gastrectomy, as long as seven years may elapse before development of megaloblastic anaemia. It took 10 years before evidence of frank vitamin-B₁₂ deficiency occurred in our patient, in whom some secretion of intrinsic factor was almost certainly present immediately after the operation. The time interval is of the same order as in the two cases described by Rowlands and Simpson (1932), the symptoms in these cases developing 6 and 15 years after the operation.

Summary

A case is described of subacute combined degeneration of the cord after partial gastrectomy for gastric ulceration in a man aged 32. Evidence is presented to show that the vitamin-B₁₂ deficiency was consequent upon impaired secretion of intrinsic factor owing to the presence of chronic atrophic gastritis. Attention is drawn to the higher incidence of atrophic gastritis and malabsorption of vitamin B₁₂ after partial gastrectomy for gastric ulceration as compared with that following this operation for duodenal ulceration.

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"A further example of prolonged research which in this year has progressed substantially towards a successful solution is provided by the Audible Playing Ball. All such balls so far developed have been based upon the concept of a hollow ball containing small objects, such as dried peas, which rattled as the ball was bounced. All suffered from the disadvantage that, when at rest, the ball ceased to sound. The new ball, at present undergoing intensive tests, consists of a small electronic sounding unit housed in sponge rubber within an outer casing. Powered by a miniature accumulator, rechargeable from a dry battery, this emits a bleeping sound continuously for up to ten hours on one charge. The built-in on/off switch is so designed as not to interfere with true bounce. Simple though such an object may appear to those with sight, it will fill a long-felt want in the lives of blind children and, it is hoped, enable them to add both cricket and football—those twin national pastimes—to the growing list of sports in which they now take part." (*Report of the Executive Council for the year ended March 31, 1959*, Royal National Institute for the Blind.)

PROLAPSED CORD

BY

S. A. SELIGMAN, M.B., F.R.C.S., M.R.C.O.G.

From the Institute of Obstetrics and Gynaecology,
and Queen Charlotte's Maternity Hospital, London

The last 10 years have seen little change in stillbirth and neonatal death rates. Prolapse of the umbilical cord accounts for some of these deaths, and, as it is believed that prompt and adequate treatment would save many more of these potentially normal babies, it seemed worth while to report the results of treatment of 100 consecutive cases seen at Queen Charlotte's Hospital during the period 1951–9. The mode of death in such cases has been investigated by post-mortem examination and by experimental investigations into the role of arterial hypotension.

TABLE I.—Incidence and Results

	Total Cases	Prolapsed Cord	Incidence
Deliveries ..	25,885	100	0.4%
Stillbirths ..	589	19	3.2%
Neonatal deaths ..	461	9	2%

The overall results in this series are given in Table I, there being an uncorrected perinatal mortality of 28% with no maternal deaths.

Aetiology

Clinical

Numerous factors have been invoked as being of importance in the production of cord prolapse, including disproportion, hydramnios, surgical and medical induction of labour, low placental implantation, tumours, anaesthesia, and obstetrical manipulations. No correlation could be demonstrated with any of these conditions in the present series. The ratio of primigravidae to multigravidae was 45 to 55, this being a reversal of the ratio of total admissions over the same period. Only five patients had had more than four children. The factors which appeared to be of importance were as follows:

TABLE II.—Mortality Associated with Presentation

Presentation	Total No. ^a	Stillbirths	Neonatal Deaths
Vertex ..	56	6	4
Breech ..	26	7	3
Oblique or transverse ..	10	4	1
Compound ..	6	2	1
Brow ..	2	0	0

Malpresentation.—The presentation in these cases is shown in Table II. Of the cases in which the vertex presented, the head was not engaged in nine; thus the head was presenting and engaged in only 47% of cases. It is also of interest that of the 26 cases in which the breech presented the legs were flexed in all except two.

Birth Weight.—The baby weighed 5½ lb. (2,495 g.) or less in 32 cases. Fifteen deaths occurred in this group, including all 10 babies weighing 3½ lb. (1,590 g.) or less. In three cases the baby weighed 10 lb. (4,535 g.) or more, and all three died. Of the 65 remaining babies, between 5½ and 10 lb. (2,496 and 4,535 g.), 10 died in the perinatal period.

Multiple Pregnancy.—There were 14 cases of twin pregnancy in the series. During the period under consideration there were 400 twin pregnancies altogether,

prolapse of the cord occurring in 3.5% of these cases. One foetus was dead on admission in a case of locked twins. All the others were live-born, but one subsequently died, its birth-weight being 2 lb. (907 g.). The baby was premature in six cases, and a malpresentation was present at the time of prolapse in eight cases.

