food introduced is a lower-lactose, freely available "normal" infant-feeding formula such as Ostermilk Complete.

So much for the theory. What is an appropriate practical regimen? One is given which individual doctors might wish to modify. Many parents find it reassuring to follow a didactic regimen.

Firstly, home or hospital? The presence of any of the following signs urges caution, and hospital admission should be considered: a deteriorating level of consciousness, a poor urine output—almost impossible to measure in the home, and torrential diarrhoea in the young infant may be mistaken for urine—hypothermia (rectal temperature $<35.5^{\circ}\text{C}$), jaundice or a palpable spleen (which raises the possibility of septicaemia), purpura, evidence that the child has recently lost 10% of his body weight, or clinical signs of severe dehydration. Gastroenteritis in young infants (below 6 months) is generally more troublesome and leads to more severe metabolic disturbance than in older children. They need particular attention and may be in danger in an inadequate home.

Treatment should be divided into five periods. In the first, offer oral rehydration fluid only, every three hours or more frequently if the child is thirsty. Set no upper limit on consumption, but the child's condition should be carefully reassessed if he or she consumes less than 200 ml per kg body weight—that is, one sachet of electrolyte powder dissolved in 200 ml for each kg body weight in 24 hours.

In the second period give quarter-strength formula made up with rehydration fluid (such as, for a four-ounce feed: one level scoop (3.5–4.5 g, depending on brand) of milk powder plus 4 oz (113 g) rehydration fluid), again aiming at a total intake of 200 ml/kg/24 hours, offered in three-hourly feeds. If the child is still thirsty after a quarter-strength feed more of the rehydration fluid is offered.

During period three give half-strength formula, made up with rehydration fluid. A more "normal" intake of 150 ml/kg/24 hours is a reasonable target to aim for.

In periods four and five progress to full-strength formula, made up now with water.

Thereafter reintroduce solid foods—if any. Check that normal weight gain has been re-established.¹⁷ If the child is bottle-fed, check that the methods used for making up the feeds are microbiologically safe.

Breast feeding should continue during the episode and in this case it is sufficient to offer a complement of rehydration fluid three-hourly. Each period above is usually 24 hours but may be reduced to 12 hours for mild episodes. If diarrhoea persists then the regrading process may be restarted from period one or put back by two periods. If after two attempts at regrading at the normal rate (each period 24 hours) diarrhoea continues or if any of the worrying signs described above occur then a more detailed assessment is necessary, probably in hospital. Recurrent episodes, especially when accompanied by poor weight gain or abdominal distension, require further investigation: in one series of 12 infants with coeliac disease three had presented with acute gastroenteritis.¹⁸

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Policeman's heel

Pain around the heel may result from disorders of the Achilles tendon or of the bursa deep to it, from infections, from Paget's disease of the calcaneum—or from the condition sometimes known as "policeman's heel," plantar fasciitis, calcaneal spur, or just painful heel syndrome. This is said to be a common disorder, but there are no figures for its prevalence; plantar fasciitis was, however, the most frequent foot problem in a group of hotel workers.¹ In that study of 328 patients and another² of 115 patients obesity, though undefined, was the most common associated condition, being found in one-third of those affected. As the name policeman's heel implies, the disorder is often seen in people who walk and stand a good deal; waiters and maids figured prominently in the hotel study.¹ Presumably the condition is different from the transient heel pain that may follow an attack of influenza or trauma in athletes, because in most series the patients have been over 40 years old. Though usually unilateral it may affect both heels and is then more likely to be part of some generalised disorder² such as rheumatoid arthritis. A rheumatoid nodule beneath the calcaneum is an occasional cause of heel pain.

Apart from these generalisations publications on plantar fasciitis give surprisingly conflicting views, even about presentation. Some hold that the pain is usually central, others that it is usually over the medial tubercle of the calcaneum. This disagreement leads to others. For example, if the pain were central this would support the view that calcaneal spurs (whether they project forwards or downwards¹) play a part in the disorder. Most rheumatologists now think that spurs are irrelevant unless fluffy on the x-ray film and hence part of an enthesopathy (ankylosing spondylitis or Reiter's syndrome). Spurs are usually bilateral, but pain is unilateral. Whether or not there is a subcalcaneal bursa has also been argued.^{1,3} Anatomy textbooks, however, do not describe one; and Professor E G L Bywaters, who has made a particular study of these structures,⁴ confirms that no such bursa exists (personal communication, 1981).

