poisoning did not appear as soon as the treated wheat was consumed: there was a latent period while the body concentration of mercury increased. In many cases the farmers had tested the grain by feeding chickens and other farm animals with the seeds provided. When they observed no signs of poisoning they felt safe and used the grain themselves. The first cases of poisoning were reported in the last week of December 1971, and no patients were admitted to hospital after March. All ages were affected, but the largest group was those aged 1-9 years; there were 459 hospital deaths.

In this outbreak the severity of the symptoms and signs was dose-dependent; the onset of symptoms was gradual, with a latent period between 16 and 38 days after the last intake of the contaminated bread. Neurological signs and symptoms were the most evident. These included muscle weakness, paraesthesiae, ataxia, dysarthria, hyperreflexia, intention tremor, and muscle pain. Examination occasionally showed reduced appreciation of pin prick peripherally, defective position sensation, astereognosis, and abnormal two-point discrimination. Although the clinical picture in some patients resembled peripheral neuropathy,[7] tests of motor and sensory conduction velocity, sensory threshold and sensory latency, the H reflex of the tibial nerve, and myoneural transmission failed to confirm such a disorder. The site of damage in methylmercury poisoning leading to the picture of neuropathy seems to be either in the lower brain stem or at some point in the neuromuscular linkage.[8 9]

Visual disturbances developed in half the cases. Psychological symptoms included headache, sleep disturbances, dizziness, and irritability. Oral, cardiovascular, genitourinary, visceral, or cutaneous manifestations were unusual.[10] Infants showed variable degrees of nervous system damage similar to the congenital Minamata disease described by the Japanese authors—namely, cerebral palsy, blindness, deafness, hyperreflexia, and mental retardation.[11]

In this epidemic patients only mildly affected recovered completely, while those moderately disabled improved gradually over several months. The most severely poisoned died early in the outbreak, irrespective of the treatment they received. Some surviving bed-ridden patients had recovered substantially when examined six months later. In a small-scale follow-up study of 13 severely affected patients, mainly children, two years after the epidemic, one had recovered completely, four were greatly improved, and of the eight who were blind five had regained some sight. Patients from families who had been victims of the 1960 epidemic and who were re-examined in 1976 had mostly recovered completely, but those who had been severely affected still complained of transient dizziness and occasional numbness of their palms and soles, without signs of sensory damage.

Japanese reports on organic mercury poisoning[13] had suggested a progressive downhill course and had concluded that the condition was irreversible. Experience in Iraq has shown a different pattern. In the Minamata cases the poisoning from contaminated fish was not discovered for several years, by which time the total body burden of mercury was dangerously high. In Iraq the epidemic was more acute, the diagnosis was immediate, and once the intake had been stopped the concentration of mercury fell with its average half-life of 65 days.

A WHO/FAO meeting[14] on the use of mercury and alternative compounds as seed dressing, held in Geneva in 1974, made two recommendations: firstly, that the use of mercurial fungicides should never be permitted for treating seeds to be exported for production of food; and, secondly, that dressed cereal grains should be immediately and permanently distinguishable from undressed seed by colouring and the addition of a permanent odour or taste. Finally, doctors everywhere need to be aware of the symptoms of mercurial poisoning and to be alert to the possibility of an epidemic whenever there are outbreaks of an unexplained illness, especially in rural areas.

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Intra-articular steroids

Intra-articular injections of corticosteroids are a valuable adjunct to the treatment of arthritis both in man[1-3] and in animals.[4] While their value in rheumatoid arthritis is undoubted, their use in osteoarthrosis is more controversial. Soon after cortisone was introduced its use was advocated in degenerative joint disease, but several later trials showed that it had little more value than intra-articular saline.[5-8] Enthusiasm for the treatment was further tempered by reports from Leeds[9-10] of steroid arthropathy after multiple injections of steroids in both rheumatoid arthritis and osteoarthrosis. This iatrogenic damage has since been confirmed in other clinical studies[11-17] and reproduced in studies on animals.[18-23]

The Charcot-like arthropathy produced by corticosteroids seems to be a local phenomenon, though the drugs were given systemically in most of the animal work. Hydrocortisone inhibits the incorporation of radioactive sulphur into cartilage in rats.[18] Hydrocortisone decreases the size of chondrocytes in mice,[21] while Morison *et al*[19] found that glycosaminoglycan synthesis by cartilage was reduced in specimens removed from rabbits treated with cortisone. Intra-articular injection of steroids in rabbits led to exaggerated degenerative joint changes, with loss of cartilage, subchondral cysts, and fissuring; the extent of the lesions depended on the number of injections.[21 22] Primate joints may, however, respond differently: steroid-induced changes may be transitory,[21] and a detailed study of 10 monkeys who received intra-articular methyl prednisolone showed no joint disorganisation.[25]

A recent clinical investigation[26] of patients treated with repeated intra-articular corticosteroid injections for not less than four years examined the effects on a total of 65 knees. Thirty-five were affected by rheumatoid arthritis and 30 by osteoarthrosis. The least number of injections was 15 in four years, the most 167 in 12 years (some way short of the 3000 injections[16] inflicted on one unfortunate American patient with disastrous results). Only two joints showed gross radiological deterioration over this period, and Balch *et al*[26] suggest that

intra-articular injections of steroids have an important place in the overall management of patients suffering from both types of chronic arthritis. These injections should not, however, be given in a predetermined course of treatment; and the interval between injections should not be less than four weeks. Before any repeat injection is given, the doctor should carefully appraise the effect achieved and the state of the joint.