Experimental

Until recently no experimental work had been done on this condition, and the views of Heinisch (1955), based on his experimental observations, are of interest. Prolapse of the cord can occur only when the presenting part fails to fit snugly into the surrounding maternal tissues, as when there is a malpresentation, in the absence of the so-called sealing of the lower segment. This obviously cannot be the whole story or the condition would be far more frequent than it is. Heinisch purported to show that the cord remains tense and coiled in the upper part of the uterus, while the pressure within its arteries remains within normal limits. This was done by injecting fluid under measurable pressures into the umbilical vessels. The experiment was performed with the cord immersed in a jar of fluid of appropriate specific gravity representing the uterus and amniotic fluid. Membrane rupture was stimulated by allowing the fluid to escape through an opening in the bottom of the jar, and before raising the umbilical arterial pressure this resulted in prolapse of the cord through the opening. When the pressure was raised to within normal limits the tense cord did not descend on releasing the fluid. If the fall in blood-pressure is the primary reason for cord prolapse, then the foetus is already at risk when this occurs. This concept is obviously an important one in regard to the mechanism of prolapse and its outcome to the foetus, so the experiment was repeated. Heinisch's findings were confirmed, but the experimental conditions were quite different from those *in vivo*. Because of arterial spasm in the severed cords it was quite impossible to run fluid into the arteries until the spasm had been overcome by raising the pressure of the entering fluid to 40 mm. Hg, when a column of fluid was forced along the vessels, the cord coiling and tensing with the passage of the fluid. Increasing the pressure increased these phenomena and produced shortening of the cord, so that the portion between the two fixed ends rose above its former level in a convincing Indian-rope-like manner which prevented experimental prolapse. These changes in the cord may easily be reproduced, without recourse to any special apparatus, by stripping the blood along a recently delivered cord from the placental end towards its clamped end, although here the changes are related to an increase of pressure within the umbilical vein and hence quite unrelated to normal conditions.

In order to stimulate conditions *in vivo* more closely, the spasm in the arteries of the freshly severed cords was abolished by the use of potassium cyanide solution, after which it became possible to run fluid through the umbilical vessels without recourse to pressure. The experiment was repeated, using these prepared cords, and it was found that the changes produced by the original method again occurred, but were more gradual in onset and took place over a greater range of pressure. The cords began to coil, shorten, and tense at approximately 40 mm. Hg, but to a less extent than the untreated cords. The changes increased progressively up to a pressure of 120 mm. Hg, but at 60 mm. were

sufficient to prevent experimental prolapse. From this it would appear that prolapse of the cord is more likely to occur in a foetus which already has a lowered blood-pressure due to some pre-existing condition.

Treatment

The results of treatment are set out in Table III.

Caesarean Section.—One of the two deaths in this group was due to erythroblastosis foetalis; the other

TABLE III.—Treatment and Results

Mode of Delivery	Total Cases	Deaths
Caesarean section	28	2
Forceps	37	8
Breech extraction	21	8
Internal version and breech extraction ..	4	2
Spontaneous	10	8

was associated with multiple congenital anomalies, the foetus dying *in utero* before the onset of labour, but vaginal extraction being impossible.

Forceps Delivery.—Of the eight perinatal deaths in this group, three were due to trauma.

Breech Extraction.—In three cases the foetus was dead on the mother's admission to hospital. In two cases death was traumatic in origin.

Internal Version and Breech Extraction.—This manoeuvre was performed on three occasions in association with twin delivery. In one other case it was thought that the foetus would not survive the 10 minutes necessary to perform caesarean section, death in this case being traumatic.

Spontaneous Delivery.—In two cases intrauterine death had occurred before the onset of labour, and in a further case the cord had prolapsed before admission, by which time the foetus was dead. In one case death occurred in hospital before any active treatment could be instituted; in two others, where an attempt was made to replace the cord, this manoeuvre was associated with immediate cessation of the foetal heart sounds. In one case the foetus was thought to be too small to survive, and in the remaining three cases the cord prolapsed when the cervix was fully dilated, spontaneous delivery following immediately; two of these babies were the survivors in this group.

Post-mortem Findings

Post-mortem examination was carried out in all 28 cases. There were three cases of intrauterine death before the onset of labour—one in association with a severe concealed accidental haemorrhage, and one with multiple congenital anomalies. Death was asphyxial in all three cases. Asphyxia was the commonest cause of death, accounting for 15 further cases in addition to the three above. In three instances the cause of death was given as previability, and there was one death due to erythroblastosis foetalis. The remaining six deaths were due to trauma at delivery.

Discussion

The incidence of prolapse of the umbilical cord varies from 1 in 74 to 1 in 1,018 deliveries (Slate and Randall, 1956). It appears to be decreasing with higher standards of obstetrics and the lowered incidence of contracted pelvis (Ahlgren, 1958). The perinatal mortality in most

of the published series is around 50%, but is lower in some: 36.4% (Myles, 1959) and 35.3% (Brandeberry and Kistner, 1951). Some other series have a lower mortality but are selective in that smaller foetuses have been excluded. The series by Cox (1951) is unique, the stillbirth rate being 11.4% in 35 cases, with no neonatal deaths. He is of the opinion that in urban communities with a highly efficient hospital service the child can be saved in almost every case. From the present series this appears to be an optimistic assessment of the foetal prognosis, there being a number of cases in which the foetus will be dead before prolapse occurs or where death will follow owing to some other factor; as in very small babies. These factors may not show in a selected series of cases, especially where the numbers involved are small; but no doubt many babies die who might be saved by better methods of treatment.

