This being due to associated pancreatic damage, to haemolytic, to interference with utilization of folic acid and vitamin B₁₂, or simply to liver damage. Collateral shunting of portal blood may also be important. Some patients have developed clinical haemochromatosis after a portacaval shunt, though the amount of excess iron in such cases may be less than 3 g.¹³

Because of the frequent finding of siderosis in primarily alcoholic cirrhosis and the repeated failure to produce haemochromatosis experimentally in animals by iron overload alone, R. A. MacDonald¹⁴ has proposed that idiopathic haemochromatosis is merely one end of a range of states comprising cirrhosis and siderosis. Most workers find, however, that the overlap between the two groups described is not as great as MacDonald suggests. Furthermore, L. W. Powell⁴ could find increased iron stores in only 1 out of 34 relatives of patients with an alcoholic cirrhosis and secondary siderosis as compared with the 16 out of 63 relatives of patients with idiopathic haemochromatosis mentioned above.

The response to venesection therapy in idiopathic haemochromatosis is usually gratifying. Pigmentation and hepatomegaly decrease, and in some cases there is improvement in glucose tolerance. One patient has been described in whom serial biopsies showed reversal of the cirrhosis. Venesection therapy in cirrhosis with a slight secondary siderosis would hardly seem worth while, but its value both in these and in the occasional severe case requires further assessment. The question also arises how far should relatives of patients with idiopathic haemochromatosis be investigated and treated. Unfortunately the serum level of iron and the percentage saturation of the total iron-binding capacity are poor screening tests, since the normal ranges are wide and the serum level of iron may show spontaneous fluctuations. An estimate of the body's store of iron can be obtained by measuring urinary excretion of iron over 6 or 24 hours after injection of the iron chelators calcium D.T.P.A. or desferrioxamine.⁶ ¹⁵ Alternatively the total chelatable iron stores can be estimated by the differential ferrioxamine test of J. Fielding.⁴ Though these tests of iron excretion may not show minor increases in iron storage, they will detect the more severely affected relatives. In these the next stage is liver biopsy. The risk of this in experienced hands is low, and, though some physicians have recommended that all affected relatives should be treated by multiple venesections, such therapy is probably best reserved for those with histological evidence of tissue damage as well as siderosis.

Anaesthesia for Dental Extractions

The deaths and less disastrous consequences of anaesthesia given for dental extractions have for some years now been a subject of study and debate. They are at present being inquired into by a Ministry of Health committee, and its report is awaited with interest. A growing realization of the dangers of dental anaesthesia, together with better training in its techniques, has probably helped to lower the number of deaths from the high level of 22 in 1952 in England and Wales to the present 4 to 6 per year.

For many years after F. W. Hewitt¹ popularized the addition of oxygen, little progress was achieved except for the

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introduction of more complex equipment. The need for speed in extractions became less pressing when apparatus could apparently be relied on to give precise mixtures to patients, but the newer techniques also had risks. As the late K. C. Macarthy frequently said, "No anaesthetic agent is safer than its administrator." In the 1930s the introduction of the short-acting barbiturates simplified the induction of the robust, resistant patient, and many short cases of extraction were completed entirely without the addition of any inhalation anaesthetic. The use of trichloroethylene as an adjuvant with nitrous oxide and oxygen became general in Great Britain during the second world war and allowed smoother anaesthesia to ensue. Many anaesthetists became expert in its use, employing higher percentages of oxygen than were previously the rule in treating the ambulant dental case. But deaths continued to occur, even though they were rare in relation to the vast numbers of dental anaesthetics administered.

One school of thought blamed the sitting-up position usually adopted; another pointed to the unreliability of the "demand-flow" type of apparatus to deliver accurate mixtures of the two gases under clinical conditions. Certainly cerebral damage can occur during anaesthesia, especially in the sitting-up position. But it is doubtful if the vasovagal reflex that triggers off a fainting attack will act in an anaesthetized patient. However, many patients are in a state of acute apprehension on entering the dental surgery, especially if they have been kept waiting a long time in a crowded and badly ventilated waiting-room among loquacious fellow-sufferers.