The treatment of this slightly mysterious condition is also not wholly agreed. In a major reference work⁵ on one page it is said to respond to injections of corticosteroids, while on another injections are described as painful and difficult. Some recommend phenylbutazone or other anti-inflammatory drugs; some heel pads or heel cups; some surgery, either by division of part of the plantar fascia or by removal of the medial tubercle of the calcaneum. An exotic if somewhat bizarre approach from Egypt⁶ and later China⁷ is to drill holes in the calcaneum to relieve pressure; this work has not been confirmed or refuted. The principal impression from experience and published reports is that most patients get better with rest and protection from direct pressure on the affected heel. Only in resistant cases should any other approaches be considered—and reconsidered.

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Matricide

Murder within the family accounts for almost half of all homicides in Britain. Mental abnormality is more likely in those who murder a member of their family than in those who murder a stranger.^{1 2} Matricide, the killing of a mother by her child, is one of the rarest forms of family murder³ and is almost exclusively a crime of sons.

The matricidal theme has its roots in Greek mythology and is charged with high emotion. Psychoanalysts⁴⁻⁶ have always been receptive to the matricidal fantasies of their patients and have theorised at length on the mental mechanisms behind such thoughts. Forty years ago Wertham,⁷ drawing analogies between his patients and the matricidal figure of Greek mythology, coined the expression "Orestes complex." This term described a sexually immature but homosexually orientated son, trapped in a dependent but hostile relationship with a possessive mother. Escalating tension in such vulnerable sons could erupt in the commission of a murderous act, which Wertham called a catathymic crisis.⁸

Two small studies of matricide in Canada⁹ and England¹⁰ both found a substantial association with schizophrenia, and a Scottish study¹¹ of homicide, which included just four matricides, concluded that "matricide is the schizophrenic crime." Murderers with electroencephalographic abnormalities of doubtful significance^{12 13} and with hypoglycaemia¹⁴ have also been reported.

The problem of collecting a sufficient number of cases is formidable, and a recent report by Green¹⁵ on 58 inmates of Broadmoor Hospital who had committed matricide is by far the largest study so far. Its findings require cautious interpretation, since the cohort is drawn from a population that has been subjected to the complex selection process leading to admission

to Broadmoor. By definition all Broadmoor patients have a mental disorder, but Green considers the Broadmoor matricidal patients to be representative of matricides in general. He may be right, but only a comprehensive and nationally conducted study would satisfy this crucial problem of sampling. Furthermore, the impact of the diagnostic findings of schizophrenia (74%), depressive illnesses (16%), and personality disorder (10%) is diminished by the lack of any comparison with a control group of other Broadmoor murderers. Such a comparison would have regularised the vagaries of diagnosis sometimes found in forensic psychiatry.

Not surprisingly, nearly all the sons were living at home with their mothers, and the bedroom or kitchen was the usual locus for the murderous attack. The mean ages of the victims and their assailants were 63 and 31 years respectively. Most of the women died of stabbings and batterings, with over two-thirds showing extreme degrees of violence. Some of the findings are a vindication for psychodynamic theory. About three-quarters of the households had an absent father figure and over half the offenders had had no significant sexual experience. In all but one case there was no attempt made to conceal the crime or escape detection, and a sexual element was identified in over a third of matricides. In two-thirds of the cases motivation centred on paranoid or altruistic ideation, but 11 cases could be explained by nothing other than sudden rage.

The apparent high level of psychiatric morbidity, particularly of schizophrenia, in matricidal killers cannot be ignored but must be put in context. Firstly, even if all matricides in England were committed by schizophrenics there would be only a dozen or so such cases each year. Moreover, thousands of schizophrenic sons live in peace with their mothers, neither assaulting nor killing them; and the contribution made by schizophrenics to the homicide rate in general is insignificant.^{16 17} Secondly, family murder does not occur in a vacuum. It is a complex interaction of individual characteristics, precipitation by the victim, and environmental chance.¹⁸ Victims do not give interviews, and the inevitable medicolegal preoccupation with the psychopathology of the offender has hampered psychiatric knowledge of these often strange crimes.

An association does appear to exist between matricide and schizophrenia, but how far the relation is causative remains unknown; simply allocating the diagnosis of schizophrenia to an individual is an incomplete and unsatisfactory explanation for his crime. The relation of schizophrenia to matricide, as distinct from other forms of family murder, might owe more to opportunity than psychodynamics. The socially disabling effects of the disease reduce the likelihood of marriage and prolong dependency on parents.

Prospective anticipation is a giant step from retrospective description, but how might doctors reasonably be expected to recognise potential danger? Many cases of matricide are neither predictable nor preventable, but the possibility should be borne in mind in treating schizophrenic sons who lead a socially isolated existence with their mothers and have little other family support. Sudden changes in mental state, delusional ideas concerning the mother, and threatened or actual violence call for swift intervention. Mothers who admit to being frightened of their sons should not be ignored.

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