EMERGENCIES IN GENERAL PRACTICE**ACUTE ALCOHOLIC POISONING**

BY

J. A. IMRIE, M.D., F.R.C.S.Ed.*Chief Medical Officer, City of Glasgow Police*

Cases of mild or medium intoxication are chiefly of medico-legal interest. The more severe states of intoxication are clinical emergencies presenting themselves to the general practitioner, the hospital casualty staff, and the police surgeon. The individuals concerned are peculiarly liable to a variety of complications, including falls, traffic accidents, suffocation by a variety of causes, drowning, exposure, carbon monoxide poisoning, and electrocution. The subjects of cardiovascular disease may precipitate acute cardiac failure by a debauch, and post-alcoholic debility may be complicated by acute pulmonary infections. In the suffocation group, inhalation of vomitus or respiratory obstruction by the tongue or dentures is not infrequent.

Dangerous Complicating Factors

The use and abuse of the barbiturate drugs have added a serious complication. It is generally recognized that a synergism exists between alcohol and the barbiturates, the latter being potentiated so that their anaesthetic and lethal levels are lowered if alcohol is present in the blood and tissues in adequate quantity. This potentiation can be fatal with concentrations of both drugs far below their ordinary fatal levels—for example, in one fatal case the blood alcohol indicated a minimum intake of the equivalent of three and a half large whiskies and a blood barbiturate level equivalent to taking 9 gr. (0.6 g.) of quinalbarbitone sodium. In such cases death can occur within a few hours. There is evidence also that morphine, or morphine-scopolamine, injected in therapeutic doses in persons with blood alcohols over 0.18% (180 mg./100 ml.) may cause severe morphine poisoning and perhaps death. Such cases have occurred when morphine was given to control violent alcoholics. Acute and chronic alcoholics are notably bad anaesthetic risks.

The combination of psychopathic personality with alcoholic intoxication may produce a clinical state out of all proportion to the amount of alcohol taken. *Mania a potu* may develop in those who have suffered previous cerebral trauma, or in the presence of epilepsy, cerebral arteriosclerosis, or unstable mentalities; even small amounts may produce a violent intoxication, greater than the same dose would cause in normal people. A more dangerous state arises in cases of chronic alcoholism where, either as a result of a short intense orgy or a more prolonged one or the sudden withdrawal of alcohol, delirium tremens or an acute hallucinosis develops.

Alcoholic Content of Various Liquors

The active principle of the common alcoholic beverages is ethyl alcohol. The distilled liquors—whisky, gin, rum, brandy, etc.—contain between 40 and 60% ethyl alcohol by volume. Wines vary from 10 to 20%, the fortified ones being the more potent. Fermented liquors—for example, beer and stout—vary, but the average content is 3–4% by volume. Sometimes methylated spirits is taken. This consists of ethyl and methyl alcohol in the proportion of 19:1, with certain impurities. The addition of a cheap red wine or other flavouring agent is common practice. This makes a potent mixture.

Clinical Progress

The dangerous states of intoxication are reached when there is gross locomotor incoordination and dulling of the intellect, with impaired judgment, and when coma develops. The time taken to reach these states and the severity of the symptoms depend on individual susceptibility, habituation, the amount of drink taken, the concentration of the alcohol, the speed of ingestion, and the presence of circumstances promoting absorption—for example, fasting—or liable to enhance the effect—for example, fatigue. Usually the clinical condition progresses by obvious stages, but occasionally the advanced states, even coma, may be reached very quickly and without preceding signs of intoxication. *Mania a potu* may be very early in onset, and epileptics are specially liable to become quickly intoxicated.

Clinically the distinctive symptom of these states is the vinous odour from the breath. This is usually not masked by deodorants in the severe cases and is to be distinguished from the smell of fresh alcohol recently taken, or perhaps given therapeutically by some good Samaritan in non-alcoholic conditions.

The well-known symptoms of moderate intoxication proceed to gross incoordination and inarticulateness, and the power of locomotion may be lost. At this stage vomiting may occur without affecting the progress of the intoxication but involving the risk of asphyxia. Unconsciousness develops; the face is usually flushed, the pulse rapid, and the blood pressure lowered; breathing is heavy and stertorous, and the temperature subnormal. The condition of the pupils is variable: they may be dilated, reacting sluggishly to light; or contracted, when they may show Macewen's sign. This is the dilatation of the pupil which occurs with shaking or slapping the patient without his regaining consciousness; when the stimulus is withdrawn the pupil slowly returns to the contracted state. This sign is strong evidence in favour of alcoholic coma.

