two months, fewer patients show the degree of recovery which is common in the Guillain-Barré syndrome, and it is probably best to set two months as the limit for diagnosis of it.

The syndrome is probably due to a cell-mediated autoimmune process—that is, an immunity reaction mediated by lymphocytes and allied cells. The evidence comes partly from work on animals and partly from the finding of lymphocytic infiltration in the nerves of patients within the first few days of the illness. Corticosteroid treatment has not given much benefit in the acute syndrome. An occasional patient may apparently improve dramatically, but for others, particularly those on respirators, the complications of high doses of corticosteroids may prove lethal. A multicentre trial is required to define the therapeutic place of these drugs.

Autoimmune activity seems to continue many years after recovery from the acute syndrome. Infiltration of the peripheral nerves by inflammatory cells has been noted, and lymphocytes sensitized to peripheral-nerve myelin have been found in the peripheral blood, though these cells sometimes disappear with clinical recovery. It is perhaps surprising, in view of this continuing activity, that relapses are so uncommon after acute idiopathic polynuropathy.

A fluctuating neurological deficit, or occasionally a pattern of complete remissions and relapses, more frequently follows an attack lasting longer than two months than a shorter one. Some of these patients—and the percentage remains to be defined—respond dramatically to treatment with corticosteroids, though the dose may have to be higher than 50 mg of prednisone or 80 IU of corticotrophin per day. These patients may also show an equally dramatic relapse on reduction of the dose, often by quite a small amount. The best-documented example of this was a patient reported by J. H. Austin, with 20 relapses over five years related to variation in the dose of corticosteroids. Such patients are usually described as having a relapsing or recurrent polynuropathy, though it is perhaps inappropriate to use this term when exacerbations are related to treatment.

W. B. Matthews and his colleagues have added a further three cases of chronic progressive neuropathy showing improvement with corticosteroid treatment and relapses on reduction of the dose. In their first case a gradually increasing dose of prednisone was required to hold the disease in check. In some remissions the patients became free of symptoms, but relapses were so profound as to lead to tetraplegia and respiratory embarrassment. At that time the patients were obviously worse than before the beginning of corticosteroid therapy. The authors rightly conclude that it is impossible to relate their clinical condition during such a relapse to what it would have been if corticosteroids had never been started, but they suggest that such a severe progression might not have been expected. Their unfortunate experience is not necessarily seen in all cases. Despite the dramatic sensitivity of these patients to slight reductions in dosage it is possible to treat effectively and even to cure some of them, eventually withdrawing the corticosteroid without producing a relapse.

Some patients with a chronic peripheral neuropathy may show no response to corticosteroids, while others may recover on treatment with them. The report by Matthews and his colleagues suggests that there is a third group who may initially improve but finally deteriorate. The proportion in each group is not at present known, and it seems impossible to forecast from the clinical features, including the level of protein in the cerebrospinal fluid, what the effect of treatment will be on an individual patient. But it is important to be aware of this third group in weighing the potential dangers of corticosteroid therapy against the present disability of the patient.

Disorders of the Distal Airways

Acute bronchiolitis is a more dramatic illness in small children than in their elders. A rational basis for this clinical observation has now been provided by J. C. Hogg and colleagues, for they have shown that the diameters of distal airways remain constant from birth until about the age of 5 years and only then increase in size. It follows that infants have a higher peripheral airways resistance than older children and adults and thus a greater tendency to airways occlusion. In the absence during early life of an adequate system of collateral ventilation from other alveoli this may lead to atelectasis and bronchiectasis.

The conducting airways of the adult lung progressively increase in total cross-sectional area at successive levels of branching. Distal airways as a whole should therefore offer less resistance to airflow than proximal ones, and this has been confirmed in animal experiments. This work suggests that conventional clinical and spirometric tests might be normal in adult patients despite narrowing of the distal airways. However, special techniques can detect disorders of function. For example, increased residual volumes attributed to premature closure of narrowed peripheral airways during expiration have been reported in bronchitic patients with relatively normal spirometric tests of ventilatory capacity. Abnormal distribution of ventilation and perfusion have also been found by means of radioactive xenon in patients with apparently mild chronic bronchitis. Recently G. Levine and colleagues examined groups of bronchitic and asthmatic patients in clinical remission and found that alveolar-arterial oxygen gradients and the ratios of physiological dead space to tidal volume were abnormally high. In a similar group of patients investigated by A. J. Woolcock and colleagues dynamic compliance was reduced and "frequency-dependent," that is, the lung was less distensible at fast than at slow respiratory rates because more time is needed during each breath to fill the alveoli supplied by a partially occluded airway.

