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THE GUILLAIN-BARRÉ SYNDROME

The Guillain-Barré syndrome may be defined as 'an acute diffuse infective disease of the nervous system involving the spinal cord and peripheral nerves and occasionally the brain'. The condition has variously been called acute toxic polyneuritis, acute febrile polyneuritis, acute infective polyneuritis, and polyradiculoneuritis.[1] In the original paper, Guillain, Barré and Strohl laid down three criteria necessary for the diagnosis of this group of diseases, viz. (1) albuminocytological dissociation, (2) preponderance of motor weakness over sensory disturbances, this being mostly of a subjective nature, and (3) remarkably rapid and complete recovery despite the initially serious appearance. However, in 1938 Guillain dropped the rigid criteria, agreeing that the protein in the cerebrospinal fluid was not always so high, and that the disease was sometimes fatal. The relation of Landry's paralysis to this syndrome has always been debated. In 1949 Haymaker and Kernohan[2] concluded that the various disorders, Guillain-Barré syndrome, acute infectious polyneuritis and Landry's paralysis fell into the same syndrome, and they used the term 'Landry-Guillain-Barré syndrome'. Crozier and Ainley[3] would agree with this, but Brain, while admitting that the distinction between Landry's paralysis and the Guillain-Barré syndrome is somewhat indefinite, considers that, in the typical Landry's paralysis, while the motor disturbances are similar to those of the Guillain-Barré syndrome, the sensory loss is slight or absent.

The pathology of this syndrome has been well described.[4-6] The basic pathological process is a pronounced oedema of the nerve fibres of the spinal roots and the proximal portions of the cranial and peripheral nerves. This appearance is seen early in the disease. There is narrowing and obliteration of the perineural spaces, and in more severe cases strangulation of the radicular trunks due to the oedema. Degeneration of the myelin sheath in the peripheral nerves and central nervous system may follow. Obliteration of the perineural spaces blocks the absorption of the cerebrospinal fluid along these channels and leads to stagnation and trapping of the fluid within the subarachnoid space. This obstruction permits the absorption of fluid and electrolytes but not the larger protein molecules, the classical albuminocytologic dissociation resulting. Austin,[7] however, notes that simple 'stagnation' of previously elaborated protein does not alone explain the increase in protein in the cerebrospinal fluid.

Typically the Guillain-Barré syndrome develops a few days after a mild upper-respiratory-tract infection. It may also follow upon a mild gastro-intestinal upset. The motor signs are those of bilateral, symmetrical, progressive weakness and, later, flaccid paralysis with selective involvement of the proximal parts of the limbs. The superficial and deep reflexes are either diminished or absent. Sensory disturbances may be subjective or objective. The prognosis must be guarded,[8] and the course is variable. The usual duration of the illness is from 3 to 6 months, but it sometimes continues for as long as three years. Recovery is usually complete, though 5-10% of cases are left with significant permanent sequelae. The mortality rate varies in different series from 20% to 42%.[6,9]

Numerous drugs have been used in the treatment of the condition, and it is difficult, if not impossible, to determine whether any drug favourably changes the natural course of the disease. The concept of an allergic basis in this disease prompted the use of steroid hormones, and their use was first reported in 1952.[10] It is stressed that ACTH and cortisone must be given at an early stage, while the oedema is still present, and not at the stage of chronic root strangulation—when the drugs will be of no value.[6] However, not everyone is agreed on the value of steroid therapy, and in 1953 Plum[11] wrote: 'Thus improvement of polyneuropathy following corticotropin or cortisone therapy appears to be but an occasional unpredictable phenomenon, possibly only of coincidental significance'.