Clinicians following this balanced advice will do much good to their reputation and little harm to the patient. The case for local corticosteroid injections in rheumatoid arthritis is well substantiated. In osteoarthrosis a more rational approach may be to infiltrate tender areas around the joint intralesionally, unless there is an obvious reactive synovitis secondary to the degenerative condition, when intra-articular therapy may help.

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Umbilical cord prolapse

Prolapse of the umbilical cord occurs in about four deliveries in every thousand.[1] Though it is an infrequent complication, the high associated fetal mortality rate gives it unusual importance.

The primary cause of cord prolapse is incomplete filling of the pelvic cavity by the presenting part of the fetus at the time the membranes rupture. Non-engagement of the presenting part, commonly associated with an abnormal presentation, was a factor in 83% of 424 cases reported by Cushner.[1] The risk of cord prolapse is greatest when the fetal lie is transverse or there is a compound presentation. Numerically, however, breech presentation is the most common single cause of these accidents, accounting for 40-50% of all cases. The risk is three times greater when the fetal legs are flexed than when they are extended.[2 3] Other recognised factors include prematurity, multiple pregnancy, long cord,[4 5] polyhydramnios, and fetal hypotension.[6] There is no uniform agreement on whether or not amniotomy is relevant. Despite the increased frequency of induction in recent years the incidence of prolapsed cord has remained fairly constant.[2] In one series prolapse occurred twice during 1000 labours induced by amniotomy but five times during 1000 matched control spontaneous labours[7]; in contrast in another study there were four episodes of prolapsed cord after 1000 amniotomies but only two in 1000 matched control patients with spontaneous labours.[8] Provided that the usual precautions are observed the frequency of prolapse seems unlikely to be more than marginally increased by amniotomy.

Immediate delivery of the fetus remains the cornerstone of treatment. Attempts to replace the prolapsed cord have no place in modern obstetrics. Rapid expert assessment is necessary to determine the best course of action. If the fetal heart beat is present and the fetus is sufficiently mature to be viable then immediate delivery is mandatory. Provided the cervix is fully dilated and vaginal delivery can be achieved without delay—and without undue trauma—then this may be the preferable course of action. If not, then the fetus should be delivered by immediate caesarean section. Absence of pulsation in the prolapsed umbilical cord is an unreliable sign of fetal death. There are many recorded instances[5 9 10] in which cord pulsations were absent but the fetal heart was audible, and in which the infants survived.

Procedures that have been used to relieve pressure on the umbilical cord as a first-aid measure pending delivery include manual elevation of the presenting part, and placing the patient in the knee-chest or Trendelenburg position. Perinatal mortality seems lower when some attempt has been made to relieve pressure on the cord.[1]

Fetal prognosis has improved in recent years with the more liberal use of caesarean section in place of heroic vaginal manipulations to achieve delivery through an incompletely dilated cervix. Nevertheless, the perinatal mortality rate is still around 20-30%, and cord prolapse results in the loss of 600 infants annually in England and Wales. Surviving infants may also suffer late sequelae as a result of cerebral hypoxia, prematurity, and birth trauma. The incidence of these is difficult to assess, there having been few long-term follow-up studies. Cushner[1] traced 63 children who had survived cord prolapse 5-25 years previously: 60 were apparently normal.

Though we can do little to prevent prolapse of the umbilical cord, emphasis must be placed on earlier diagnosis of the condition if we are to improve the appalling perinatal mortality rate. Ideally continuous fetal heart rate monitoring should be routine from the onset of labour, whether or not the membranes are ruptured. Variable decelerations and other fetal heart rate patterns have been described[11 12] which are suggestive of umbilical cord compression. Recognition of these patterns may give early warning of cord complications in 90% of cases.[11] Unfortunately few hospitals have sufficient equipment to monitor the fetal heart rate continuously in all labouring women, so we need to identify those patients at particular risk. The incidence of prolapsed cord may be as high as 11% in compound presentations[9] and over 6% when the breech presents,[2] and these malpresentations at least should therefore be regarded as indications for continuous fetal heart rate monitoring.

Cord prolapse occurs before hospital admission in up to 25% of cases.[1 3 13] Patients should therefore be encouraged to come into hospital at the first sign of labour rather than waiting until regular contractions are occurring.