PAPERS AND ORIGINALS

Very low birth weight children at school age: comparison of neonatal management methods

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Summary and conclusions

The outcome of 293 infants born to a geographically defined community and weighing 501-1500 g was investigated. Medical intervention in the newborn period had been avoided. Morbidity was assessed at school age. Of the infants, 236 had been live born in the labour ward of this hospital; of these, 117 (49·6%) died in the neonatal period, one (0·4%) died in the first year, four (1·7%) were untraced, 13 (5·5%) had major handicap, 29 (12·3%) had minor handicap, and 72 (30·5%) were considered to be normal. In terms of survival, handicap, and intellectual capacity at school age the outcome compared favourably with that of infants born over the same period (1963-71) in areas where intensive methods of perinatal care were used.

These results imply that postnatal survival and potential of infants of very low birth weight are by no means prejudiced when only experienced nursing care is available.

Introduction

Obstetricians, midwives, paediatricians, and family doctors are frequently confronted with the premature delivery of babies of very low birth weight ($\leq$1500 g). In a maternity unit with 3000 deliveries a year this occurs on average once a fortnight. Although more infants of very low birth weight now survive than formerly,[1] some evidence suggests that handicap in survivors is little different from what it was before neonatal intensive care methods were introduced in the 1950s[2 3] and that minimal intervention does not prejudice survival at least of the smallest infants.[4] On the other hand, reports of lower mortality and rates of handicap in centres where intensive neonatal care is practised seem to carry more weight,[5 6] and since many reports attribute the improved prognosis to advances in medical technology, skilled intervention, and a better understanding of perinatal pathophysiology, the extension of intensive care facilities for such infants is urged.[7 8] Reports of controlled trials showing long-term benefit from intensive care of babies of very low birth weight, however, are few. This is perhaps partly because belief in the benefits of intensive care methods would seem to render some trials unethical, numbers are necessarily small, and treatment programmes change so often. Regional referral centres are therefore able to compare their results only with those of previous years, those from similar centres, or national or regional statistics for the years studied.

In Mansfield in 1973 one of us (CRM) inherited a unit where standards of newborn nursing care and record keeping had been consistently high while conscientious medical supervision had been almost exclusively of a non-interventionist type. Between 1963 and 1971 the unit's consultant and senior nursing staff had not changed, and its policy had changed only in respect of the timing of the first feed. We were therefore able to examine the outcome of infants of very low weight who did not receive "special" or "intensive" care, using the same standards and definitions for live birth, handicap, social class, and gestation-birthweight factors[9] and the same tests for assessing intelligence and learning difficulties as were used by Jones *et al*[10] in an analysis of the experience of the neonatal intensive care unit at Hammersmith Hospital, London.

Patients and methods

Between May 1963 and July 1971, 293 infants weighing 501-1500 g were born in Mansfield and district. All but 57 were delivered in the labour ward of this hospital. Six infants born in the hospital showed only transitory signs of life, but the remainder were admitted to the adjacent baby nursery within three to 20 minutes of birth.

Most mothers had had some antenatal care, but none in premature labour was referred elsewhere. If spontaneous delivery appeared to

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be uncomplicated medical staff were not routinely called to the labour ward. The attending midwife treated birth asphyxia by aspiration of the airways and gave oxygen by face mask without positive-pressure ventilation. Stimulus to the infants' feet, intragastric oxygen, and postural manoeuvres[11] were also occasionally used. Only 13 infants (4·4%) were intubated. One received mouth-to-mouth ventilation.

