predictable. It is usually described as being of a steady dull aching character and of mild to moderate intensity. It is not directly related to exertion but tends to occur towards the end of the day and often seems to come on when a patient has exerted himself rather more than he feels he ought to have done. It is frequently affected by movement of the body, and many patients have areas of localized tenderness over the chest.[6]

The mechanism of this left chest pain remains uncertain. It is unlikely to be ischaemic in origin, and has been likened to the pain of Da Costa's syndrome. It appears to be correlated with responsiveness of the sympathetic nervous system,[7] and Professor Lovell and Dr. A. Verghese show in their paper published this week in the *B.M.J.* at page 327 that it is also linked with neuroticism. They find in addition that patients with left chest pain tend to be long and narrow in body build and to be less fat than others. The authors indicate that these features have been shown to be linked with constitutional neuroticism and suggest that it might be worth trying to identify early in convalescence patients likely to develop these pains. Some of the disabling features of the pain might then be prevented. Once anxiety about it has been met with adequate reassurance and explanation the pain usually disappears or at least ceases to worry the patient.

Left chest pain has also been compared with the "frozen shoulder" and "shoulder-hand" syndromes, usually attributed to reflex muscular spasm and disturbances of sympathetic innervation. Disuse also is probably important, and these conditions are seen less frequently now that early mobilization after infarction is encouraged. They had previously been described as occurring in 10–20% of patients. Persistent pain may develop in one or both shoulders, usually with tenderness and limitation of movement.[8] Sometimes there is wasting of the shoulder muscles. In a minority of patients the hands are also affected, with stiffness, pain, swelling, and discoloration.[9] Dupuytren's contracture and other trophic changes are described. The pain can persist for many months, and may recur. Physiotherapy often helps, and infiltration with procaine or hydrocortisone may also do so.

Any pain in the chest or arm which occurs after the pain of myocardial infarction is commonly taken by the patient to indicate serious cardiac disease. The doctor too may misinterpret its significance or unintentionally transmit anxiety to the patient. Rehabilitation is then often held up without need, and A. J. Goble and his colleagues[10] have given a dramatic description of the incapacity which may ensue. It is the doctor's task to prevent this.

[1] Verghese, A., and Lovell, R. R. H., *Brit. med. J.*, 1966, **2**, 1102.
[2] Solem, J. H., Helle, I., and Jörgensen, W., *Acta med. scand.*, 1963, **174**, 315
[3] Palmer, J. H., *Quart. J. Med.*, 1937, **6**, 49.
[4] Dressler, W., *Arch intern. Med.*, 1959, **103**, 28.
[5] Edwards, W. L. J., *Amer. Heart J.*, 1955, **49**, 713.
[6] Prinzmetal, M., and Massumi, R. A., *J. Amer. med. Ass.*, 1955, **159**, 177.
[7] Nestel, P. J., Verghese, A., and Lovell, R. R. H., *Amer. Heart J.*, 1967, **73**, 227.
[8] Ernstene, A. C., and Kinell, J., *Arch. intern. Med.*, 1940, **66**, 800.
[9] Johnson, A. C., *Ann. intern. Med.*, 1943, **19**, 433.
[10] Goble, A. J., Adey, G. M., and Bullen, J. F., *Med. J. Aust.*, 1963, **2**, 975.

Risky Grandmothers in Rh Disease

Red blood cells from a foetus can pass through the placenta into the mother's blood stream, and most of this leak occurs during labour. It is this leak that sensitizes an Rh-negative mother who has an Rh-positive foetus, so that Rh-antibodies are produced by the mother's immunological system. When the mother becomes pregnant again she already has some Rh antibodies in the blood stream, and they can pass the placenta and affect the red cells of an Rh-positive foetus.

