for glucose and reducing substances, to see if this concurs with the presumptive diagnosis of hyperglycaemia.

Clinicians should exercise caution when using POCT instruments, as they are intended to supplement, rather than replace laboratory testing.

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Continuing danger of glucose point-ofcare test devices in the neonatal setting

To the Editor: We write to alert medical and nursing staff to a continuing problem in the neonatal setting, which has featured in the correspondence pages of the *SAMJ* on at least two prior occasions.^[1,2] Recently, in KwaZulu-Natal province, at least 4 infants with galactosaemia have been affected by the phenomenon of galactose interfering with glucose meters and producing high readings, resulting in the misdiagnosis of hyperglycaemia.

Unlike some developed countries, there is no routine newborn screening programme for galactosaemia in South Africa (SA), with the result that the correct diagnosis is made only after significant illness has developed. A recent study in SA estimated the incidence in African patients to be approximately 1/14 400;^[3] accordingly, it is likely that many cases are undetected.

Often, blood sampling for important laboratory tests is technically extremely difficult in the sick neonate and point-of-care test (POCT) devices are used, which only require a few μ l of whole blood. The most ubiquitous of these is probably the handheld glucometer (Roche Accuchek is the instrument typically supplied to public hospitals). Under optimal conditions of careful usage and quality control, such POCT devices are extremely useful for clinical management. However, many use the glucose dehydrogenase pyrroloquinoline quinone (GDH-PQQ)-based glucose measuring system. While physiological levels of plasma galactose, at the clinically relevant concentrations found in galactosaemia, is able to cause positive interference. The result is the misdiagnosis of hyperglycaemia. Levels of galactose >0.83 mmol/l will cause overestimation of glucose levels.

In the cases referred to above, neonates were judged to have 'hyperglycaemia' and, based on this finding, were treated with insulin, with disastrous consequences. Galactosaemia, in fact, often results in hypoglycaemia caused by hepatic accumulation of galactose-1phosphate, which inhibits enzymes of glycogenolysis; including glucose-6phosphatase, glucose-6-phosphate dehydrogenase, phosphoglucomutase and glycogen phosphorylase. The inadvertent administration of insulin to such affected neonates, in whom blood glucose is already dangerously low, obviously has the effect of lowering glucose levels further.

It is of critical importance that high readings from POCT glucometers that suggest hyperglycaemia in neonates are confirmed by a formal measurement of glucose in the laboratory before treatment is instituted. The clinician is well advised to check the urine