In the nursery minimal handling and disturbance were considered to be paramount. Incubators were used to maintain body temperature (measured rectally by mercury thermometer), the mean rectal temperature during the first 10 days in survivors being 35·8±0·47°C. Feeds with half-strength, half-cream National Dried Milk were begun when the nursing staff considered it to be prudent and were delivered by intermittent oesophageal gavage[12] (using rubber tubes). Forty-one infants who were small for gestational age received an appreciably higher mean energy intake/kg body weight (1·6±SD 0·6 MJ; 373±142 kcal) than 90 who were of appropriate weight for gestation (1·2±0·4 MJ; 292±92 kcal) during the first seven days. Almost all infants received vitamin K after birth, three days of intramuscular crystalline penicillin or oral ampicillin in the early neonatal period, a multivitamin preparation after 10 days, and oral iron after three weeks.

Added oxygen was given to 74 of the survivors in concentrations about 5% more than that required to abolish cyanosis. The average duration of treatment was less than three days, and concentrations of more than 40% do not seem to have been given to survivors. Inspired oxygen concentrations were only occasionally sampled, however, using a Beckman oxygen analyser. Apnoea monitors were not used. Two infants underwent radiography; blood glucose concentrations were estimated in two and serum bilirubin concentrations in two. Two infants underwent repeated exchange transfusions for rhesus incompatibility and one died elsewhere after referral for an operation for oesophageal atresia. No other investigation was carried out. Apnoeic attacks were treated by the same methods as birth asphyxia.

The clinical records indicated the feeds given, the rectal temperature, and the occurrence of respiratory distress, apnoeic attacks, and jaundice. Jaundice was not precisely defined.

There were 137 long-term survivors, who were reviewed between 1977 and 1979. About three-quarters of the parents and children agreed to visit the hospital; the remainder were examined either at home or at school. Two were living abroad but reports were received from them, and four could not be traced although there were medical records of their attendances for review at 12 months of age. Thus, 131 (95·6%) of the survivors were seen. At review the youngest child was aged 6 years 5 months and the oldest 16 years. Clinical examination included a neurological assessment based on a selection of tests scored for age,[13] a test of visual acuity, and examination of optic fundi without mydriatic. Most of the children had undergone group audiometric testing at their school medical examination, and the results of this were available, together with results of any further tests performed in the hospital ear, nose, and throat department. Pure-tone audiometry had been undertaken when indicated. One hundred and twenty-two children were assessed by intelligence tests (Wechsler) and by the Neale reading analysis, and further information on scholastic achievement was obtained from the children's schoolteachers, who also completed the Rutter behaviour questionnaire.[14]

Respiratory distress was defined as the presence of two of the following features after 4 hours of age, or earlier if death had occurred before that time: grunting respirations, costal recession, and respiratory rate of over 60 per minute. Major handicap was defined as severely impaired intelligence (IQ less than 70), cerebral palsy, or a visual or hearing defect severe enough to require special schooling. Minor handicap was defined as any other neurological abnormality or an IQ between 70 and 84, or both. Differences in comparisons are reported as significant when the χ^2 test for frequencies or the t test for means of normally distributed values yielded $p<0·05$.

Results

Table Im gives the birth weights, gestational ages, and obstetric and perinatal details of the neonates who died and the survivors. Of the 154 neonatal deaths, 115 (74·7%) occurred in the first 24 hours and two (1·3%) after the first week. Necropsy was performed on all but 13. Hyaline membrane disease and intraventricular haemorrhage were the most common findings post mortem. One very immature infant and one who underwent exchange transfusion for rhesus incompatibility showed yellow staining of the basal ganglia. Necrotising enterocolitis, heart failure secondary to patent ductus arteriosus, bronchopulmonary dysplasia, hydrocephalus without spina bifida, and milk aspiration were not recorded, though unilateral pneumothorax was found in one intubated infant. Two infants died suddenly and unexpectedly at two and four months of age respectively.

Reports of the two children living abroad suggested that they were physically and intellectually normal. The four untraced children had shown normal development at 12 months of age. The following information refers to the 131 survivors who were examined at school age.

