weekly regimen in rapid inactivators is due to the shorter duration of exposure to isoniazid than in slow inactivators. The duration of exposure to isoniazid might be increased in rapid inactivators by increasing the dose of isoniazid. Little increase in dosage is possible, however, because of the risk of acute toxicity. The addition of P.A.S. slightly enhances isoniazid blood levels but does not appreciably improve the efficacy of the once-weekly regimen. Slow-release preparations of isoniazid might prolong absorption, or alternatively derivatives of isoniazid might prolong high concentrations in the blood. These possibilities merit investigation.

P. Stradling and G. W. Poole have treated outpatients in London with streptomycin, isoniazid, and P.A.S. daily for the first three months and thereafter continued therapy with the fully supervised administration of streptomycin 1 g. and isoniazid 600 mg. twice-weekly. Disease was satisfactorily controlled in all patients, and the authors considered that the results were better than were likely to have followed the self-administration of drugs on a conventional regimen.

Twice-weekly supervised therapy has been compared with daily self-administered therapy in two recent controlled studies. No important advantage for supervised therapy was detected in either. The International Union against Tuberculosis conducted a study in seven developing countries. Daily supervised streptomycin, isoniazid, and thiactazone were given for the first month followed by twice-weekly streptomycin and isoniazid. This was compared with the same initial treatment followed by daily self-administered isoniazid and thiactazone. The results of the two regimens were similarly good in respect of therapeutic efficacy, toxicity, and patient cooperation.

In a study in Czechoslovakia initial daily supervised therapy with streptomycin, isoniazid, and P.A.S. for three months was followed by either twice-weekly streptomycin and isoniazid or self-administered daily isoniazid and P.A.S. Equally good results occurred in both groups.

In well-organized treatment services it is possible to monitor self-administered regimens closely by means of urine tests, table counting, surprise home visits, and frequent interrogation of patients. In these circumstances they may have no advantage over supervised intermittent regimens. Where self-administered treatment gives poor results, an intermittent supervised regimen may improve them.

Recurrent Abdominal Pain in Children

Many medical parents, most family doctors, and all paediatricians have to take responsibility for the diagnosis and management of children with recurrent abdominal pain. J. Apley in a survey in Bristol schools found that 1 in 7 children complained of recurrent headaches, 1 in 9 of recurrent abdominal pain, and 1 in 25 of recurrent limb pains. He sought a common denominator for these pains and found that there was a strong emotional element in most of them, often with a family history of the same complaint. The problem of the child with recurrent abdominal pain was admirably summarized in his classic book.

Now R. T. Stone and G. J. Barbero in Philadelphia have studied 102 children aged 2 to 14 years with recurrent abdominal pain. The pain was central in 78, vaguely localized in a further 10, and on the right or left side in the remainder. It was described as cramp-like in 7%, dull aching in 18%, and acute spasmic in the others. Associated symptoms included mainly headache, sickness, dizziness, poor appetite, and vomiting. The symptoms in two-thirds were related to stress. Previous diagnoses included appendicitis in 17%, duodenal ulcer in 14%, an emotional problem in 13%, and fibrocystic disease of the pancreas, allergy, regional ileitis, and constipation in others. Stone and Barbero performed a proctoscopy in 90 children and found a mixture of signs—rectal dilatation, hyperaemia, lymphoid hyperplasia, oedema, friability of the mucous membranes, and pellet stools. All laboratory investigations were negative. They thought that the symptoms were part of the irritable colon syndrome as described by M. Davidson and R. Wasserman.

As there are no pathognomonic signs of the condition, the difficulty facing the family doctor and paediatrician is that of satisfying himself of the correctness of the diagnosis. It is always wrong to decide that a symptom is entirely emotional merely because one cannot detect organic disease. The diagnosis must be made on the basis of positive evidence of emotional disturbance and the exclusion of organic disease—not only at the time of the initial diagnosis but on follow-up examination. Apley found evidence of organic disease in about 6% of his cases. Microscopy and culture of a clean midstream specimen of urine are essential parts of the investigation. A good nonspecific test is the erythrocyte sedimentation rate. An abnormal figure points immediately to organic disease, but a normal figure does not exclude it. Sometimes a paediatrician will want to see an intravenous pyelogram in order to eliminate a hydronephrosis. He may ask for occult blood tests as a guide to the diagnosis of a peptic ulcer and rarely a barium meal examination for the same condition. In countries abroad it is wise to eliminate round-worm infestation by examining the stools and tuberculosis by a tuberculin test. It is psychologically unsound to carry out a series of investigations, week after week or month after month; it is better to satisfy oneself once and for all that there is no discoverable organic disease. The family doctor may feel that none of the tests mentioned would eliminate a “grumbling appendix,” but such a diagnosis is extremely unlikely to be correct—though the removal of a normal appendix is common in these children. An allergic cause can be virtually excluded if there is no other indication of allergy. In some cases the diagnosis may be migraine, the so-called periodic syndrome, in which there is any combination of abdominal pain, vomiting, headache, and fever, nearly always with a family history of migraine. Some children with migraine lose their attacks when they avoid cheese, chocolate, and some related foodstuffs.

If the family doctor concludes that the cause is entirely emotional, some understanding of the mechanism would be desirable. Unfortunately we do not fully understand how emotion and stress cause abdominal pain. It is clear that the pains may be related to imitation—for example, of a parent who is constantly complaining of his gastric discomfort—or fear in the child’s mind that he will experience the same pain as his parent. The child may feel fear and anxiety about stress at school—bullying by a teacher or child, distaste for arithmetic or other subject. Or the pain may be due to suggestion.