As coma supervenes and deepens, muscular relaxation becomes complete and the face may become pale and sweaty, the pupils being contracted or dilated and fixed, the pulse rapid and of low tension, and the temperature subnormal. The duration of coma varies. In unaided recovery it gradually lightens into a deep sleep with improvement in all the symptoms and in the patient's condition. Coma persisting beyond five hours is of very serious moment, and apt to be fatal. Death from respiratory paralysis is likely if it persists for 12 hours.

Delirium tremens is one of the acute confusional states occurring in chronic alcoholics after a prolonged or a short intensive bout of drinking, or as a result of the sudden withdrawal of alcohol. It lasts for about three to five days, during which period there is a definite risk of death from cardiac or renal failure or an acute pulmonary infection. In the majority of cases recovery is to be expected. The distinctive feature of this form of delirium as compared with other delirious states is the generalized coarse tremor of the face, tongue, and hands, associated with prostration, tachycardia, lowered blood pressure, and a dry, furred tongue. Epileptiform convulsions and visual hallucinations are common, usually of small fast-moving objects like rats or insects. Intervals of apparent lucidity may occur with persisting disorientation for time and events. The mood varies, there being sometimes fear amounting to terror, excitement with violence, or these may alternate with jocularity. Restlessness and insomnia are prominent.

Differential Diagnosis

Alcoholic and other forms of coma have many features in common.* Comatose patients should not be left in police cells or unattended, and it may be fatal to assume that the smell of alcohol establishes the diagnosis. The diagnosis presents many difficulties, the chief being the association of head injury with alcoholic intoxication. Whenever there is the history, or the presence, of a head injury, a comprehensive general examination with particular attention to the nervous system should be supplemented by an immediate radiological examination of the skull, which may establish the presence of fracture. An estimation of the blood alcohol will indicate the part played by alcohol, since levels of 400 mg./100 ml. (0.4%) and over are likely of themselves to cause coma, and levels over 700 mg./100 ml. (0.7%) to result in death.

In extradural haemorrhage, the history and the pupillary changes are important. If alcohol has been consumed and there is also a head injury, the persistence of coma despite adequate treatment for intoxication, or initial recovery with treatment followed by recurrence of the coma, points to the probability of an intracranial lesion. Alcoholic coma may simulate pontine haemorrhage, but hyperpyrexia is absent. Other cerebral vascular conditions may be suspected if there is valvular heart disease or hypertension.

Coal-gas poisoning produces a red colour in the mucosae and cheeks, and the distinctive odour of the gas may be present. Death may occur with less than the usual lethal dose of carbon monoxide if alcohol is present in the body in sufficient concentration.

Coma due to barbiturates can usually be differentiated by the history and by the finding of congestion and cyanosis, shallow respiration, and signs of pulmonary oedema. When alcohol has also been taken barbiturate poisoning may be overlooked. If no history is available, a search of the clothing may reveal the drug or the empty container. Coma due to morphine or belladonna has characteristic features, though alcohol in moderate amount will potentiate morphine. In diabetic or hypoglycaemic coma the absence of the smell of alcohol should help to avoid confusion.

Treatment of Severe Intoxication

Once the diagnosis is established, treatment should aim at detoxication. Full recovery is not essential: recovery of consciousness, or at least a state of sleep, is adequate. Until then the patient should be under constant observation. Once he is detoxicated adequately he may be left to complete his own recovery. Since he will in all probability have passed his peak blood level by the time he is seen, it is doubtful if gastric lavage will be of material benefit. But if the patient is seen early, lavage can be undertaken, using a weak sodium bicarbonate solution and leaving 5-10 oz. (140-280 ml.) in the stomach. Continuous oxygen, in the form of a 90% mixture with carbon dioxide by mask or nasal tube, is recommended; it will allay excitement and cut short coma. Vitamin therapy has become popular and produces some startling results. Vitamin B₆ (pyridoxine hydrochloride) may be given in doses of 50-100 mg. intravenously, but if one injection fails to cut short the coma it is hardly worth repeating the treatment. More intensive therapy consists in the intravenous administration of vitamin B₁, 1,000 mg., vitamin B₁₂, 200 mg., and vitamin C, 1,500 mg. This is particularly applicable after prolonged bouts of drinking, when delirium tremens is impending.