Such patients are pale and many have a hypotensive attack before the administration of the anaesthetic. From his traditional position the dental anaesthetist, standing behind the patient, may be unable to appreciate the onset of such a response, and if anaesthesia is started under such conditions the patient may be in grave peril. It is a wise anaesthetist who faces his patient in the dental chair and carries on a short conversation with him so that he can assess his reaction to the strain of the ordeal. If hypotension is suspected the induction should take place in the supine position after administration of oxygen. Or it may be advisable to postpone the operation to another occasion when the patient can be safely sedated.

Recently J. B. Brierley and A. A. Miller have described one case of fatal brain damage and admirably discussed the relevant literature. Undoubtedly the safest method of operating in the oral cavity is under endotracheal anaesthesia, which allows adequate packing and the use of the supine position. But the numbers of patients are too great for such a methodical approach.

The use of halothane as an adjuvant has made possible the maintenance of anaesthesia with high percentages of oxygen hitherto considered impracticable in nitrous-oxide-oxygen mixtures. The use of premixed cylinders containing 50% of each gas in conjunction with halothane has been suggested. Demand-flow types of apparatus have been greatly improved, and two accurate machines are now readily available in Britain. The British Standards Institute is at present endeavouring to apply standards of performance to them—not only when supplied by the manufacturers but also when maintained by their service engineers. But despite such advances prolonged anaesthesia in the dental chair should not be attempted by the inexpert, nor should patients be submitted to it without a thorough medical examination. The use of intravenous anaesthesia for conservative dentistry, even when given so lightly that reflexes are not completely obtunded, must increase the risk to the patient of what would otherwise be a thoroughly safe procedure. The operator-anaesthetist is not, as a rule, the best person to judge a patient's medical fitness for prolonged administrations, and in the event of an emergency the patient's best interests could be served only by the presence of two qualified people.

Though neglected for many years as a branch of anaesthesia deserving special study, dental anaesthesia is now receiving its rightful attention, but much more clinical research is desirable. For instance, it is possible that intermitting intravenous anaesthesia may reduce the saturation of oxyhaemoglobin below 85%—an undesirable consequence in patients who have any impairment of the cardiac output. If in fact does so, then additional oxygen should be given to patients anaesthetized in this way, and here the premixed gases would prove valuable. Indeed J. S. Lundy recommended such supplementation with oxygen as far back as 1938. The drugs employed may have changed since then, but not the physiology of respiration nor the response of a patient to known respiratory depressants.

Amines and Depression
Depression in its many forms has been exhaustively studied for decades by psychiatrists of all kinds; it has been a challenge to the drug firms; and undeniably it has responded to treatment. Yet psychiatrists are still not agreed on the classification of its varied forms, its psychodynamics and aetiology, or its pathology. With his usual courage Professor Henry Miller recently entered the fray. The result was a lively, practical exposition of what may be called the contemporary materialist picture of endogenous depression. Professor Miller maintains that a neurologist may quite properly discourse on a psychiatric topic, such as endogenous depression. Certainly it is not so long since a large section of what is now psychiatric practice was in the hands of neurologists, and Professor Miller has already ventured, with telling effect, into the psychiatric field in his Milroy lectures on accident neurosis. And has he not got a diploma in psychological medicine?

Much of what Professor Miller presents on the treatment and symptomatology of depressive illness is accepted teaching, but not everyone would agree with his section on the nature of the disorder. He stresses the many causal factors—genetic, psychological, and temporal—which in differing combinations and valencies determine the clinical form, course, and responsiveness to treatment of attacks of depression in individuals, and he infers "a final common pathway of pathophysiological disturbance." The supporting evidence for this is chiefly biochemical, and as yet inconclusive. This is not surprising, since there is little justification at present for