Physical development—At review most of the children without major handicap were of normal physique and appearance. Fourteen were below the third centile for weight (10 had been small for gestational age at birth), nine were below the third centile for height (six had been small for dates), and 13 were below the third centile for head circumference (10 small for dates). One child had had an elective operation for a patent ductus arteriosus at 4 years of age, and another had a patent ductus that had not been operated on. Three had had operations on the middle ear.

Handicap—There was no significant difference in the incidence of major and minor handicaps between infants who had been fed early or late after birth (table IIm). Table IIIm gives details of the 18 children who had major handicaps. One child had cicatricial retrolental fibroplasia, but none was considered to have kernicterus. Thirty-one children had minor handicaps, of whom six had minor neurological signs; the other 25 were physically normal but of below average intelligence (IQ 70-84). Further information on these children with minor handicaps is available from us. Fourteen children had a squint, of whom six also had major handicaps. Amblyopia secondary to delayed treatment of the squint was found in five. Three children had sustained hearing defects; of these, two had nerve deafness or mixed conductive and nerve deafness, in one case severe enough to require special schooling.

Intelligence—Table IVm shows the distribution of intelligence quotients as assessed with the Wechsler test of 122 of the children.

MINIPRINT TABLES I-III

Im

TABLE I—*Birth weight, gestational age, social class, and obstetric and neonatal details*

	Survivors (n = 139)	Deaths* (n = 154)
Mean (±SD) birth weight (and range) (g)	1250 ± 160 (822 ± 1500)	1060 ± 240 (501 ± 1500)
Mean (±SD) gestational age (weeks)	31·2 ± 2·6	28·8 ± 3·7
Male infants	57	82
Non-Caucasian	1	1
Social class†: I, II, III (non-manual)	12	—
III (manual), IV, V unemployed	119	—
Antepartum haemorrhage	24	28
Pre-eclamptic toxaemia	16	12
Multiple pregnancy	28	26
Breech	31	20
Shirodkar suture	1	5
Caesarean section: Placenta praevia	0	3
Other reasons	2	5
Forceps (vertex deliveries only)	5	4
Episiotomy	25	19
Intubation for birth asphyxia	3	10
Central cyanosis on admission to nursery	36	40
Respiratory distress	32	19
Recurrent apnoea causing cyanosis: Responsive to tactile stimulus	25	23
Unresponsive to tactile stimulus	9	11
Convulsions	5	9

*Deficient information in nine records.
†Only survivors examined; Registrar General's classification used.

IIm

TABLE II—*Mean (±SD) delay in feeding in 131 long-term survivors, and numbers with handicap*

Years of study	No of long-term survivors	No with handicap: Major	No with handicap: Minor	Interval between birth and first feed (hours)
1963–6	69	11	16	21·9 ± 12·7* (n = 67†)
1967–71	62	7	15	8·7 ± 4·7* (n = 59†)

*Difference in means significant: $p<0·01$.
†Remaining records yielded deficient information.
$\chi^2 = 0·58$; df = 2; NS.

