

under way several random trials of the two procedures from which it is hoped information on this point will be obtained.

Selective vagotomy implies the division of only the vagal fibres passing to the stomach with deliberate preservation of those fibres passing to other intra-abdominal organs such as the gall-bladder, the pancreas, and the small intestine. It has been argued that the parasympathetic denervation of these other intra-abdominal organs may be responsible for the diarrhoea which occasionally follows vagotomy with a drainage procedure. However, it is not certain yet whether the diarrhoea is caused by the vagal denervation or by the drainage procedure, and there is some experimental evidence in support of each hypothesis.

Different types of selective vagotomy have been advocated which preserve varying proportions of either anterior or posterior vagus or both. It would seem of particular importance to ascertain that any particular pattern of selective vagotomy employed adequately denervates the stomach, thus reducing the gastric acid secretion sufficiently to protect against recurrent ulceration. Such information has not yet been published. Insulin-testing of patients thought to have had total vagotomy has shown a surprisingly high incidence of incomplete vagal denervation, and it may be that a similar or greater incidence of incompleteness may accompany the selective operation.

Pyloroplasty without vagotomy has been performed for chronic benign gastric ulcer on the theoretical basis that some degree of gastric stasis may be a factor in the aetiology of this condition. There have been no reports of pyloroplasty alone for duodenal ulcer, and it seems unlikely that it would be any more successful than gastrojejunostomy alone, which carries a risk of recurrent ulcer probably in the region of 50%. Pyloroplasty alone does nothing to reduce the acid secretion of the stomach, neither does it have the theoretical virtue of gastrojejunostomy that the gastric contents are diverted away from the region of the ulcer. For these reasons it seems unlikely that pyloroplasty without vagotomy will have a place in the treatment of duodenal ulcer.

Retinal "Oedema"

Q.—*What treatment is there for a retinal oedema in a patient aged 70 which is presumably due to congestive cardiac failure, though this is apparently controlled by digitalis and diuretics ?*

A.—The retina, like the brain, has no true extracellular fluid space and is therefore unlikely to collect oedema fluid in the same sense as other tissues. The greyish sheen in the retina which is often referred to as oedema most probably represents a swelling of cellular elements in the retina. The normal iridescent sheen of the retina in children and in young adults is often mistakenly described as oedema when these patients happen to suffer from the nephrotic syndrome, hypertension, or heart failure.

It is not stated whether the 70-year-old patient has hypertension or some other cause of congestive cardiac failure, nor is the distribution of the retinal oedema given. Severe

hypertension might be the explanation of the finding. If the patient is normotensive and the abnormal retinal appearances are confined to the macula area the patient may have a macula dystrophy, for this condition is common in the elderly. Congestive cardiac failure does not usually lead to retinal oedema, because the central venous pressure never approaches the level of the intraocular pressure and thus cannot directly disturb the circulatory dynamics of the eye. However, a low cardiac output and hypoxia may lead to venous dilatation.

Cell Turnover

Q.—*What is the rate of turnover of somatic cells? Is every system included in this turnover? What would be the proportion of new cells to old cells at the end of six months? How much time would pass before there was maximum replacement of cells?*

A.—Very roughly somatic cells may be put in two categories, those which have a high rate of turnover and those in whom turnover is very slow or non-existent.¹ In the latter group are neural cells, muscle cells of all kinds, renal, hepatic, and pancreatic cells. Three systems possess cells in a constant and rapid state of reduplication and turnover—the gut, the blood, and the respiratory tract. Here six days is a more meaningful unit of time than six months.

