diarrhoea developed in 23, transient diarrhoea in 10, and a tendency to constipation in 5. Only 8 of the patients in the whole group were disappointed in their bowel condition, 3 because they now suffered from constipation. No fewer than 55 of the patients were actually pleased with their increased frequency of bowel action. The same authors collected data from 25 papers on the incidence of diarrhoea after vagotomy and found an overall figure of about 2% of troublesome diarrhoea.

The cause (or perhaps causes) of this post-vagotomy diarrhoea, which is often episodic, remains a mystery. It is tempting to blame the denervation of the rest of the gastrointestinal tract and its adnexae which must inevitably accompany total division of the main vagal tracts as they enter the abdomen. Yet extensive investigations have failed to show convincing evidence of disturbed biliary, 10 11 pancreatic, 12 or small-intestinal function.¹³ Nor have many careful studies, both clinical and experimental, shown any significant metabolic, absorptive, or radiological differences in either men or dogs undergoing total vagal division or selective vagotomy in which either the coeliac or the hepatic or both these branches of the vagus have been preserved. 10 11 15-18 In spite of these findings several surgeons have staunchly advocated the preservation of the coeliac and hepatic tracts of the vagus as a method of preventing post-vagotomy diarrhoea. In Great Britain Mr. Harold Burge has long championed this cause, and in this issue of the B.M.J. (page 481) he and his colleagues put forward a strong case for bilateral selective vagotomy. And they add: "We believe it more reasonable to think that post-vagotomy diarrhoea is caused by denervation of organs other than the stomach rather than by denervation of the stomach itself. Because no functional disturbance of these extragastric organs can be found after total abdominal vagotomy, it must not be concluded that when denervated they are not to blame for this complica-This contribution will give food for thought and perhaps encourage still further studies, both clinical and experimental, in this important field.

Nail-gun Injuries

Working at his desk on the eleventh storey, a bank clerk felt a sudden piercing pain in the left arm and chest. Looking down, he saw blood and collapsed. Investigation showed that he had been hit in the chest with a stud fired by a blank cartridge from a building site across the street. This type of accident has been reported from Germany, ¹ America, ³ the Soviet Union,4 and Great Britain,5-11 where cartridgeoperated fixing-tools (nail guns) have been in common use for about 14 years. A letter in our correspondence columns (p. 511) draws attention to the lack of effective measures for the control of these dangerous tools.

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Some of these guns have a muzzle velocity of up to 1,400 ft. (430 m.)/sec.—a 0.22 in. (6 mm.) rifle has a muzzle velocity of about 1,145 ft. (350 m.)/sec.—and the injuries which have been reported are often severe, because the sharp, highvelocity projectiles, designed to penetrate masonry or wood, may enter deeply into the thorax or abdomen. They can do so after traversing a 10-cm. brieze wall³ or a brick wall (travelling in the mortar between bricks),9 or after ricocheting off hard material such as concrete.6

In Britain nearly all the accidents reported from these tools have resulted from activities on building sites, and the victims have usually been workers on the sites. The responsibility for the enforcement of statutory safety measures in this area rests with the Factory Inspectorate of the Ministry of Labour, but at present the only statutory requirement is that explosives should be handled or used by, or under the immediate control of, a competent person.¹² It would seem that such a regulation was intended for quite different circumstances. Furthermore, it is possible that cartridge-operated fixing-tools may be used on premises that are not legally "factories" and to which the requirements of the Factories Act do not apply. If such use is widespread it may be asked whether such activities are covered by any wider legislation.

R. Wander¹³ has recently reviewed the safety of these appliances, and the Ministry of Works has issued an advisory The British Standards Institute has issued a Specification for Cartridge-operated Fixing Tools.¹⁵ It recommends that the tools should incorporate a splinter guard to withstand ricochet and to retain flying pins, that tools should fire only when the barrel and splinter guard are pressed against the fixing surface with a pressure of not less than 5 kg., and that they should not fire when the angle formed between the perpendicular and the axis of the tool is greater than 7°. An appendix to the specification gives commonsense recommendations for the use of cartridge-operated fixing-tools. The pins should not be driven into brittle or hard materials such as vitreous-faced bricks or hardened steel. They should not be driven into structures where there is a risk of their passing through, unless special precautions are taken. Operators of fixing-tools should receive adequate training, and persons less than 18 years of age should not be permitted to use them.

Such a British Standard has no legal force. The recent reports of accidental injury pose the question whether the time has come for stricter legislative control. The recommendations of the British Standard would form a useful basis for a statutory code.

