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 subcommittee[1] 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 them. 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.

[1] *Ministry of Health 1967. Dental Anaesthesia. Report of a Joint Subcommittee of the Standing Medical and Dental Committees on Dental Anaesthesia.* H.M.S.O., London.

Alcoholic Hypoglycaemia

Most of the 120 cases of alcohol-induced hypoglycaemia reported since 1941[1] 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 colleagues[7] 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 dogs[8]) 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.[4,6] 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,[9] 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] Brown, T. M., and Harvey, A. M., *J. Amer. med. Ass.*, 1941, 117, 12.
[2] deMoura, M. C., Correia, J. P., and Madeira, F., *Ann. intern. Med.*, 1967, 66, 893.
[3] Kahil, M. E., Brown, H., and Dobson, H. L., *Gastroenterology*, 1964, 46 467.
[4] Neame, P. B., and Joubert, S. M., *Lancet*, 1961, 2, 893.
[5] Cummins, L. H., *J. Pediat.*, 1961, 58, 23.
[6] Freinkel, N., Singer, D. L., Arky, R. A., Bleicher, S. J., Anderson, J. B., and Silbert, C. K., *J. clin. Invest.*, 1963, 42, 1112.
[7] Freinkel, N., Singer, D. L., Arky, R. A., Bleicher, S. J., Anderson, J. B., Silbert,, C. K., Cohen, A. K., and Foster, A. E., *Diabetes*, 1965, 14, 350.
[8] Kahil, M. E., Cashaw, J., Simons, E. L., and Brown, H., *J. Lab. clin. Med.*, 1964, 64, 808.
[9] Field, J. B., Williams, H. E., and Mortimore, G. E., *J. clin. Invest.*, 1963, 42, 497.
[10] Kedes, L. H., and Field, J. B., *New Engl. J. Med.*, 1964, 271, 785.
[11] Floyd, J. C., jun., Fajans, S. S., Knopf, R. F., and Conn, J. W., *J. clin. Endocr.*, 1964, 24, 747.
[12] Bleicher, S. J., Freinkel, N., Byrne, J. J., and Seifert D., *Proc. Soc. Exp. Biol. (N.Y.)*, 1964, 115, 369.
[13] Woeber, K. A., and Arky, R. A., *Brit. med. J.*, 1965, 2, 857.

The most urgent phase in diagnosis is recognition of hypoglycaemia as the cause of coma or of bizarre neurological signs, particularly when these occur in a wasted patient with a history of alcoholism or alcohol in the breath. These features should alert the doctor to the chance of the condition, and he should estimate the level of blood glucose (initially by Dextrostix). A therapeutic trial of intravenous glucose may be justified. Hypothermia[10] and trismus[1 2] are non-specific signs that may accompany alcoholic hypoglycaemia.

Alcohol is more likely to be the cause of hypoglycaemic episodes if there is a history of taking drink before the attacks, an absence of attacks during abstinence, and if hypoglycaemia can be induced by infusion of alcohol after an 18-hour fast. An insulinoma may mimic alcoholic hypoglycaemia, and the response of the level of blood sugar to fasting, tolbutamide, and leucine may give identical results in both instances. Measurement of plasma insulin levels in response to tolbutamide and leucine should help distinguish these conditions, since most patients with insulinoma show an excessive rise in response to tolbutamide or leucine,[11] whereas levels are low in alcoholic hypoglycaemia.[3 12]

Hypoglycaemia due to deficiency of A.C.T.H. can be precipitated by alcohol,[13] but it should be diagnosed correctly by assessment of adrenal and pituitary function.

Medical Social Work in General Practice

When the N.H.S. was framed it was assumed that medical practice was mainly about organic disease, with social and emotional factors playing little part. There might be a place in a large hospital for a social worker but not in general practice. The general practitioner, traditional adviser on non-medical matters, though trained only for clinical ones, and the health visitor, whom he seldom saw, would no doubt cope with all social problems.