The gastro-intestinal epithelium is almost completely replaced in six days, and in parts, such as the small intestine, in less than this.² It has been estimated that in man half a pound (0.22 kg.) of cells may be shed daily by the gut (of course most of these are reabsorbed). The blood is not so uniform. Red cells last 120 days and polymorphs and platelets 9 to 10 days. Lymphocytes probably have two populations, one with a short life of 3 to 4 days and the other with a long life of 100 or more days. The respiratory epithelium is in similar state with a turnover time of about 6 to 7 days for the trachea and 7 to 8 days for the bronchi.³

REFERENCES

- 1 Leblond, C. P., and Walker, B. E., *Physiol. Rev.*, 1956, **36**, 255.
- 2 Creamer, B., Shorter, R. G., and Bamforth, J., *Gut*, 1961, **2**, 110.
- 3 Shorter, R. G., Titus, J. L., and Divertie, M. B., *Thorax*, 1966, **21**, 32.

Oral Vitamin B₁₂

Q.—*In what circumstances can vitamin B₁₂ be given orally with an assurance that it will be efficacious?*

A.—The absorption of physiological amounts of vitamin B₁₂—that is probably less than 10 µg. daily—is dependent upon an adequate supply of intrinsic factor in the gastric secretion and upon a reasonably normal absorptive capacity of the ileum. When intrinsic factor is lacking—that is, in Addisonian pernicious anaemia and after gastric resection—absorption of physiological amounts of vitamin B₁₂ can be restored by giving it orally with an additional source of intrinsic factor. This intrinsic factor is usually supplied in the form of dried extracts

of hog stomach. Unfortunately with time hog preparations cease to be effective in a significant proportion of patients, probably because of the development of antibodies to the hog preparation.

Adequate amounts of vitamin B₁₂ can enter the blood stream from the small gut when amounts in excess of 1,000 µg. of vitamin B₁₂ are taken by mouth. This mechanism, possibly a diffusion into the lining cells of the villi, is not dependent on the presence of intrinsic factor, since the peak blood level of vitamin B₁₂ appears much sooner than when the vitamin is being absorbed via the agency of intrinsic factor.

Except under exceptional circumstances, such as when a patient is absolutely unwilling to accept the monthly injection required in normal maintenance in pernicious anaemia or other vitamin-B₁₂ deficiency states, oral administration of vitamin B₁₂ is not recommended.

Nicotine and Blood-pressure

Q.—*Does nicotine produce any significant rise in blood-pressure in man? Is snuff or any form of tobacco smoking (cigarette, cigar, or pipe) contraindicated in cases of hypertension?*

A.—There is no doubt that both the parenteral administration of small doses of nicotine and the smoking of cigarettes cause a rise in blood-pressure in man. The evidence is well summarized by Roth¹ and by McDevitt and Wright.² In normal subjects the greatest responses are seen in those who have labile blood-pressures, in whom the smoking of two cigarettes can cause a rise of the order of 40 mm. Hg systolic and 30 mm. Hg diastolic. Similar results are observed in hypertensive patients.

There is, however, no convincing evidence that smoking is definitely harmful in patients with essential hypertension. There is not an increased prevalence of hypertension among smokers,³ but there is a suggestion that the death rate from hypertension in smokers is about one and a half times that in non-smokers.³

REFERENCES

- 1 Roth, G. M., *Tobacco and the Cardiovascular System: The Effects of Smoking and of Nicotine on Normal Persons*, 1951. Thomas, Springfield, Ill., U.S.A.
- 2 McDevitt, E., and Wright, I. S., in *The Biologic Effects of Tobacco*, 1955, edited by E. L. Wynder. Churchill, London.
- 3 Report of the Advisory Committee to the Surgeon General of the U.S. Public Health Service, 1964, *Smoking and Health*, Public Health Service Publication No. 1103. U.S. Govt. Printing Office, Washington.

Corrections

Alcohol and Driving: In the letter by Dr. B. Hirsh (4 June, p. 1422) the first sentence in the second paragraph should have read, "Readers of your journal may recollect a correspondence between myself, Dr. C. H. Johnson, and others between June and September 1962." We regret any inconvenience this may have caused Dr. I. F. B. Johnston, Hon. Secretary of the Association of Police Surgeons.

We regret that in the Current Practice article on "Acute Bronchitis—Aetiology, Diagnosis, and Management" (16 April, p. 963), by Dr. Gordon Edwards, the drug piperidone was misspelt.