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improbable in view of the fact that the cord had been clamped at thirty seconds of delivery. Neonatal polycythemia may also be encountered following chronic fetal hypoxia. However, baby N was not only well grown but had no history of fetal distress and had not required resuscitation. As a result a search was made for the possibility of materno-fetal transfusion. This was complicated by the fact that both mother and baby’s major blood groups were identical, Group A, Rh positive. The late Dr. Geoffrey Tovey (Fig 4), Director of the National Blood Transfusion Service based at Southmead Hospital took a special interest in this case and undertook the differential agglutination tests.

While microscopic leaks of erythrocytes between maternal and fetal circulations were well recognised at birth and as a cause of sensitization to the Rhesus factor, massive materno-fetal transfusion was a condition that I had not recognised during a decade working in maternity hospitals in spite of a particular interest in neonatal polycythemia. As a result I presented this case to a clinical meeting of senior paediatricians in October, 1969. None had previously encountered such a case. However, a subsequent search of the literature revealed that the condition had been described first in 1961 by Michael and Mauer. A year later Andrews and Thompson had reported a further two cases and suggested that materno-fetal transfusion might indeed not be a rare phenomenon. Be that as it may, massive materno-fetal transfusion still remains very unreported as a cause of neonatal polycythemia.

REFERENCES