In some cases death is due to trauma associated with delivery, and in others to factors not directly associated with the prolapse. In most cases death is due to anoxia, relative to cessation of circulation in the umbilical vessels which is generally ascribed to pressure, the cord being compressed between the presenting part and the maternal tissues; this, however, seems unlikely in many cases where prolapse is anatomically possible, especially where the lie is not longitudinal, and is obviously not so in many patients examined at the time of prolapse. Rhodes (1956) believes that cessation of circulation in the umbilical cord is due to spasm, quoting experimental evidence by Barcroft (1946). This view seems likely, especially when taken in conjunction with the experiments described above. In addition, Rhodes believes that manipulation and cooling of the cord are added factors producing spasm and thus killing the foetus.

The blood-pressure in the umbilical arteries at term in the human foetus has been found by Barcroft (1946) to be 64–110 mm. Hg. Barcroft (1952) has also shown that there is a gradual rise in arterial pressure as intrauterine development proceeds, comparable figures in the sheep foetus being 38 mm. Hg at 120 days and 56 mm. Hg at 147 days. This in conjunction with the above experimental findings explains why prolapse of the cord occurs more often in the premature than in the mature foetus.

In order to reduce the foetal loss it is important to discover the actual mode of death in these cases, yet this has been neglected in previous series. In the present series no fewer than six of the 28 deaths were due to trauma. Although immediate delivery is indicated in these cases, even more care than usual must be taken, since the foetus is already in a precarious state and degrees of trauma which do not affect the healthy foetus may be sufficient to jeopardize its existence.

It is significant that, excluding the case of intrauterine death and the erythroblastotic foetus, the baby survived in every case submitted to caesarean section. Although some deaths in the remaining cases were due to trauma, the more usual mode of death was asphyxial. It is probable that the handling and manipulation of the cord necessary when performing vaginal delivery in these cases produces spasm in the umbilical vessels of a foetus already at risk, accounting for some of the deaths.

In the 10 cases where the baby weighed 3½ lb. (1,590 g.) or less there were no survivors. Although caesarean section was not performed in this group it seems unlikely that it has a real place in the treatment of cord prolapse of the very small foetus. Myles (1959) did not find

any essential difference in mortality rates between babies weighing more than 5½ lb. (2,495 g.) and those weighing less, but does not state how many were substantially below this limit. All babies weighing 10 lb. (4,535 g.) or more died, but this number is too small to determine whether this is of any significance.

To achieve and improve upon the comparatively low perinatal mortality rate of this series it is necessary to have adequate facilities, so that caesarean section or any other treatment can be performed within 15 minutes of the diagnosis of prolapse of the cord. Early diagnosis by routine vaginal examination at the time of membrane-rupture will still further reduce the time-lag between occurrence and the institution of treatment.

Summary

One hundred cases of prolapse of the umbilical cord have been analysed and the aetiological factors and methods of treatment assessed.

The important predisposing factors are malpresentation, prematurity, twin pregnancy, and multiparity.

Caesarean section is superior to all other methods of treatment.

The recent improvement in the results of treatment in these cases is confirmed, but the claim that the child can be saved in almost every case is disputed.

The concept of lowered foetal blood-pressure as an aetiological factor in the occurrence of prolapse of the umbilical cord has been confirmed, and is advanced as the reason for the relative frequency of this condition in the premature foetus.

I thank the Medical Committee of Queen Charlotte's Hospital for permission to publish these results, and Professor J. H. M. Pinkerton for encouragement and help with this paper. I also acknowledge the part played by the various members of the medical and nursing staffs in the care of the patients, and by the pathology department in providing the post-mortem examination findings.

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"One of the most pathetic forms in which sickle cell disease may present is the tearful mother, who comes with a sick child, whom she describes as suffering from a disease similar to one which has already stolen many of her children. If one may digress a bit, I should like to express my belief that the widespread Ibo belief in Ogbanje—that is, the concept of children who come to the world again and again only to die, breaking their parents' hearts with a sadistic regularity—probably arises from the widespread existence of sickle cell anaemia, and suggests that the disease must have been with our ancestors for many generations. There is, of course, another disease which could present such a picture, and that is Rh haemolytic disease of the new-born, which for some unknown reason and in total defiance of the laws of immunity is fortunately rare in Nigeria." (Dr. C. Nwokolo, *West African Medical Journal*, October, 1960.)