The main practical conclusion to be drawn from these physiological studies is that narrowing of the smaller distal
Causalgia

"Causalgia" originally denoted a burning quality in pain.1 Later it was used to describe a painful syndrome commonly found in wartime traumatic casualties.2-4 Though the severity of the pain may be variable, its description always includes a burning, scalding, searing, or hot quality. The cause of the distressing symptoms is usually an incomplete lesion of a peripheral nerve in an arm or leg. Causalgia can also follow injury to the plexuses. The pain may be spontaneous but may be aggravated or precipitated by touch or movement. The limb may show typical skin changes with tightness, redness, and sweating. If the pain is severe enough to prevent the full use of the limb trophic changes appear in the skin and nails, and the bones become osteoporotic.

The essential lesion in causalgia is thought to be damage to the sympathetic fibres along the nerve; possibly an abnormal synapse may develop between afferent sympathetic and afferent somatic fibres at the site of injury.5-7 The most effective treatment is sympatheticotomy or sympathetic block. Preganglionic sympatheticotomy is the most effective operation for the relief of the pain.8

A less well recognized form of causalgia, not so disabling but with similar pain, may be encountered in peacetime.9 Recently F. P. Wirth and R. B. Rutherford9 reported 32 such examples of "minor causalgia" taken from the records of the Johns Hopkins Hospital. The main symptom was again burning pain, with increased sensitivity to touch or pressure in some cases. Symptoms affected the leg in 23 and the arm in 9. The cause of the pain was variable; a nearby fracture in

10, surgery in 5, sprains in 4, crush injury in 4, and a miscellaneous variety in the remaining 9. Twenty-seven of the patients were treated by sympathectomy, while in four others sympathetic block was sufficient to give lasting relief. Twenty-four obtained relief from sympathectomy, and in most the improvement was maintained.

The difference between major and minor causalgia may be simply that of degree. Certainly when 310 subjects9 with peripheral nerve injuries were questioned closely 19% had symptoms of major causalgia and 8% had transient symptoms of minor causalgia. In another report of 160 cases10 minor causalgia occurred in 14%. The disparity between the cause and the severity of the symptoms in minor causalgia raises certain difficulties. The diagnosis may not be obvious at first, and neurological disease may be suspected. Multiple sclerosis, syringomyelia, tabes, post-herpetic neuralgia, or thalamic pain may mimic the same symptoms. The paucity of physical signs may raise the suspicion of psychoneurosis or hysteria, and any question of compensation will only complicate the problem further. However, and perhaps surprisingly, in Wirth and Rutherford's report9 all 6 cases of the 32 where compensation was known to have been implicated were relieved by sympathectomy. Where symptoms are suggestive and there is a possible predisposing cause such as an injury then paravertebral sympathetic block is probably the most reliable diagnostic test.

Sickness and Job Satisfaction

Sickness benefit may seem barely adequate to the off-sick breadwinner trying to maintain a family. However, the total annual cost of such benefits—now running at around £380 million—when added to estimated gross income lost to the community as a result of the more than 300 million days lost from sickness makes a sum rather larger than the annual cost of the N.H.S.

It is notoriously difficult to make realistic estimates of the cost of a particular social phenomenon, but the Office of Health Economics in its latest pamphlet Off-Sick1 has attempted to do so, while at the same time assembling a useful array of information on sickness absence. The absolute level of sickness absence cannot be accurately estimated, but the official statistics do show trends from which changes in the pattern of sickness absence can be deduced. A particular omission from the figures is the non-records of periods of three days or less, which nevertheless are common. Sickness absence, particularly short-term spells, is bound to disrupt industry. Recent strikes where a small handful of men have thrown out of work whole industries demonstrate clearly the fine equilibrium of an advanced economy. So it is not surprising that short but frequent spells off by workers or the unexpected sickness of one or two key people can have a serious effect