But often this sensitization does not occur. Figures vary from 1 in 19 to 1 in 23 of expected pregnancies, so that the incidence of children with erythroblastosis is much less than theoretically expected.[1] One of the attempts to explain the deficit has involved the role of the grandmother of the affected child—that is, the mother of the Rh-negative woman who has an Rh-positive child. It is sometimes not realized that maternal red blood cells can pass the placenta into the circulation of the foetus. R. R. Race and Ruth Sanger show from published reports that this can happen in 1 in 6 pregnancies or even more often.[1] Two theories about the effect of this maternal invasion have been proposed. One theory suggests that the passage of maternal cells may induce immunological tolerance in the foetus; the other theory is directly opposed and suggests that the passage of maternal cells increases the liability of the child to respond to Rh stimulation in later life.

Jane F. Taylor,[2] of Columbus, Ohio, has set out to investigate which of these theories is likely to be correct. She has done this by examining a series of families each consisting of a grandmother, her Rh-negative daughter, and the daughter's children. When ABO blood groups of mother and child are incompatible, foetal Rh-positive cells that cross the placenta may be destroyed before they are able to induce sensitization in the Rh-negative mother; consequently such families where this might occur were not included in the trial. She also eliminated any mothers who had been transfused, and did not include any non-white families. With these limitations, 157 out of the original 236 families were included in her trial and they were derived from two groups: families in which the mother had had a child affected by erythroblastosis in any pregnancy, and families referred for routine prenatal testing in which the mother had had at least three Rh-positive children none of whom had developed erythroblastosis. The grandmothers, the mothers, and the children all had their ABO and Rh groups determined, and erythroblastosis was diagnosed on clinical evidence and the occurrence of a positive direct antiglobulin test with the child's red cells and the presence of Rh antibodies in the mother's serum.

The results showed a statistically significant excess of Rh-positive grandmothers in the families who had an erythroblastotic child. In families with Rh-positive grandmothers 79% of the children had erythroblastosis, whereas in the families with Rh-negative grandmothers 60% of children were affected. By an ingenious calculation Taylor showed that the effect of the Rh-positive grandmother was equivalent

[1] Race, R. R., and Sanger, R., *Blood Groups in Man*, 1962, 4th ed., p. 387. Oxford.
[2] Taylor, J. F., *New Engl J. Med.*, 1967, **276**, 547.

to one Rh-positive pregnancy. Consequently Taylor firmly supports the view that the passage of maternal cells from an Rh-positive mother to her Rh-negative female foetus means that when the daughter bears children in her turn she will be more, not less, liable to have an affected child if her husband is Rh-positive. And the risk of erythroblastosis occurring in the daughter's children is about equivalent to one Rh-positive pregnancy.

Taylor also cites data which suggest that for the majority of Rh-negative women who have Rh-positive husbands the first child to be affected is most likely to be that of the third pregnancy. But the data from the examination of grandmothers suggest that if the woman has an Rh-positive grandmother the first affected child may well come from the second pregnancy. However, mothers in these circumstances should not receive too gloomy a prognosis from their medical advisers, because the rule still applies that only about 1 in 20 pregnancies that might result in a child affected by erythroblastosis actually does so.

Disorders of Defaecation in Children

Disorders of bowel habit in children are notoriously difficult to manage. To the mother failure in toilet training is considered to be a reflection upon herself, and she may make strenuous efforts to conceal the evidence of faulty control of the bowel in her child from relatives, friends, and doctors.

In considering the problem of abnormal bowel habits in children a clear distinction must be made between organic and " functional " disease. Apart from anatomical deformities such as anal stenosis, Hirschsprung's disease is the most important organic condition which must be considered. Symptoms of this disease are invariably present from the neonatal period, with prolonged periods without a bowel motion and accompanied by extreme gaseous distension of the abdomen or actual intestinal obstruction. The infrequent stools are small and " rabbity " in type, but occasionally an infant may present with severe diarrhoea. Thus any disorder of bowel habit with infrequent stools which comes on after a period of months or years of normal defaecation is almost certainly not due to Hirschsprung's disease.

