Stagno, and Alford, 1972). Lastly, the asymptomatic carrier state may be too localized to evoke an abnormal host response. There certainly was no evidence of generalized CMV-induced anergy such as is seen in some viral infections (Salaman, 1970). Thus, many of the mothers and infants studied here showed a high titre serum anti-CMV antibody response.

Summary

Serum immunological abnormalities were sought in 13 mothers and 11 infants with asymptomatic cytomegalovirus (CMV) infection. Only a few minor abnormalities were detected, contrasting sharply with the well-described abnormalities in patients with post-perfusion or adult CMV syndrome.

REFERENCES

- Kantor, G. L., Goldberg, L. S., Johnson, B. L., Derechin, M. M., and Barnett, E. V. (1970). Immunologic abnormalities induced by postperfusion cytomegalovirus infection. *Annals of Internal Medicine*, **73**, 553.
- Kantor, G. L., and Johnson, B. L. (1970). Cytomegalovirus infection associated with cardiopulmonary bypass. *Archives of Internal Medicine*, **125**, 488.
- Klemola, E., von Essen, R., Wager, O., Haltia, K., Koivuniemi, A., and Salmi, I. (1969). Cytomegalovirus mononucleosis in previously healthy individuals: five new cases and follow-up of 13 previously published cases. *Annals of Internal Medicine*, **71**, 11.
- McCracken, G. H., and Shinefield, H. R. (1965). Immunoglobulin concentrations in newborn infants with congenital cytomegalic inclusion disease. *Pediatrics*, **38**, 933.
- Reynolds, D. W., Stagno, S., and Alford, C. A. (1972). The relation between cervical and perinatal cytomegalovirus (CMV) infections. (Abst.) *Pediatric Research*, **6**, 391.
- Salaman, M. H. (1970). Immunosuppressive effects in infection. *Proceedings of the Royal Society of Medicine*, **63**, 11.
- Stern, H., and Elek, S. D. (1965). The incidence of infection with cytomegalovirus in a normal population. A serological study in Greater London. *Journal of Hygiene*, **63**, 79.
- Stern, H., and Tucker, S. M. (1965). Cytomegalovirus infection in the newborn and in early childhood: three atypical cases. *Lancet*, **2**, 1268.
- Stiehm, E. R., and Fudenberg, H. H. (1966). Serum levels of immune globulins in health and disease: a survey. *Pediatrics*, **37**, 715.
- Wager, O., Räsänen, J. A., Hagman, A., and Klemola, E. (1968). Mixed cryoglobulinemia in infectious mononucleosis and cytomegalovirus mononucleosis. *International Archives of Allergy and Applied Immunology*, **34**, 345.
- Weller, T. H. (1971). The cytomegaloviruses: ubiquitous agents with protean clinical manifestations. *New England Journal of Medicine*, **285**, 203.

R. BLUESTONE,* L. S. GOLDBERG, S. M. TUCKER, and H. STERN
VA Wadsworth Hospital and UCLA School of Medicine, Los Angeles; Hillingdon Hospital, Middlesex; and St. George's Hospital Medical School, London.

*Correspondence to Dr. R. Bluestone, Rheumatology Section, VA Wadsworth Hospital, Los Angeles, California 90073, U.S.A.

Hyperglycaemia in hyperosmolar dehydration

Hyperosmolar dehydration is a well recognized clinical state in which hyperglycaemia has been noted as a coincidental finding. Hitherto this hyperglycaemia has been considered of little importance, but we wish to report 6 cases where it was a prominent feature and discuss its possible aetiology and significance, stressing the dangers of insulin therapy in such patients.

Materials and methods

The ages of our patients ranged from 11 days to 22 months. Gastroenteritis was the precipitating illness in all. Intake of carbohydrate had not been excessive and none had a family history of diabetes mellitus. The Table gives the initial laboratory findings.

The clinical management was not identical in each case since several hospitals were involved, particularly in Cases 1, 2, and 3. All received intravenous fluids.

Hyperosmolar dehydration is a well-recognized clinical entity in paediatrics, but the finding of hyperglycaemia was unexpected and it confused the initial diagnosis in Cases 1, 2, and 3 in whom diabetes mellitus was suspected and insulin therapy instituted. In Case 2 this produced hypoglycaemic convulsions (venous blood glucose 16 mg/100 ml) which were corrected by an intravenous injection of 20 ml 20% glucose. Case 3 died 13 hours after admission but was not hypoglycaemic at any time: necropsy confirmed gastroenteritis; pancreatic histology was normal and death was attributed to 'metabolic causes'. Cases 4, 5, and 6 received no insulin. Of the 5 survivors, 4 were considered physically and mentally normal after follow-up periods ranging from 6 months to 3 years. Case 5 is physically normal but may be slightly handicapped intellectually.

Discussion

Diabetes mellitus is rare in infancy and certainly less frequent than hyperosmolar dehydration. A transient diabetic state in infancy has been described (Hutchison, Keay, and Kerr, 1962), but our patients did not show the typical features of this condition, namely dysmaturity, polyuria, gross clinical dehydration, the need for insulin therapy for some months, and poor psychomotor development. Hyperosmolar nonketotic diabetes is well known, but our patients did not have diabetes mellitus. Hyperglycaemia occurs in cerebral abnormalities, e.g. head injury, epilepsy, encephalitis, tumour, abscess, and in asphyxia, acidosis, burns, and ether anaesthesia.

