hydrocephalus. In a series of 70 such episodes, colonization was almost invariably caused by coagulase-negative staphylococci; application of Baird-Parker's classification scheme for micrococcaceae revealed that all such organisms recovered from colonized shunts belonged to his subgroup *Staphylococcus* II. Because this subgroup was found very commonly in skin and nasal cultures from hospital patients and staff, it was necessary to devise a scheme capable of distinguishing biotypes within this subgroup.

The biotyping procedure revealed that (1) more than one biotype may occasionally coexist in the shunt, ventricles and blood of these patients; (2) successive recolonization of replaced shunts is not necessarily caused by the same biotype; and (3) the colonizing biotypes are almost always present at at least one body site, either skin, faeces, or nose.

The significance of these observations is discussed.

AN LDH ISOENZYME ANOMALY

E. L. PEEL (*Stepping Hill Hospital, Stockport*) The ensuing is a preliminary report on a case presenting an unusual anomaly in the pattern of serum lactate dehydrogenase isoenzymes. Many anomalies in such patterns have been reported in recent years, but in general these have involved either the presence of one or more additional components or a component with an altered electrophoretic mobility. In the present case, however, four of the usual five isoenzymes are lacking.

The patient in question, a farmer's wife aged 41, was

admitted to hospital with severe chest pain suspected of being due to a myocardial infarction. The initial ECG was equivocal but a later one showed no evidence of infarction. The serum transaminases (both GOT and GPT) and serum lactate dehydrogenase were all raised to approximately five times the upper limit of normal, but, contrary to the normal trend in myocardial infarction, these levels showed no significant change during the first two weeks of illness. The serum LDH isoenzyme pattern run on cellulose acetate was remarkable in showing only a single component, this being in the LDH₅, *ie*, M₄, position thereby indicating the absence of those isoenzymes containing 'H' subunits.

A possible explanation of this abnormality was sought. (1) The existence of a familial abnormality was virtually ruled out by the finding of normal isoenzyme patterns in the patient's four children. (2) A haemolysate of the patient's washed red blood cells revealed a normal isoenzyme pattern, *ie*, having a preponderance of the fast-moving components, thereby indicating the patient's ability to produce H subunits and suggesting the presence in the patient's serum of an inhibitor or antibody to these H subunits. (3) The latter possibility was supported by demonstrating the suppression by the patient's serum of the fast-moving isoenzymes of control red cell haemolysates and of sera from cases of myocardial infarction.

The presence of a circulating antibody would seem to be the most feasible explanation of the anomaly, but the possible origin of such an antibody at present remains obscure.

Investigation of the serum immunoglobulins is at present being pursued.