In infancy, however, the passage of hard stools is not uncommon and may cause an anal fissure, with a resulting fear of defaecation. This may ultimately lead to voluntary retention of the faeces, which in turn become hard and painful to pass. A vicious circle may then be set up, which may last for many weeks or months. In the older child, on the other hand, faecal soiling is more difficult to evaluate. A detailed history will usually show whether the soiling is due to leakage of soft faecal material through the anal sphincter, or whether it is due to an involuntary or voluntary act of defaecation which is inappropriate in time and place. Constant faecal soiling is due to the softening of impacted faeces in the rectum and anal canal or to mucus and faeces leaking round a firm faecal mass. There is usually a history of a normal bowel habit until a period of constipation which is followed by faecal soiling: the cycle may be ended by the passage of a very large motion often described in revealing terms,[1] after which the sequence will be repeated. The circumstances which set off this dismal story can often be easily identified. A period of mild dehydration with constipation may accompany a febrile illness, a spell of hot weather, a long car journey, or a summer holiday—or an admission to hospital for a minor operation, or starting school, may be the initial cause. There is obvious perianal soiling, the anal sphincter is lax, and firm faeces fill the anal canal. There is no question of voluntary defaecation in such cases; the child may spend long periods attempting unsuccessfully to defaecate. Management is not easy in long-established cases; enemas and wash-outs should be avoided unless other measures fail, and most children will respond to the use of liquid paraffin initially followed by carefully regulated doses of proprietary senna preparations. A full explanation to both parent and child is essential. Nevertheless, relapse is common, and it is difficult to avoid the conclusion that in many instances there is an underlying emotional disorder in child or parents, or both, which prevents the re-establishment of a normal bowel habit.

Margit Bellman[2] in Stockholm has recently conducted a survey into " encopresis." Most of the children in this group had either never gained normal bowel control (" primary encopresis "), or had broken down after a period of normal control (" secondary encopresis "). Constipation did not play an important part in the troubles of these children, who seem to constitute a separate group from those with chronic constipation and faecal soiling. Dr. Bellman's findings are of interest. The condition was commoner in boys, and of the whole series encopresis was mentioned in the school record card in only 11%. The mothers of affected children tended to be over-anxious, and coercive pot-training was common. " Accidents " were punished severely, and yet at other times the mother was indulgent. The children were lacking in self-assertion and rarely had the normal period of defiance at the age of 2–3 years. Happily the majority had lost their bowel symptoms by the age of 10 years. Thus the conclusions of this interesting study seem to justify the introduction in 1925 of the distinctive term " enkopresis " by C. Pototsky.[3]

Musculo-skeletal Disorders after Renal Transplantation

A variety of skeletal and muscular disorders have been described after renal transplantation. They fall into three main categories: complication of high doses of corticosteroids, consequences of inadequate renal function, and changes in parathyroid function.

It is often necessary to treat patients with transplants with very high doses of corticosteroids, rising to as much as 200 mg. of prednisone a day during rejection crises. In addition to the usual features of Cushing's syndrome, demineralization of the bones may result in renal stones and pathological fractures. If the high corticosteroid dosage needs continuing it can stunt the growth of children. Arthropathy resembling gout can follow renal transplantation.[1] However, chemical analysis of the periarticular deposits shows high concentrations of calcium and phosphorus, with negligible amounts of urate. The joint surfaces themselves are not involved, and the lesions are probably due to a crystal-induced arthropathy which has been called pseudogout.[2] It has been reported in patients on chronic intermittent dialysis[3] and is probably due to insufficient dialysis, while in patients with renal transplants it is almost certainly due to poor function in the transplant.

Secondary parathyroid hyperplasia is a common sequel to

[1] Gairdner, D., *Brit. med. J.*, 1965, 2, 91.
[2] Bellman, M., *Acta paediat. scand.*, 1966, Suppl. No. 170.
[3] Pototsky, C., *Die Enkopresis*; in Schwarz, O., *Psychogenese und Psychotherapie körperlicher Symptome*, 1925. Wien.