Dental Anaesthesia

Over the past 20 years it has become apparent that the arrangements for general anaesthesia in dentists' surgeries are sometimes less than perfect. This uneasiness coincided with the explosive post-war growth of interest in anaesthesia in general, and, as a result, general anaesthesia for dental surgery received a close look by experts in anaesthesia who brought to it standards of comparison and assessment which were hitherto unknown. General anaesthesia for dentistry, like anaesthesia for other kinds of surgery, had been regarded for nearly 100 years as a matter of technique alone, the acquisition of which was all that was necessary for a satisfactory performance. It is now accepted that anaesthetists must, in addition, be knowledgeable about a wide range of basic medical subjects, of which physiology and pharmacology are two of the most

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important. If this was to apply to dental anaesthesia as well, as it seemed it should, then the future of dental anaesthesia needed careful consideration. So a joint subcommittee¹ was set up to review the future of dental anaesthesia in the light of the changes that had occurred. The subcommittee consisted of two professors of dental surgery who were also deans of dental schools, two consultant anaesthetists, one of them a head of a university department, two general dental practitioners and one general medical practitioner, a professor of medicine, a consultant dental surgeon, and a county chief dental officer. While collectively the members of the subcommittee had a wide experience of the problems of dental anaesthesia, they nevertheless sought advice and opinion from every interested organization and a number of individuals connected with this field. The subcommittee held numerous meetings extending over nearly two years. Thus, whatever the conclusions reached in its report, at least it cannot be accused of having reached them either hastily or without wide consultation.

In dealing with the clinical aspects of dental anaesthesia, the report does little more than draw attention to the risks inherent in the use of various methods of general anaesthesia and to the need for a proper level of competence in those who use With this aspect of the report there can be little informed dissent. One of the questions put to the subcommittee concerned the indications for general anaesthesia for conservative dentistry. This has particular importance not only because the public must be assured of safe and competently administered anaesthesia but also because of the financial implications for the Ministry. The committee could do little more than summarize the opinions of a wide range of experienced dental surgeons and organizations. If some dentists hold different opinions it is to their colleagues that they should address their dissent.

Who is best fitted to provide general anaesthesia to the public in the dentist's surgery? The subcommittee emphasized that general anaesthesia for dentistry is but one application of general anaesthesia, and that whether the operation is the removal of a tooth or the removal of tonsils the medical, pharmacological, and physiological problems and risks are identical. This inevitably led the subcommittee to the conclusion that ideally general anaesthesia for this type of surgery—like general anaesthesia for any other operation -can be administered safely only by those who as well as having made a special study of anaesthetic problems have also had a medical education. There is some slight hope that ultimately, though probably distantly, this ideal may be achieved. In the meantime the community must make the best arrangements it can. Improved training at all levels is an important immediate task. However, in the present climate of scientific medicine training for a job as potentially dangerous as anaesthesia—not just in terms of mortality but of morbidity as well—can no longer be considered in terms of practical technique alone, important though this is.

The subcommittee's report urges the Minister and the universities to get down to the job of providing and encouraging training in dental anaesthesia for those who desire it. And there are other organizations which feel they are making a contribution in this direction. Already the Ministry with commendable speed has circulated postgraduate deans in medical and dental schools. The coming year will show the nature of their response.

Alcoholic Hypoglycaemia

Most of the 120 cases of alcohol-induced hypoglycaemia reported since 19411 have presented in coma six to twelve hours after alcohol ingestion. Several of these patients died, and at least two underwent surgical exploration for pancreatic insulinomas.^{2 3} Many of the patients were undernourished alcoholics, but chronic alcoholism is not invariable,4 and the condition has been reported in two young children, one of whom died after drinking gin.⁵ It is now known that alcohol itself induces the hypoglycaemia, and it will induce hypoglycaemia in normal man and animals after prolonged fasting.6

N. Freinkel and his colleagues⁷ have performed detailed studies on 15 patients. The first nine rapidly recovered consciousness from deep coma when treated with intravenous glucose. Hypoglycaemia could be reproduced in these patients when they were given oral or intravenous alcohol. The blood sugar levels fall more rapidly after a three-day fast than after overnight fasting. Ten normal subjects infused with alcohol after a three-day fast became hypoglycaemic, whereas after an overnight fast their levels of blood sugar rose. There were no changes in plasma transaminase, amylase, phosphate, or free fatty acids during infusions of alcohol and no increase in immunoreactive insulin. The islet cell response to a glucose load, glucose utilization, and fructose conversion to glucose were all normal. Most of the patients showed an abnormal blood glucose response to fasting, a glucose load, intravenous tolbutamide, or leucine, but no regular pattern has emerged.6 These observations have been confirmed by M. C. deMoura and his colleagues.² A more consistent finding has been the absence of a hyperglycaemic response to glucagon, and this has reverted to normal in some patients during their time in hospital. This failure to respond to glucagon was also observed during infusion of alcohol in man (but not in dogs8) when hypoglycaemia was produced.

In most instances disturbances of liver function have been mild or absent and the liver biopsies either normal or deficient in glycogen.46 The abnormal response to glucagon coupled with the absence of glycogen in the liver in these patients has led to the suggestion that glycogen deficiency may be partly responsible for the hypoglycaemia.7 9 Furthermore, in vitro studies indicate that alcohol interferes with both hepatic glycogen synthesis and gluconeogenesis, and indeed under appropriate conditions alcohol can stimulate or block hepatic gluconeogenesis.7 Freinkel and his colleagues suggest that taking alcohol precipitates hypoglycaemia only when hepatic gluconeogenesis becomes impaired in malnourished people with depleted glycogen stores.

¹ Ministry of Health 1967. Dental Anaesthesia. Report of a Joint Sub-committee of the Standing Medical and Dental Committees on Dental Anaesthesia. H.M.S.O., London.

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