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DIE BESTRYDING VAN KAAKKLEM

Kaakklem val vandag nog eweveel binne die bestek van die epidemiologie as van die kliniekwerk. Totdat aktiewe bestandmaking op die hele bevolking toegepas word[1] sal kaakklem voortgaan om sy jaarlikse aantal slagoffers te eis. Die hoogste sterftesyfer kom onder adolessente voor; hulle kom tewens baie meer met besmette grond in aanraking as ander ouderdomsgroepe. In Brittanje, waar die epidemiologiese standaarde betreklik hoog is, sterf gemiddeld 50 mense elke jaar aan kaakklem.[2] Die Noord-Amerikaners het baanbrekerswerk gedoen insake die voorbehoedende gebruik van kaakklem-toksoïed om aktiewe onvatbaarheid te verleen, met die gevolg dat daar in die laaste oorlog slegs een geval van kaakklem onder al die slagveld-gewondes van die Amerikaanse leër voorgekom het. Soortgelyke resultate, wel minder skouspelagtig, is ook in Brittanje behaal. Twee dosisse, 6 weke uit mekaar, word aanbeveel, met 'n derde 6 maande later. By kinders kan dit saam met die difterie-inenting gedoen word; dit is vandag verpligtend in Denemarke en Frankryk[3] en gebruiklik in die VSA en Kanada. Opwekkingsdosisse al om die 4 jaar word aanbeveel. Al die Amerikaanse gewondes het so 'n opwekkingsdosis gekry in plaas van die kaakklem teengif wat gewoonlik gebruik word en wat op sy beste die pasiënt maar twee weke lank passief onvatbaar maak. Gewoontes word maar seker net so langsaam gebore as wat hulle uitsterf, sodat dit miskien 'n tyd lank sal duur voordat aktiewe bestandmaking teen kaakklem as reël in Suid-Afrika toegepas sal word. Nogtans het die ondervinding geleer dat die velbesering waardeur kaakklem kan ontstaan dikwels so gering is dat dit verontagsaam word totdat die besmetting intree.

Intussen vind daar fundamentele her-ondersoek en verandering plaas op die gebied van die gebruiklike begrippe oor die beheer van die akute kaakklem-aanval. Dit word beweer[4] dat die werking van die kaakklem-toksien op die onderdrukking van die remprikkels op die senuweetussenselle berus. Hierdie verwydering van fisiologiese stremming stel die sinaptiese verbindings bloot aan al die aangevoerde opwekkende prikkels, en selfs geringe prikkels word in aktiwi-

EDITORIAL

COMBATING TETANUS

Tetanus remains as much the field of the epidemiologist as of the clinician. Until active immunization is applied to the whole population,[1] tetanus will continue to take its annual toll of lives. The highest death rate occurs amongst adolescents, whose contact with infected soil is likely to be greater than that of people in other age-groups. In Britain, where epidemiological standards are fairly high, about 50 persons die each year of tetanus.[2] The North Americans have pioneered the prophylactic use of tetanus toxoid to induce active immunity, and as a result only one case of tetanus occurred among all the battle casualties in the American army during the last war. Similar, though less spectacular, results were achieved by the British. Two doses are advised, spaced 6 weeks apart, followed by a third dose 6 months later. In children this may be combined with diphtheria immunization, as is now compulsory in Denmark and France,[3] and widely practised in the United States and Canada. Booster doses every 4 years are advised. All American casualties were given a booster dose instead of the tetanus antitoxin customarily employed, which at best invests the patient with only 2 weeks of passive immunity. Customs are likely to take as long to be born as to die, so that one cannot anticipate the introduction of active immunization against tetanus in South Africa for some time. Nevertheless, experience has shown time and again that the offending skin-lesion in tetanus is often so trivial as to be ignored until the infection sets in.

Meanwhile, traditional concepts on the management of the acute attack of tetanus are undergoing fundamental re-examination and change. The tetanus toxin acts, it is said,[4] by depressing inhibitory stimuli on the interneurones of the nervous system. This removal of physiological restraint leaves the synaptic junctions

