shown in fig. 1. The rate of rise was greatest at the start of the cigarette followed by a levelling off or even a fall of COHb towards the end. This plateau effect may be related to the fact that smokers tend to take fewer puffs towards the end of a cigarette than at the beginning, possibly because the nicotine in the stub becomes more concentrated as the cigarette burns down. The fall in COHb levels immediately after smoking in six smokers at rest (one smoking a small cigar) is shown in fig. 2.

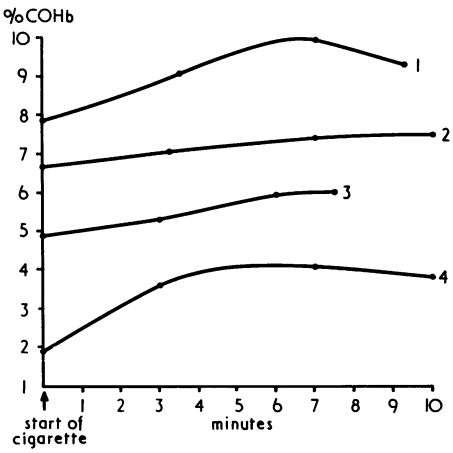

FIG. 1—Changes in blood COHb levels in four subjects while smoking one cigarette.

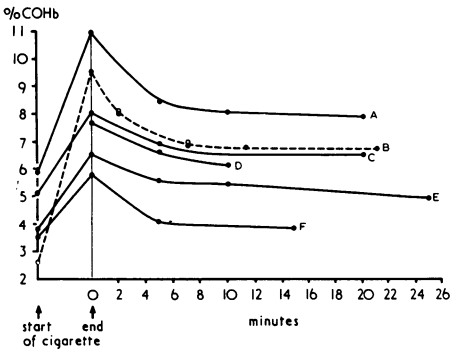

FIG. 2—Change in blood COHb level in six smokers after smoking. Subject B smoked (and inhaled) a small cigar; the other subjects smoked a cigarette. (O = blood samples taken from antecubital vein; all other blood samples taken by finger prick).

In all subjects there was a relatively rapid fall (1-2·5%) of COHb in the first five minutes after smoking, followed by a levelling off to a very slow rate of decline, about 0·5% between five and 25 minutes. The cause of the initial sharp fall of COHb in the first five minutes is not clear. It does not appear to be due to an artefact due to unequal distribution of carbon monoxide in the circulation, since in one subject blood samples taken simultaneously from the right antecubital vein and the left middle finger tip gave identical results. Possibly binding with other molecules such as myoglobin is involved. Extrapolation of the curves in fig. 2 gives a half life for COHb of approximately 2-2½ hours, agreeing with the findings of Russell *et al.*[2]—We are, etc.,

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2 Russell, M. A. H., Wilson, C., Patel, U. A., Cole, P. V., and Feyerabend, C., *British Medical Journal*, 1973, **4**, 512.
3 Commins, B. T., and Lawther, P. J., *British Journal of Industrial Medicine*, 1965, **22**, 139.