(again from a friend or parent). It may be an attention-seeking device (if his complaints cause anxiety and consternation in his parents), an escape device (from arithmetic or troubles resulting from inadequately prepared homework), or be due to feelings of insecurity arising from unkindness or excessive strictness at home or school. The family doctor is in the best position to elucidate the causative factors, though he should bear in mind that a child who has 10 attacks of abdominal pain of emotional origin may still develop acute appendicitis or other acute abdominal condition—the diagnosis is not necessarily the same every time.

Treatment is difficult. It includes reassurance that there is no organic disease, the reduction or removal of stress where possible, advice about the parental attitude to the symptoms (or to any disease), with special regard to the importance of avoiding suggestion of symptoms, anxiety about them, or any of the factors which may lead to psychogenic pains. But the treatment decidedly does not include purgatives, sedatives, tranquilizers, or antidepressants; and antispasmodics are apparently of no avail.

### Syphtilitic Lymphadenitis

Syphilis can still play strange tricks on clinicians, and clinical diagnosis may be anything but easy. Quite apart from the extraordinary variations shown in symptoms and signs, the moral stigma which still attaches to the condition acts as a barrier to awareness, resulting from an instinctive but false conviction that this cannot be an infection of people of good standing. Even so, modern diagnostic methods are so accurate and so easy to apply that it is surprising to hear of cases in which the pathologist has made the diagnosis by section of lymphatic nodes. R. J. Hartsock and colleagues found that syphilis had not been suspected in three out of four cases of luetic lymphadenitis from which material was examined at the U.S. Armed Forces Institute of Pathology during the years 1965 and 1966.

In reviewing the material at the institute they found an additional 16 cases in which Treponema pallidum had been identified in lymph nodes. The nodes had been removed from the inguinal region in 17 cases, from axillae in 2, from the neck in 1, and from the occipital region in 1. The histological changes showed considerable variations. Of the inguinal nodes 14 had varying degrees of chronic inflammation and extensive fibrosis of capsular and pericapsular areas; sheets of plasma cells in the interfollicular, capsular, and pericapsular areas; vascular changes, such as phlebitis and endarteritis; and follicular hyperplasia. It was thought that this combination of findings, though not specific for syphilitic lymphadenitis, was sufficiently distinctive to suggest the possibility in the differential diagnosis. Treponemes were found in all these lymph nodes. Other findings were variable numbers of polymorphonuclear leucocytes between the follicles and in the sinuses. A distinctive feature was the degree of inflammation and fibrosis of the capsular and pericapsular tissues, which was usually greater in these specimens than in non-syphilitic lymph nodes.

The lymph nodes removed from axillary, cervical, and occipital regions showed a non-specific follicular hyperplasia which did not suggest the diagnosis of syphilis, but the patients had positive serological tests for the disease, and sections showed treponemes in the walls of blood vessels and in one case in the germinal centres. An inguinal lymphatic node from one patient showed follicular hyperplasia with numerous non-caseating granulomata in and around germinal centres and also multiple foci of organizing supplicative inflammation. Treponemes were identified in the granulomata, and the authors believed that these appearances represented part of the tuberculoid reaction which can occur in syphilis. Five other inguinal nodes showed tuberculoid reactions, characterized by organized collections of histiocytes, frequently associated with giant cells of the Langhans type. Five lymph nodes in the series showed foci of supplicative inflammation, but treponemes were identified both in the walls and in the necrotic centres of these abscesses. No other causative agent was detected after careful investigation.

In one case, believed to be of early primary syphilis, the lymph nodes showed diffuse hyperplasia with dilated sinuses. There were only a few plasma cells and minimal inflammation of the capsule. The cellular response consisted mainly of lymphocytes and reticular lymphoblasts. Treponemes were demonstrated in the walls of the blood vessels and of the subcapsular sinuses. These changes possibly represent the earliest histological response to syphilitic infection.

Study of the clinical records of the 20 patients showed that nine of them complained of a painful mass in the inguinal region, though the complaint of pain in these cases is usually regarded as unusual. Six of the patients were submitted to surgical procedures because the diagnosis of incarcerated hernia could not be excluded; only three of these patients were known to have syphilis before the operation was undertaken. Apart from incarcerated hernia, early diagnoses included tuberculoid glandular fever, Hodgkin's disease, malignant lymphoma, undescended testicle, lymphogranuloma venereum, and granuloma inguinale. The final conclusion was that 9 patients had primary and 11 secondary syphilis. Thus it seems that this disease is still a considerable problem for the diagnostician.

### Infected Feeding-stuffs

Two substantial problems are associated with the high-protein dehydrated foods supplied to animals. One is related to the use of antibiotics in feeding-stuffs and the danger of bacterial resistance, both direct and transferred. Much publicity has already been given to this hazard. The second is that salmonellae and perhaps other pathogens may lurk in the finished products.

The public health is in danger when animals excreting salmonellae are slaughtered and processed under conditions which allow the spread, survival, and multiplication of the organisms in carcass meat, poultry, offal, mince-meat, and sausage skins. The hazards to those who handle the contaminated materials during manufacture, in the shop, and at home are undoubted. Though cooking should destroy salmonellae in meat and poultry products, safety from heat treatment is not always assured. Furthermore contamination from hands, surfaces, utensils, cloths, and the equipment used in preparation for cooking may be readily passed to foods which are not cooked or require only light cooking, or it may be

---


---