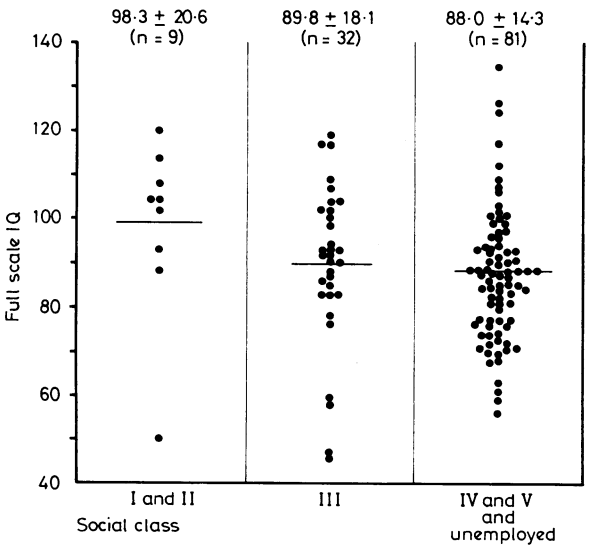
IIIm

TABLE III—*Details of 18 children with major handicap*

Case No	Birth weight* (g)	Maternal pregnancy factors	Neonatal details	Handicap	Type of school
Twin 3	1160, AFD	Second twin. Spontaneous vertex delivery	Convulsions and apnoeic attacks	Spastic cerebral palsy, amblyopia left eye. IQ 76	Physically handicapped
4†	1190, SFD	Spontaneous vertex delivery	Respiratory distress syndrome	Severe right hemiparesis after convulsive illness at 9 months. ? venous sinus thrombosis. IQ 50	Educationally subnormal (severely)
9	1100, AFD	Spontaneous vertex delivery	Received 30% oxygen for 24 hours	Severe myopia. Nystagmus; pallor of discs, tortuosity of vessels on fundoscopy. Squint	Partially sighted
22	1200, SFD	Severe toxaemia. Blood pressure 200/130 mm Hg. Oligohydramnios. Artificial rupture of membranes. Infarcted placenta		Delayed development. Athetoid cerebral palsy. IQ 47. Squint	Educationally subnormal (severely)
Twin 10	1200, AFD	Second twin, first born at home. Version and breech extraction	Gasped aged 8 minutes, regular respirations after 10 minutes	Moderate myopia. Impaired balance in lower limbs. IQ 68	Normal (coping)
30	‡1220, AFD	Antepartum haemorrhage. Spontaneous vertex delivery	Severe jaundice	Moderately severe left hemiparesis. Squint. IQ 61	Educationally subnormal (severely)
40	820, AFD	Spontaneous vertex delivery		Single convulsion aged 6 months. Delayed development subsequently. IQ 46	Educationally subnormal (severely)
47†	1320, SFD	Spontaneous vertex delivery	Born 65 minutes before admission. Received 30% oxygen for less than 24 hours	Blind. Retrolental fibroplasia	Blind
Twin 14†	1500, AFD	Probable vertex. Born unattended		Mental retardation. IQ 59	Normal (? coping)
Twin 15†	1200, ? SFD	Probable breech. Born unattended		50 db hearing loss after exploration of right ear for cholesteatoma. IQ 62	Normal (coping; ? underestimate
51	1100, SFD	Severe hypertension; blood pressure 200/140 mm Hg and albuminuria. Artificial rupture of membranes. Vertex	Mild apnoeic attacks	Impaired co-ordination of hands and fingers. IQ 59	Educationally subnormal (severely)
62	1240, AFD	‡ APH. Spontaneous rupture of membranes. Prolapsed cord. Breech		Mild spastic diplegia. IQ 81	Normal
73	1470, SFD	2 previous infants had exchange transfusions for Rh incompatibility. Liquor showed this infant severely affected. Artificial rupture of membranes. Vertex	Exchange transfusion within 2 hours of birth. Repeated at 36 hours	Severe bilateral nerve deafness	Deaf
82	1000; SFD	Breech delivery		Mild impairment of balance in both lower limbs. IQ 86	Normal. No attendance for two years
91	1100, AFD	Spontaneous vertex delivery. Infarcted placenta	Very feeble respiratory efforts during first 3 hours	Impairment of fine movements in upper limbs and balance. Squint. IQ 55	Educationally subnormal (moderately)
Twin 25	1310, SFD	Undiagnosed twin. Spontaneous vertex delivery		Spastic cerebral palsy. IQ 56	Normal (coping)
Twin 26†	1440; SFD	Undiagnosed twin. Spontaneous vertex delivery		Mental retardation. IQ 58	Educationally subnormal (moderately)
100	1460; AFD	Spontaneous vertex delivery	Respiratory distress syndrome and severe apnoeic attacks	Spastic cerebral palsy. Squint. Verbal IQ 72	Physically handicapped

*AFD = Birth weight appropriate for gestational age. SFD = Small for dates.
†Born outside hospital.
‡APH Antepartum haemorrhage.