Ten years later E. M. Backett, R. P. Maybin, and Yvonne Dudgeon[1] described the need for a social worker in general practice. In 1962 the Porritt Committee[2] suggested that medical social workers in the community services were a necessity, though Joan Collins,[3] describing in 1965 her attachment to a group practice in Cardiff, doubted whether they should be attached to every general practice. Before a medical social worker could be at home in the surgery something would need to change in the doctor–patient relationship, she thought, so that the doctor could make an effective assessment of the patient's whole need and the patient regard the doctor as more than a provider of medical care.

Now J. A. S. Forman and E. M. Fairbairn describe[4] the experimental attachment of a medical social worker to a large group practice for three years. They show that the doctor–patient relationship can be adapted to this arrangement. Once the general practitioners had appreciated the medical social worker's capabilities and limitations and she had assimilated the content and methods of their work, effective communication was established at regular meetings. The doctor–patient relationship was enlarged and strengthened by including the medical social worker. Her function was to assess the family's total situation and help the patient to define his problems, find solutions, and, where situations are unalterable, support him. She became a personal social worker in a way that a traditional social worker could never do, if only because many patients are reluctant to discuss personal problems with an outsider. They accepted her, as they accepted their own doctor's partners, as part of the practice team and trusted her to see them through socially as he saw them through medically, co-ordinating outside social agencies as he co-ordinated outside medical ones. Her work was not mainly with psychotic patients or problem families; rather it was with people normally able to cope but temporarily disabled by illness and emotional and social reactions to it. She improved the service the practice as a whole was able to give in ways whose importance is often underestimated.

The special relationship of the family doctor to his patients gives ready access to them by associates working closely with him. Ideally one of these should be a medical social worker. She can help to make the work of the practice both easier and more effectual,[5] thereby contributing also to make it a "worthwhile career," on which Dr. J. C. Cameron writes in our correspondence columns at page 506. But since there are too few medical social workers the middle grade of social worker, whose new two-year training course emphasizes understanding of people, might be an adequate substitute. She would help even patients with complex problems to understand them, enabling them to use the service of highly trained medical social workers in a consultant role, and by recognizing deteriorating situations before they reached crisis point could fill a much-needed preventive role in the community.

[1] Backett, E. M., Maybin, R. P., and Dudgeon, Y., *Lancet*, 1957, 1, 37.
[2] Porritt, Sir A. (1962). *Review of Medical Services in Great Britain.*
[3] Collins, Joan, *Social Casework in a General Medical Practice.* 1965. London.
[4] Forman, J. A. S., and Fairbairn, E. M., *Social Casework in General Practice.* 1968. Published by Nuffield Provincial Hospital Trust by Oxford University Press (pp. viii+118; 12s. 6d. net).
[5] *Health Centres and Group Practices*, 1966. British Medical Association.

Wrist-watch Tinea

A red scaling and itching eruption often develops in the area of skin on the back of the wrist immediately beneath the wrist watch. In most cases this is caused by a contact allergic sensitivity to the metal of the watch usually due to nickel or more rarely to chrome. This is commonly part of a general sensitivity, and other sites in contact with metal may be affected. In a few cases the wrist-watch strap is responsible, but the eruption in these cases is likely to affect the whole area of contact with the strap.

Another possible cause of an eruption developing beneath the wrist watch is an infection with a ringworm fungus or, rarely, with candida. Recently H. E. Kleine-Natrop[1] has drawn attention to this and described three patients all of whom had *Trichophyton rubrum* infection. Other ringworm fungi can also cause the condition, including *Epidermophyton floccosum*, and in these patients evidence of a primary ringworm infection elsewhere is likely to be found.

The clinical differential diagnosis may be difficult, but Kleine-Natrop points to several features in the development and appearance of ringworm infection that help to distinguish it from a sensitivity reaction. In particular, ringworm tends to start in one small area beneath the watch, it then spreads gradually, and the final area affected may not exactly correspond with the watch site. If there is doubt mycological examination of scrapings will give the correct diagnosis.

[1] Kleine-Natrop, H. E., *Germ. med. Mth*, 1967, 12, 598.