Table 1 Infant characteristics

	Group 1 (n=105)	Group 2 (n=92)
Median gestation (range)	39 weeks (37–41)	39 weeks (37–41)
Median age at review (range)	17 days (13–56)	18 days (13–49)
Sex	Male 76	Male 53
	Female 29	Female 39
Mode of feeding	Breast 84 (80%)	Breast 74 (80%)
	Bottle 9	Bottle 8
	Mixed 12	Mixed 10

Table 2 Investigation algorithms for each group of infants

Group 1 (n=105)	Group 2 (n=92)
Full blood count	Split bilirubin
Blood group and Coomb's test	G6PD where ethnically appropriate
Thyroid function tests	
Split bilirubin	
Liver function tests	
Urine culture	
Urinary reducing substances	
G6PD where ethnically appropriate	

G6PD, glucose-6-phosphate dehydrogenase.

NICE recommendations for the formal assessment of babies with prolonged jaundice: too much for well infants?

Prolonged jaundice (PJ) is common, affecting 2–15% of all neonates and up to 40% of breastfed infants.[1] It presents a challenge to health professionals, who must identify those infants with pathology while avoiding the unnecessary investigation of normal babies. National Institute for Health and Clinical Excellence (NICE) recently recommended, that in addition to a thorough examination, the formal assessment of PJ should include conjugated bilirubin, urine culture, glucose-6-phosphate dehydrogenase where ethnically appropriate, full blood count, blood group determination and Coomb's test.[2]

The three studies referenced had methodological flaws, including all being retrospective studies. One UK study concluded that while neonates with PJ may have detectable problems, the number of investigations could safely be reduced if a thorough examination was performed.[1] However, prospective audit was not undertaken. The two remaining Turkish studies[3 4] had a high incidence of established conjugated hyperbilirubinaemia (66%). Unsurprisingly, this was associated with a higher rate of pathology and unlikely to be typical of a population of well, thriving babies.

We recently performed a prospective study of the investigation of 'well' term neonates referred from community for assessment of PJ. History and physical examination was performed and demographic data obtained (table 1). Existing local guidelines were followed during the first half of the audit (table 2, group 1) and following interim analysis a rationalised approach to investigation was introduced (table 2, group 2), derived from the British Society of Paediatric Gastroenterology, Hepatology and Nutrition guidelines for the investigation of hyperbilirubinaemia and recommendations from the UK Children's Liver Disease Foundation.[5 6] We opted not to include urine culture in our rationalised approach for two reasons – antenatal ultrasound screening was routine in our health board and likely to detect any structural renal malformations, and also the literature supporting routine urine testing is inconsistent.[7 8]

One hundred and ninety-seven of 12 986 live births (1.5%) were referred for assessment of PJ. No significant pathology associated with PJ was detected. The number of repeat investigations (37 vs 7, p<0.0001) and return appointments (28 vs 7, p=0.0009) fell following the introduction of the rationalised investigation algorithm.

Our data suggest that in screening of well, term neonates who are thriving, a streamlined approach may be safe and reduce unnecessary workload. It is difficult to justify further investigations in this cohort, which frequently generates repeat testing for little diagnostic gain. We acknowledge that our study was limited not only by its small numbers, but also by the low referral rate for infants with PJ. However, we feel it is reasonable to call for better evidence to justify all the investigations recommended in the NICE guidelines.

M E Rodie,[1] A Barclay,[2] C Harry,[3] J Simpson[4]

[1]Department of Child Health, Royal Hospital for Sick Children, Glasgow, UK
[2]Department of Gastroenterology, Royal Hospital for Sick Children, Glasgow, UK
[3]Community Paediatrics, Rainbow House, Ayrshire Central Hospital, Irvine, North Ayrshire, UK
[4]Neonatal Intensive Care Unit, Royal Hospital for Sick Children, Glasgow, UK

Correspondence to Dr Martina Rodie, Department of Child Health, Royal Hospital for Sick Children, Dalnair St, Yorkhill, Glasgow G3 8SJ, UK; martinarodie@hotmail.com

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Accepted 24 August 2010
Published Online First 27 October 2010

Arch Dis Child 2011;**96**:111–112.
doi:10.1136/adc.2010.199984

REFERENCES

1. **Hannam S,** McDonnell M, Rennie JM. Investigation of prolonged neonatal jaundice. *Acta Paediatr* 2000;**89**:694–7.
2. National Institute for Health and Clinical Excellence. Neonatal Jaundice. Clinical Guideline 98, 2010. http://www.nice.org.uk/CG98 (accessed 1 September 2010).
3. **Unal S,** Koc E, Aktas A, *et al.* Prolonged jaundice in newborns: what is it actually due to? *Gazi Med J* 2003;**14**:147–51.
4. **Tiker F,** Tarcan A, Kilicdag H, *et al.* Early onset conjugated hyperbilirubinemia in newborn infants. *Indian J Pediatr* 2006;**73**:409–12.

5. British Society of Paediatric Gastroenterology, Hepatology and Nutrition. Guidelines for Investigation of Conjugated Hyperbilirubinaemia, 2007. http://bspghan.org.uk/document/liver/InvestigationofNeonatalConjugatedhyperbilirubinaemia.pdf.
6. Children's Liver Disease Foundation. Early Identification and Referral of Liver Disease in Infants: Yellow Alert Campaign, 2007. http://www.childliverdisease.org.
7. **Sarici SU,** Kul M, Alpay F. Neonatal jaundice coinciding with or resulting from urinary tract infections? *Pediatrics* 2003;**112**:1212–13; author reply 1212–13.
8. **Maisels MJ,** Newman TB. Neonatal jaundice and urinary tract infections. *Pediatrics* 2003;**112**:1213–14; author reply 1213–14.

1