In the present case the bilateral fractured patellae cannot be directly related to the cauda equina lesion, as no part of the reflex quadriceps arc was found defective. His fractures were undoubtedly due to faulty balance caused by muscular weakness below the knees.

J. PETT TURNEY, F.R.C.S.

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Chronic Intermittent Juvenile Jaundice

Meulengracht (1947) published a review of 35 cases of chronic intermittent juvenile jaundice, and a further case was described by Parkes Weber (1948). This condition must be considered in the differential diagnosis of mild jaundice and is probably more common than the number of published cases suggest, for three cases have recently been seen in this hospital. The familial incidence in two of these and the different modes of presentation may be features of interest. The biochemical and haematological investigations were repeated in each case at different times, but only one set of typical results for each subject is given in the Table.

Test	Method and Normal Range	Case				
		1a	2a	2b	2c	3
Blood:						
Haemoglobin (g./100 ml.)	Haldane	13.3	16.2	15.9	18.2	14.8
Haematocrit (mm./100 mm.)		40.0	48.0	—	52.0	44.5
Reticulocytes		0.3%	<1%	0.8%	0.4%	0.4%
Van den Bergh (direct)		Weak, pos.	Pos.	Pos.	—	Weak, pos.
Total bilirubin (mg./100 ml.)	Evelyn and Malloy. <0.75	2.0	4.1	2.7	4.0	1.8
Phosphatase (units)	King and Armstrong. 3.7-13.1	5.9	5.7	7.1	—	9.6
Thymol turbidity (units)	Maclagan. <4	3	1	1	—	1
Kunkel's turbidity (units)	<4	2	2	4	—	1
Plasma proteins (total) (g./100 ml.)	Micro-Kjeldahl. 5.6-8.5	6.25	—	—	—	6.07
Albumin (g./100 ml.)	3.4-6.7	4.70	—	—	—	4.25
Globulin (g./100 ml.)	1.2-2.9	1.55	—	—	—	1.82
Red-cell fragility (% saline)	Commenced lysis 100% lysis	0.46 0.28	0.46 0.28	0.46 0.28	"Normal"	0.44 0.28
Direct Coombs test		Neg.	—	—	—	Neg.
Faeces:						
Stercobilinogen (mg./24 hr.)	Schwartz, Sborov, and Watson. 40-280	125	—	—	—	69
Urine:						
Urobilinogen	Ehrlich	0	Tr.	Tr.	—	0
Urobilin	Schlesinger	0	—	—	—	0
Bilirubin	Fouchet	0	+	++	—	0
Bile salts	Hay	0	0	0	—	0

CASE REPORT

Case 1a.—A woman aged 20 complained of nausea and lassitude. On examination her conjunctivae were slightly yellow, but on physical examination she was otherwise normal, neither her liver nor spleen being palpable. Recurrent attacks of jaundice had occurred in the past, but these had spontaneously improved as did this episode. There was no significant change in the physical and laboratory findings during a further attack some six months later. **Case 1b:** Her brother, aged 24, had experienced two similar attacks of jaundice in addition to prolonged neonatal physiological jaundice. When seen, he was normal physically and his blood and urine showed no abnormality on laboratory testing. Opportunity has not occurred for laboratory investigations to be made during an icteric phase.

Case 2a.—A man aged 29 suffered attacks of jaundice with pain in the right hypochondrium at the ages of 7 and 17. During a similar attack, when aged 23, he had a cholecystectomy at another hospital, when "20 to 30 small brown currant-like stones" were removed. Despite this his jaundice persisted, but he was free from pain for five years. He was referred for investigation because of another attack which had been present for about two months. On examination he was slightly jaundiced though free from any other abnormalities apart from his operation scar.

Case 2b: His brother, aged 27, was first noticed to be jaundiced when aged 12 and has been so since. Otherwise he is normal and suffers no inconvenience. **Case 2c:** The third brother, seen elsewhere, has very slight jaundice, said to have been present from birth. No other members of this family are known to have shown similar symptoms.

Case 3.—A man aged 25 was noticed to have an icteric serum during routine bleeding for antibody investigations. He had been perceptibly jaundiced for as long as he could remember, but had no other symptoms. His condition was unchanged over a period of more than a year. There was no relevant family history.

COMMENT

Each case presented in a different guise. Case 1a was investigated because of recurrent attacks of jaundice accompanied by nausea and lassitude, and the possibility of a subacute hepatitis of insidious onset as described by Kelsall, Stewart, and Witts (1947) was considered. The normal laboratory findings, with the exception of raised serum bilirubin, in conjunction with the long history and the suggestive history of her brother confirmed the present diagnosis. Case 2a had already undergone cholecystectomy and when first seen the possibility of familial haemolytic jaundice was considered. Once more the haematological findings excluded this, the biochemical findings were equivocal and could be interpreted as those of a hepatitis, but again the long history,

the absence of abnormalities in turbidity tests and the alkaline-phosphatase tests in conjunction with the family history led to the diagnosis of chronic intermittent juvenile jaundice. The appearance of bilirubin in the urine of Case 2a and one of his brothers is presumably related to a higher plasma concentration of bilirubin in these cases, though Vickers (1950) emphasized that bilirubin cannot be considered a threshold substance. The third case was asymptomatic, and a slightly raised total serum-bilirubin was the only laboratory abnormality.

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A. P. H. RANDLE, M.B.,
Medical Registrar.

M. J. H. SMITH, M.Pharm., Ph.D., F.R.I.C.,
Assistant Chemical Pathologist.

B. W. MEADE, M.B.,
Demonstrator in Pathology.

King's College Hospital, London, S.E.5.

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