Nikethamide, intravenously or intramuscularly, has been in use for many years. It is usually given intravenously and is rapidly inactivated after injection. Doses of between 5 and 10 ml. have been given with striking results. When more than 3 ml. is given, the injection should be made slowly and a careful watch kept for muscular twitchings to

avoid the production of convulsions. The drug should be administered only by the doctor in attendance. Recovery is often rapid, and is ushered in by sneezing or vomiting or both. The best results seem to be obtained in the young and non-habituated. In the older, more habituated group a combination of vitamin B₆ and nikethamide in smaller doses may be effective. These forms of treatment have been found to allay excitement. Caffeine and sodium benzoate B.P., 1 g., intravenously is stated to be beneficial in both the comatose and the excited such as those with *mania a potu*. In these latter cases the forms of intravenous therapy described are often successful and obviate sedation.

Delirium Tremens

Delirium tremens requires the sedation of the patient and the correction of the nutritional deficiency. Home treatment involves much strain on the attendants. Hospitalization is more appropriately carried out in general hospitals, certification being undesirable unless there is also alcoholic dementia. Cases of alcoholic dementia are appropriately admitted to mental hospitals.

Morphine, morphine with hyoscine, and paraldehyde are unsafe in delirium tremens. Nor is the use of barbiturates without danger if alcohol is present in the blood and tissues. Detoxication with vitamins B₁, B₆, and C intravenously in the doses already indicated is particularly suitable for these cases. This treatment may be repeated daily in rather smaller dosage. A rapid abatement of the nervous symptoms is to be looked for, and from a nutritional point of view the vitamins are essential. Sedation may be unnecessary with this treatment. Alternatively, if there is no barbiturate-alcohol risk and sedation is necessary, a quick-acting barbiturate, in full dose, can be given by mouth, followed by a slow-acting one. If oral administration is impossible, an initial dose of phenobarbitone sodium, 3 gr. (0.2 g.), can be given intramuscularly.

Gastric lavage with weak sodium bicarbonate is a useful preliminary to tube feeding, should this prove necessary. If nourishment cannot be given by mouth a tube feed of 10-15 oz. (280-420 ml.), containing glucose, 3 oz. (93 g.), hydrolysed protein, 1 oz. (31 g.), with 300 mg. of ascorbic acid and vitamin B complex, is given four-hourly. The amount of fluid is gradually increased. When the delirium abates and the patient can swallow, fluid with high carbohydrate content is given by mouth. The administration of brandy or whisky (up to 1 oz. (28 ml.) and repeated four-hourly till delirium subsides) in cases with extreme weakness is a question for the attending physician. The use of corticotrophin (A.C.T.H.) in 25-mg. doses intramuscularly every six hours is stated to have encouraging results during the acute stage of the delirium.

Once the delirium is over and nourishment is being taken adequately, treatment consists in improving the general health.

Methyl Alcohol Poisoning

Poisoning by methyl alcohol is uncommon and usually occurs in minor outbreaks, but individual cases do occur and may be missed.

Methyl alcohol is used commercially as a solvent for paints, varnishes, and shellacs, and is present in some anti-freeze mixtures. Sometimes alcoholic beverages—for example, whisky—are adulterated with methyl alcohol. Many of the fatalities from alcohol during prohibition in America were due to this alcohol. It is one of the constituents of methylated spirits. The sale of methyl alcohol is controlled by the Customs and Excise.

The response to methyl alcohol is variable. Large amounts—for example, 500 ml.—have been drunk without death resulting, while 15 ml. of 40% methyl alcohol has caused death. It gives the same symptoms of intoxication as ethyl alcohol. The symptoms of poisoning do not appear until after a latent period of 15-24 hours, or occasionally longer (up to 40 hours). Rarely they develop within an hour or two. During this latent period a condition of acidosis develops,

*See also article in this series by Dr. M. J. Parsonage on "The Differential Diagnosis of Coma" (January 22, p. 217).

the result of the oxidation of methyl alcohol to formic acid and formaldehyde. The plasma-bicarbonate level is lowered and the prognosis can, in fact, be measured in terms of the plasma-bicarbonate level, death being likely when this is below 10 mEq per litre.

When poisoning occurs it is ushered in by headache, usually severe, nausea, giddiness, and epigastric pain. Accompanying these there are visual symptoms, usually blurring of vision. At this stage retinal changes may be evident, with hyperaemia of the optic disk and oedema of the adjacent retina. Cyanosis and dyspnoea develop, and coma sets in early. The skin is cold and clammy and there is tachycardia. The pupils are often dilated and show sluggish reactions. Prostration is severe, and finally there is respiratory failure and death. The coma may last for several days. The ocular changes may occur early or may follow recovery. Photophobia and blurred vision, or even complete blindness, may occur within 12-24 hours.