The mean score on the performance scale of infants who were small for gestational age was significantly inferior to that of the infants born with weights appropriate for gestational age. Nineteen of the total sample showed a discrepancy of 15 or more points between verbal and performance scores. The distribution according to social class (figure) serves to emphasise that many of these children were born into disadvantaged families. Nine children were not tested by Wechsler intelligence scale and reading analysis; teachers' reports were available as a basis for the information assembled on them (table Vm).



Full scale intelligence quotients (Wechsler test) of children of very low birth weight related to social class. Mean (±SD) values also given.

Learning difficulties—Poor reading performance was characteristic of the group as a whole, but no reliable norm was known for the schools of the region. The reading performance of 63 children tested by the Neale reading analysis was two or more years below average, a finding consistent with their verbal intelligence. Ten were non-readers (IQ range 46-90), and 34 showed three years' reading retardation.

Behaviour—School teachers completed the Rutter behaviour questionnaire for 130 children. Although the questionnaire was not designed for the entire age group in this study, it documents teachers' perceptions on such factors as overactivity, abnormal muscular movements, anxiety, unjustifiable absence from school, attention span, sphincter control, and stammering or other speech defect. No specific undesirable pattern of behaviour, emotional disturbance, or neurosis was apparent. Maladjustment was not reported. Teachers from many different schools, however, indicated that poor concentration and attention span were present in 48 and distinctive features in 35. This problem is thus perhaps nine times more common in children of very low birth weight than in British children of all birth weights,[15] presenting either as an isolated disability or compounding other handicaps.

Discussion

To compare the results of the Mansfield births with those of other centres we reviewed reports in which live birth was defined in a comparable way and 95% or more survivors were examined[10 16-20] (and P A Davies, personal communication on Hammersmith Hospital data, 1979). We also attempted to classify and compare morbidity (reported in various ways) according to other previously defined criteria.[6 18 21] Although the Mansfield children rarely received neonatal medical intervention, such comparison shows that their rates of neonatal survival and postneonatal death were favourable, the proportion with major handicap was similar to that in children born in other centres, and differences in the incidences of retrolental fibroplasia and hearing defect were negligible. Only the Hammersmith Hospital analysis,[10] however, attempted to assess the impact of neonatal intensive care on a community-derived cohort of children of very low birth weight at school age. Table VIm compares the outcome in the different centres.

Neonatal mortality was higher (though not significantly so) in infants born at King's Mill Hospital than in those born at University College Hospital, London. The mortality at King's Mill Hospital, however, was significantly lower than that in infants born in the Hammersmith Hospital for the birthweight group as a whole and also for those of 1000 g or less. Nevertheless, despite increased medical staffing, neonatal intervention, and referral of some infants for respiratory support to "good units,"[7] there was no appreciable improvement in the neonatal mortality at King's Mill Hospital during the years after the study period (table VII).

We made no attempt to establish causal factors for differences in neonatal mortality nor can we say how much better (or worse) the Mansfield results might have been had the infants been

TABLE VII—*Neonatal mortality of infants weighing 501-1500 g at birth compared between hospitals*

Birth weight (g)	No of survivors	No of deaths	Mortality (%)
University College (1966-70): born in hospital only[18]			
501–1000	7	29	80·6
1001–1500	60	27	31·0
Total	67	56*	45·5
Hammersmith (1961–75): born in hospital only[10]			
501–1000	13	97†	88·2
1001–1500	135	112	45·3
Total	148	209*‡	58·5
Coventry (1973–4): born in and outside hospital[20]			
501–1000	8	30	78·9
1001–1500	39	21	35·0
Total	47	51	52·0
King's Mill (1963–71): born in hospital only			
501–1000	15	45†	75·0
1001–1500	104	72	40·9
Total	119	117‡	49·6
King's Mill (1972–8): born in and outside hospital			
501–1000	10	51	83·6
1001–1500	74	43	36·8
Total	84	94	52·8
King's Mill (1972–8): born in hospital only			
501–1000	9	28	75·7
1001–1500	71	39	35·5
Total	80	67	45·6

*$\chi^2 = 6{\cdot}2$, $p < 0{\cdot}02$. †$\chi^2 = 4{\cdot}8$, $p < 0{\cdot}05$. ‡$\chi^2 = 4{\cdot}6$, $p < 0{\cdot}05$.