Hyperglycaemia has been recorded in non-diabetic infants with hyperosmolar dehydration (Bruck, Abal, and Aceto, 1968; Burman and

TABLE
Initial laboratory findings

Case no.	Age	Sex	Plasma						Urine	
			Na (mEq/l.)	K (mEq/l.)	Cl (mEq/l.)	HCO ₃ (mEq/l.)	Glucose (mg/100 ml)	Urea (mg/100 ml)	Clinitest (%)	Acetest
1	7 wk	F	189	5.6	151	11.5	380	522	1	Nil
2	11 wk	M	174	5.6	121	9.5	620	375	2	Nil
3	8 mth	F	151	5.1	—	10.0	670	102	1	Nil
4	11 dy	F	173	5.8	132	9.4	431	138	1	Nil
5	22 mth	M	173	5.0	140	—	540	260	2	Nil
6	5 mth	M	164	6.1	128	8.8	492	185	2	Nil

Glaspole, 1964; Keidan, 1955; Stevenson and Bowyer, 1970). Our own experience suggests that it occurs more frequently than was previously suspected. Personal discussion with many paediatricians on both sides of the Atlantic suggests that neither its existence nor the danger of its treatment with insulin is widely recognized.

The aetiology is unknown. Intravenous glucose has been suggested as a cause, but in all our patients the high glucose levels were present before any intravenous fluids were given. Severe acidosis inhibits insulin response at cellular level and defective glucose utilization seems likely in hyperosmolar dehydration.

The hyperglycaemia in our patients proved remarkably insulin-sensitive and, in retrospect, insulin was unnecessary. Insulin in standard dosage ($\frac{1}{2}$ unit/kg) produced significant hypoglycaemia in Case 2, as it did in the case described by Burman and Glaspole. In Cases 4, 5, and 6 we were able to observe the satisfactory fall in blood glucose when the hyperosmolar dehydration was treated by adequate fluid therapy alone.

Moreover, the raised blood glucose contributes to hyperosmolarity. It is generally agreed that *rapid* correction of fluid and electrolyte imbalance is dangerous in hyperosmolar states and may result in convulsions. Recent work (Clements, Prockop, and Winegrad, 1968) suggests an inherent potential for the development of increased intracranial pressure when blood glucose and plasma osmolarity of hyperglycaemic animals are *rapidly* returned to normal. Cerebral oedema occurring during treatment of nonketotic hyperglycaemia has been recorded in man (Maccario and Messis, 1969) and confirms the experimental evidence of an asynchronous decrease in blood and CSF glucose with a resultant shift of water from the plasma into the CSF and brain cells, though clearly this is not the sole explanation of convulsions in such patients.

The finding of hyperglycaemia in hyperosmolar dehydration, therefore, and its incorrect treatment

with insulin assume an importance not previously suspected. The risk of serious hypoglycaemia and the possibility of producing cerebral oedema with convulsions make insulin therapy both dangerous and unnecessary in the hyperglycaemia of hyperosmolar dehydration, a condition which is already life-threatening.

Summary

Hyperglycaemia was noted in 6 children with hyperosmolar dehydration resulting from gastroenteritis. In 3 of these 6 diabetes was erroneously diagnosed, and the danger of insulin therapy in such patients is stressed.

We thank Drs. R. R. Gordon, W. Henderson, M. W. Arthurton, and B. A. M. Smith for permission to study their patients; Dr. A. J. N. Warrack for the necropsy report on Case 3; Professor R. S. Illingworth for encouragement and guidance; and Mrs. Joan Barnby for secretarial help.

REFERENCES

- Bruck, R., Abal, G., and Aceto, T. (1968). Pathogenesis and pathophysiology of hypertonic dehydration with diarrhea. *American Journal of Diseases of Children*, **115**, 122.
- Burman, D., and Glaspole, D. W. (1964). Alimentary hyperglycaemia simulating diabetic ketosis. *Lancet*, **2**, 208.
- Clements, R. S., Prockop, L. D., and Winegrad, A. I. (1968). Acute cerebral oedema during treatment of hyperglycaemia. *Lancet*, **2**, 384.
- Hutchison, J. H., Keay, A. J., and Kerr, M. M. (1962). Congenital temporary diabetes mellitus. *British Medical Journal*, **2**, 436.
- Keidan, S. E. (1955). Transient diabetes in infancy. *Archives of Disease in Childhood*, **30**, 291.
- Maccario, M., and Messis, C. P. (1969). Cerebral oedema complicating treated non-ketotic hyperglycaemia. *Lancet*, **2**, 352.
- Stevenson, R. E., and Bowyer, F. P. (1970). Hyperglycemia with hyperosmolar dehydration in nondiabetic infants. *Journal of Pediatrics*, **77**, 818.

H. HEGGARTY,* P. TRINDADE, and E. M. BRYAN†
Children's Hospital, Sheffield, and the County Hospital, York.

*Correspondence to Dr. H. Heggarty, Darlington Memorial Hospital, Hollyhurst Road, Darlington, Co. Durham.

†Present address: Department of Child Health, University of Leeds.