Recurrence in the usual, untreated case of the Guillain-Barré syndrome is rare.[12] It is of great interest that from one-third to one-quarter of reported treated cases suffered recurrence. While there are various explanations for this, Austin suggests that the fact that recurrences do occur, and often after the withdrawal of the drugs, indicates a true drug effect which may separate a more homogeneous, responsive group from the heterogeneous Landry-Guillain-Barré syndrome.[7]

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BEHANDELBARE GEBREKE BY KINDERS

Die opkoms van die voorkomende medisyne gedurende die laaste aantal jare het daartoe gelei dat die aandag van geneeshere sowel as van welsynsliggame en lede van die algemene publiek sterker as ooit tevore gevestig is op die behandelbaarheid van baie gebreke wat voorheen nie altyd doeltreffend benader is nie. Twee belangrike voorbeelde in hierdie verband is die probleem van hardhorendheid (waaroor ons elders in hierdie uitgawe 'n artikel plaas)

en versteuring van die ontwikkeling van spraak by klein kindertjies (waaroor ons 'n brief in hierdie uitgawe plaas).

Dit is ontstellend om te dink dat volgens berekening ongeveer 10% van alle mense hardhorend is—te meer so omdat die probleem van hardhorendheid, anders as dié van doofheid, veelal in die verlede verwaarloos is.

Dit word vandag al meer besef dat 'n hele reeks moeilikhede met skolastiese vordering op skool, sowel as baie vorms van wanaanpassing by kinders, toegeskryf kan word aan hardhorendheid in die een of ander graad. Aan die ander kant word dit ook besef dat geweldig baie gedoen kan word om die nadelige gevolge van hardhorendheid te voorkom. In die artikel oor die probleem van hardhorendheid wat ons plaas, word daar verwys na wat behoort gedoen te word by die bekamping van hierdie toestand. Kortliks kom dit daarop neer dat ons benadering só omvangryk moet wees dat dit al die fasette van voorkoming, behandeling, konservasie en kompensasie insluit.

Dit is die plig van ouers, onderwysers, werkgewers en gesondheidsbeamptes om toe te sien dat die probleem van hardhorendheid op 'n rasionele en daadwerklike basis aangepak word. Dit is dus nie genoeg dat daar sentrale gehoorsentrums, wat ten volle uitgerus is, aan al ons opvoedkundige inrigtings en gesondheidsdepartemente bestaan nie; ons moet ook toesien dat die aktiewe belangstelling van die samelewing as geheel opgewek en wakker gehou word.

'n Soortgelyke probleem as die van hardhorendheid, is die probleem van gebrekkige ontwikkeling van spraak by kinders. Dit gebeur byvoorbeeld glad te dikwels dat onderwysers, maatskaplike werkers en geneeshere met huiwering deur ouers genader word wat kinders het wat al redelik gevorderd is wat hul algemene ontwikkeling betref, maar wat nie wil of kan praat nie, of gebrekkig praat—en dat die saak dan met 'n skertslag en met die ophaal van die skouers afgehandel word, en met die versekering dat die kinders tog vroeër of later wel sal begin praat.

Ons wil dit beklemtoon dat die soort benadering wat ons nou net geskets het, verkeerd is. Seer sekerlik moet daar nie oorhaastig opgetree word sodat die ouers skrik en gedwing word om onnodige en duur ondersoeke te laat doen nie. Maar, aan die ander kant moet daar gesorg word om nie behandelbare spraakgebreke mis te kyk nie. Want, indien dít gedoen word, kan onnoembare skade aan die ontwikkelende ego van die kind berokken word deur die gevolge van sy tekortkoming of gebrek; byvoorbeeld, wanaanpassing en frustrasie in sy omgang met ander kinders kan ontstaan en ook vertraging van sy vordering op skool.

Die twee voorbeelde wat ons hier genoem het, is maar enkele voorbeelde uit baie meer gevalle van probleme wat soms nie as direkte mediese probleme beskou word nie, maar wat dit tog in werklikheid is. Hier, soos op soveel ander gebiede, kan die voorkomende medisyne 'n groot positiewe bydrae lewer tot die welstand en geluk van kinders sowel as van volwassenes.