MINIPRINT TABLES IV-VI

IVm

TABLE IV—*Distribution of full scale intelligence quotients (Wechsler test) in 122 children. (Figures are numbers (%) of children*

Birth weight	Intelligence quotient							
	< 55	55–69	70–84	85–99	100–114	115–125	125	Mean ± SD
Small for dates (n = 41)	2 (4·8)	4 (9·8)	7 (17·1)	17 (41·5)	10 (24·4)	1 (2·4)		87·5 ± 17·2
Appropriate for dates (n = 81)	1 (1·2)	4 (4·9)	22 (27·2)	35 (43·2)	12 (14·8)	6 (7·4)	1 (1·2)	96·5 ± 14·9

Vm

TABLE V—*Details of nine children not tested by Wechsler intelligence test and Neale reading analysis*

Case No	Type of school	Information from teachers or medical records
Twin 1	Normal	Low average academic performance.
Twin 2	Normal	Left school on 16th birthday for manual employment before test
9	For partially sighted	Average performance in academic and physical activities
19	Normal	Good academic performance: third in class of 10. Living abroad
23	Normal	Low academic attainment. Frequent absence from school. Died of aplastic anaemia before IQ testing
47	For blind	Average academic performance. Satisfactory progress in Braille, swimming, music
73	For deaf	Below average academic performance.
91	For educationally subnormal (moderately)	IQ 55 (Simon Binet test) when previously assessed
100	For physically handicapped	Only Wechsler verbal IQ scorable: 72

VIm

TABLE VI—*Comparison of outcome of very low birth weight born at King's Mill and other hospitals*

Units and years of birth	No (%) of neonatal deaths	No (%) of postneonatal deaths (29 days–3 years)	No (%) untraced	No (%) with major handicap (age at assessment)	No (%) with minor handicap: Minor abnormal physical signs	No (%) with minor handicap: IQ 70–84, no abnormal physical signs	No (%) normal	Total
King's Mill, 1963–71 (born in and outside hospital)	154 (52·6)	2 (0·7)	4 (1·4)	18 (6·1) (6 y 5 m–16 y)	6 (2·0)	25 (8·5)	84 (28·7)	293
Hammersmith, 1961–75** (born in hospital only)	209 (58·5)	5 (1·4)	5 (1·4)	19 (5·3) (23 m–19 y)	6 (1·7)	14 (3·9)	99 (27·7)	357†
King's Mill, 1963–71 (born in hospital only)	117 (49·6)	1 (0·4)	4 (1·7)	13 (5·5) (6 y 5 m–16 y)	6 (2·5)	23 (9·7)	72 (30·5)	236*
University College, 1966–70** (born in and outside hospital)	88 (44·7)	11 (5·6)	3 (1·5)	9 (4·6)‡ (2 y 10 m–7 y 10 m)	Undetermined		86 (43·7)	197
Coventry, 1973–4** (born in and outside hospital)	51 (52·0)	4 (4·1)	1 (1·0)	1 (1·0) (9 m–31 m)	Undetermined		41 (41·8)	98

*P A Davies, personal communication, 1979.
†$\chi^2 = 10{\cdot}57$, df = 3, $p < 0{\cdot}05$. (The two groups of children who died were considered together, as were the two groups with minor handicaps.)
‡Major handicap defined as disability that prevents or is likely to prevent a child from going to a normal school or seriously interferes with normal function in society.**
§Major handicap defined as IQ < 70, major or minor defect of central nervous system, or moderate to severe visual or hearing defect.**

delivered and reared differently or elsewhere. No authoritative information yet exists linking social class and neonatal mortality for children of very low birth weight, as exists for total births.[22]

In whichever of three ways major handicap is defined the outstanding point is the smallness of the difference in outcome between the two London neonatal units using intensive care methods and the Mansfield unit, which relied entirely on nursing judgments and procedures.

