Hypoglycaemia should be suspected in any unconscious or semiconscious child who has taken alcohol without food, even before convulsions have occurred. In such cases it is essential to monitor the blood glucose at intervals up to five hours after ingestion or to give intravenous glucose as a precautionary measure. The amount of alcohol need not be large, as our results show, and the alcohol contained in a small glass of wine may be sufficient.

The length of the preceding fast is an important factor, probably because of the reduction in liver glycogen. In normal adults a fast of 44-72 hours is necessary to induce alcohol hypoglycaemia (Arky and Freinkel, 1966), whereas in alcoholics and adults with diseases such as thyrotoxicosis, in which glycogen stores are depleted, an overnight fast is sufficient (Arky and Freinkel, 1966).

Madison (1968) stated that in general infants and children are liable to alcohol-induced hypoglycaemia. It is, however, possible that an inherited predisposition may exist in some children. To test this hypothesis alcohol tolerance tests after an overnight fast were performed on the patient, his two sibs, and two other children with suspected hypoglycaemia. All showed a fall in blood glucose, the lowest level of which occurred four to five hours after ingestion of alcohol, which was at that time usually below 20 mg./100 ml. A pronounced rise in blood lactate combined with an unchanged and even decreased pyruvate accompanied the fall in blood glucose in all cases. The similarity of the results in all five and the failure to detect disturbance of liver or renal function tend to exclude a specific inherited metabolic defect.

Insulin and Plasma Cortisol Levels.—In all cases the insulin level was normal initially and even fell a little during the test, despite the hypoglycaemia, proving that this was not due to increased insulin production. The pronounced rise in the plasma cortisol, a response similar to that seen in normal adults, indicates a normal adrenal cortical function, in contrast with the impairment found in alcoholics, in whom the plasma cortisol falls when they are given alcohol (Merry and Marks, 1969).

A fuller account of the biochemical findings will be published elsewhere.

We thank Dr. M. J. Wilmers for permission to study this case, Dr. Ann E. Robinson, of the London Hospital Forensic Laboratory, for the alcohol estimations, and Dr. E. Ann Burgess for her assistance.

N. K. MACLAREN, M.B., M.R.A.C.P.
Resident Medical Officer.

H. B. VALMAN, M.B., M.R.C.P.
Senior Registrar.

B. LEVIN, M.D., PH.D., F.R.C. PATH.
Consultant Pathologist.
Queen Elizabeth Hospital for Children, London E.2.

REFERENCES

Arky, R. A., and Freinkel, N. (1966). *New England Journal of Medicine*. **274**, 426.
Dickerman, J. D., Bishop, W., and Marks, J. F. (1968). *Pediatrics*, **42**, 837.
Jeune, M., Cotte, J., and Nivelon, L. (1960). *Pédiatrie*, **15**, 664.
Luft, R., *et al.* (1966). *Lancet*, **2**, 254.
Madison, L. L. (1968). *Advances in Metabolic Disorders*, **3**, 85.
Merry, J., and Marks, V. (1969). *Lancet*, **1**, 921.
Perman, E. S. (1958). *Acta Physiologica Scandinavica*, **44**, 241.
Tolis, A. D. (1965). *Pediatric Clinics of North America*, **12**, 423.

Acute Alcoholic Hypoglycaemia in Two 4-year-olds

British Medical Journal, 1970, **1**, 280

Hypoglycaemia is a well-recognized complication of alcohol poisoning in adults, and over 120 cases have been recorded since 1941. Hypoglycaemia as a result of alcohol ingestion by children, however, has been recorded on very few occasions (Peluffo *et al.*, 1958; Cummins, 1961; Ramon *et al.*, 1963). This paper describes the cases of two children who developed hypoglycaemic convulsions after alcohol ingestion.

CASE 1

A 4-year-old boy was admitted to hospital via the casualty department in July 1968. Three hours earlier he had consumed 540 ml. of sherry. He had vomited several times and had gradually become drowsy. On examination he was unconscious and did not respond to painful stimuli. There were no neurological signs and respiration was not depressed. His breath smelled strongly of alcohol. Gastric lavage was not performed.

Five hours after ingestion he developed six right-sided convulsions, each lasting two minutes, though by this time he was conscious but drowsy, the blood sugar was less than 25 mg./100 ml. and blood alcohol was 100 mg./100 ml. He was given 300 ml. of milk containing 30 g. of glucose by mouth. Within 10 minutes the convulsions ceased and full consciousness was regained. He then drank a further 300 ml. of milk containing 10 g. of glucose. The convulsions did not recur and there was no residual neurological deficit.

Next day he was his usual self, apart from a slight headache and symptoms suggestive of an alcoholic "hangover." Blood sugar levels were normal. He was discharged two days after admission.

CASE 2

A 4-year-old boy was admitted to hospital in October 1969. Four hours previously he had consumed approximately 400 ml. of gin. He had become drowsy over the next three hours and had convulsed for five minutes previous to admission.

On examination he was deeply unconscious and convulsing. His capillary blood sugar was less than 40 mg./100 ml. (Dextrostix), and in venous samples. Blood sugar was found to be 12 mg./100 ml. An intravenous drip of 10% dextrose was set up and 20 ml. of 50% dextrose was injected intravenously at once. The convulsions ceased and consciousness was regained within two minutes of this injection. Venous blood sugar levels were normal after four and eight hours. The intravenous 10% dextrose was then discontinued.

The boy behaved normally thereafter and physical examination was negative. Subsequent blood sugars and an oral glucose tolerance test were normal. He was discharged on the third day after admission.

COMMENT

It is now accepted that alcohol can produce hypoglycaemia in normal subjects, though the exact mechanism is not clear. In acute alcoholism islet cell response is normal, but there is thought to be a failure of normal glycogenolysis and gluconeogenesis.

In the two cases described here the convulsions developed within five hours of ingestion of the alcohol and were clearly due to hypoglycaemia. Prolonged and severe hypoglycaemia can be fatal (Cummins, 1961) or produce irreversible brain damage.

If a child thought to have ingested alcohol develops drowsiness, coma, convulsions, or bizarre central nervous system signs, blood sugar levels should be estimated and an immediate therapeutic trial of intravenous glucose is justified.

I wish to thank Dr. W. Henderson for assistance with this paper.

H. J. HEGGARTY, M.B., M.R.C.P. (LOND., GLASG.).
Senior Registrar in Paediatrics, the Northern General Hospital and the Children's Hospital, Sheffield.

REFERENCES

Cummins, L. H. (1961). *Journal of Pediatrics*, **58**, 23.
Peluffo, E., Scolpini, V., Grezzi, J., and Tarallo, N. (1958). *Archivos de Pediatría del Uruguay*, **29**, 94.
Ramon, G., *et al.* (1963). *Archivos de Pediatría del Uruguay*, **34**, 207.