The combination of gastro-intestinal and visual symptoms following a latent period after drinking alcohol should suggest methyl alcohol poisoning, and direct attention to the plasma-bicarbonate level if facilities exist for its estimation. The fact that others who indulged in the same drinking bout are not affected does not exclude methyl alcohol, since it is recognized that if ethyl and methyl alcohol are taken together there may be considerable variation in the individual responses.

Treatment of Methyl Alcohol Poisoning

Treatment is aimed at combating the acidosis. When intense shock is present with signs of respiratory paralysis intravenous nikethamide, 2-5 ml., can be given. Continuous oxygen is also indicated, but gastric lavage is useless, if not actually dangerous.

Alkalis should be given at once by the intravenous route. Sodium lactate, a molar solution (11.2%) in an isotonic solution of the three chlorides, gives excellent results. *Injectio sodii lactatis* co., B.P., can be given. If sodium lactate is used in molar solution, the injection should not be given more rapidly than at a rate of 300 ml. an hour. The dosage and frequency of the injections are determined by the severity of the symptoms and the results of repeated estimation of the plasma-bicarbonate level. The more severe the symptoms, the higher the initial dose. If there are no facilities for plasma-bicarbonate estimation, the pH of the urine is a satisfactory guide, or its reaction to litmus paper. When the urine becomes alkaline, there is a danger of tetany, hypokalaemia, or sodium retention, and the administration of alkali should be stopped and resumed only if the symptoms of poisoning recur or the urine turns acid again.

Alternatively, sodium bicarbonate intravenously provides a simple and safe method if similar controls are observed. A solution of 50 g. of sodium bicarbonate in 1 litre of 5% glucose is given, the amount administered being determined by the severity of the initial symptoms, and thereafter by the plasma-bicarbonate level or the pH of the urine. If intravenous administration is not immediately possible and the patient can take the bicarbonate by mouth or by stomach tube, doses of 4 g. are given every 15 minutes if they can be tolerated. In all cases careful control of the plasma-bicarbonate level or the urinary pH should indicate the amounts required.

Other Alcohols

Of the remaining alcohols, propyl and isopropyl alcohol have an action similar to ethyl alcohol but are more toxic. They are used industrially in the manufacture of perfumes, toilet articles, and certain lacquers. They are much less volatile than other alcohols and are uncommon as poisoning agents. Ethylene glycol is used in the manufacture of anti-freeze mixture. On occasions it has been known to cause death accidentally and has been used suicidally.

Next article on Emergencies in General Practice.—
"Is It Polio?" by Dr. T. Anderson.

UGANDA AND GREAT ORMOND STREET AN EXPERIMENT IN GLOBAL PAEDIATRICS

BY

BERNARD SCHLESINGER, O.B.E., M.D., F.R.C.P.

Senior Physician, Hospital for Sick Children, Great Ormond Street, and to the Children's Department, University College Hospital

One of the most recent activities of the Hospital for Sick Children, Great Ormond Street, is a liaison with the paediatric services of Uganda, hitherto very scantily provided. A senior registrar from Great Ormond Street has been seconded to the Protectorate for a two-year period, and he will be followed in turn by further registrars. In addition two nursing sisters from the hospital volunteer to go out and take charge of the children's wards for a similar period, and each year one of the Great Ormond Street consultants will pay a month's visit.

Among the reasons that have led to this new venture is the desire to keep abreast of the rapidly increasing global

Children's ward, Mulago Hospital, Kampala.

nature of medicine brought about by modern air travel, better knowledge of tropical paediatrics being particularly important. Many postgraduates from the Orient attend the courses of the Institute of Child Health at Great Ormond Street, and it was realized that the special problems they would encounter on their return were not being adequately covered. There was also the pressing need for improved care of the vast number of sick children in Africa and the introduction of up-to-date methods. When it became known that the Uganda medical authorities would value such help, with the aid and financial assistance of the Colonial Office, the co-operation of Makerere College at Kampala, and the generosity of the Board of Governors of the Hospital for Sick Children, Great Ormond Street, the Uganda scheme was promoted.

So far the liaison has worked admirably. It has proved of great benefit to the Mulago Hospital, through the teaching and example set by the small band from Great Ormond Street. Equally, the experience gained by the doctors and nursing sisters from London has been invaluable in widening their horizon, taxing their ingenuity, and increasing their responsibility. Disease is encountered in a raw and florid state undreamt of in England, and offers the young doctor and research worker innumerable problems to solve. The team from Great Ormond Street has been welcomed and wholeheartedly supported. The registrar occupies a privileged position, acting virtually as aide-de-camp to Dr. HUGH TROWELL, a general physician who has taken paediatrics as an additional specialty under his wing.