The proportion of Mansfield children with minor nervous-system signs differed little from that of the Hammersmith sample, and we consider it to be unlikely therefore that the neonatal nursing techniques prejudiced the children's later performance, even when starvation during the first 24 hours was practised.[23] Minor handicap defined as slow learner status (IQ 70-84) without nervous-system signs was more common and commensurate with the excessive number of Mansfield children born to parents of manual occupational class. Mean full scale intelligence quotients of the Mansfield children, however, did not differ significantly from those of a sample of children reared at Hammersmith Hospital when compared according to occupational class, nor when the survivors who had weighed 1000 g or less were compared. (Tabulated details are available from us as an appendix.) A discrepancy of 15 or more points between verbal and performance scores was no more common in the Mansfield than the Hammersmith sample. Learning difficulties were commonplace in both groups, though direct comparison is not valid.

Broadly, we think that educational attainment and social-class factors in the Mansfield children of very low birth weight closely parallel the findings in the 6·2% of socially disadvantaged British children of all birth weights.[24] We find it difficult, therefore, to escape the conclusion that scientific and highly skilled medical intervention in the years under discussion made little impact on the outcome for infants of very low birth weight. The results in Mansfield were achieved by using well-qualified nurses as supervisors with experienced but unqualified nurses helping, using a minimum of simple equipment, and by a few staff members getting to know the parents well; this combination is as readily found in some remote mission hospitals as in prestigious teaching institutions.

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ONE HUNDRED YEARS AGO Medical witnesses, it is well known, have often considerable difficulty in obtaining remuneration for a compulsory attendance and evidence on trials for murder. The sum allowed by the county is rarely adequate to cover the expenses incurred by a necessary residence in a town during the assizes. It is in the power of a judge to order the payment of a sum beyond the tariff to a medical witness; but this power is seldom exercised for the benefit of the profession; and it is extremely rare that a judge, in the absence of any special application by counsel, will of his own accord order additional fees to be paid. It is, therefore, with great satisfaction that we notice, in reference to the trial and conviction of three persons on a charge of murder, which has just taken place at the Lincoln Assizes, that the new judge (Mr Justice Stephen) has set a good example in this respect. The principal medical witness in this case was Dr H T Stiles of Spalding; and his evidence was considered so satisfactory that the learned judge, "in recognition of the care taken by that gentleman in the *post mortem* examination, and the ability he displayed in giving his evidence, made an order on Her Majesty's Treasury that a special fee of five guineas, over and above the ordinary allowance, be granted him as a witness." Referring to the reported evidence of Dr H Stiles, we consider the commendation of the learned judge to be well merited. The medical evidence clearly proved that the deceased had died from suffocation. There was a fracture of two ribs on the right side, with other signs of violent compression of the chest, rendering it probable that one or other of the prisoners had caused suffocation by sitting or kneeling forcibly on the chest of the deceased. There was evidence of heart-disease, which might have accelerated death. There was one remarkable circumstance in the case which tended to indicate sudden death from violence. When the dead body was found, the deceased held in his right hand a door-key, tightly clenched. It was shown that the female prisoner, who was housekeeper to deceased, had gone off with this key, and the deceased, in going after her in order to obtain it, was waylaid by the two male prisoners, and met with his death in the manner described. This, with other circumstances, connected the woman with the act. The prisoners were convicted of murder; but the jury found that their intention was to commit robbery, and that they had not contemplated the death of the deceased. (*British Medical Journal*, 1880.)