teit uitgedruk. Tensy die afvoerende senuweeketting onderbreek word, of die pasiënt met kalmeermiddels fisiologies op normaal gebring word, veroorsaak hierdie abnormale beweeglikheid spoedig uitputting en die dood. Twee soorte kliniese middels is reeds gebruik. Die radikaalste metode is om totale verlamming van die hele willekeurige spierstelsel te veroorsaak met 'n spier-senuwee-blokkeringmiddel soos kurare. Om die emosionele afgryslikheid van totale verlamming met volle bewussyn te voorkom, word die pasiënt onder ligte narkose gehou. Hierdie oorspronklike metode is deur Lassen en sy medewerkers ontwikkel nadat hulle groot sukses behaal het met gevalle van bulbêre poliomiëlitis en asemhalingsversaking gedurende die poliomiëlitis epidemie van 1952 in Kopenhagen. Vroeë tracheotomie en aanhoudende positiewe druk-asemhaling het talle lewens gered. In 1954 het hulle verslag gedoen oor 4 gevalle van kaakklem wat met welslae op hierdie manier behandel is.[5] Die nadele is ooglopend. Die pasiënt wat vir 'n week of langer bewusteloos is verg die onafgebroke toesig van 'n bekwame narkotiseur en die noulettendste uur-vir-uur verpleging—iets wat nie altyd in die praktyk uitvoerbaar is nie, afgesien van die ander belangrike gevare van kunsmatig veroorsaakte bewusteloosheid. Lassen en sy medewerkers het self onlangs die aandag gevestig op die ernstige hematologiese komplikasies wat deur langdurige verdowing met stikstofsuboksied veroorsaak kan word.[6]

Die ander benadering tot die beheer van stuiptrekkings wat tans weer ondersoek word, is die gewone een van kalmering. Die ideaal waarna hierby gestrewe moet word is 'n middel wat stuiptrekkings van die spiere sal beheer sonder om die bewussyn of asemhaling te belemmer. Soos die meeste ideale sal hierdie een van 'n middel wat aan albei vereistes voldoen seker onverwesenlik bly, maar belangrike sukses is reeds behaal met die barbiturate, met mephenesin, en onlangs ook met chlorpromazine. Parentale of binneaarse gebruik van die barbiturate is reeds jare lank standaard behandeling, maar die asemhaling en bewussynsgraad word onvermydelik verlaag, en selfs met antibiotiese beheer en strenge verpleging kom komplikasies van die asemhalingsstelsel nogal dikwels voor. Dit is bekend dat mephenesin en chlorpromazine, wat albei hulle invloed uitoefen deur die geleiding van prikkels deur die senuweetussenweefsel te belemmer, doeltreffend is teen spierkrampe, en laasgenoemde is reeds met goeie gevolg gebruik by die beheer van kaakklemstuipe in 'n kind.[7] Moontlik sal kombinasies van hierdie middels, aan ons doel beantwoord, soos die suksesvolle gebruik van onderbroke binneaarse thiopentone en senuwee-spier-blokkeringsmiddels wat Forbes en Auld[8] gerapporteer het.

1. Van die Redaksie (1954): S. Afr. T. Geneesk., **28**, 584.
2. Byskrif (1956): Lancet, **1**, 493.
3. Scheibel, I. (1955): Bull. Wld. Hlth. Org., **13**, 381.
4. Brooks, V. B., Curtis, D. R. en Eccles, J. C. (1955): Nature (Lond.), **175**, 120.
5. Lassen, H. C. A., Bjorneboe, M., Ibsen, B. en Neukirch, F. (1954): Lancet, **2**, 1040.
6. Lassen, H. C. A., Henriksen, E., Neukirch, F. en Kristensen, H. S. (1956): *Ibid.*, **1**, 527.
7. Kelly, R. E. en Laurence, D. R. (1956): *Ibid.*, **1**, 119.
8. Forbes, G. B. en Auld, M. (1955): Amer. J. Med., **18**, 947.

exposed to all incoming excitatory impulses, and even trivial stimuli are now transmitted into activity. Unless the efferent nervous chain is broken or the patient sedated into physiological normality, this abnormal activity rapidly leads to exhaustion and death. Two kinds of clinical remedies have been applied. The more radical approach has been to induce total paralysis of all voluntary musculature with a neuromuscular blocking agent such as curare and—in order to obviate the emotional horror of being totally paralysed yet fully conscious—to maintain the patient in a state of permanent light anaesthesia. This novel method was evolved by Lassen and his colleagues after their success with cases of bulbar poliomyelitis and respiratory distress in the 1952 poliomyelitis epidemic in Copenhagen. Many threatened lives were saved by early tracheotomy and continuous positive-pressure respiration. In 1954 they reported 4 cases of tetanus that had been treated successfully in this way.[5] The drawbacks are obvious. A patient who is unconscious for a week or more requires the constant surveillance of a skilled anaesthetist and the most stringent round-the-clock nursing—something that is not really practicable, quite apart from all the other not inconsiderable hazards of induced unconsciousness. Moreover, Lassen and his colleagues have themselves recently drawn attention to serious haematological complications attributable to prolonged nitrous-oxide anaesthesia.[6]

The alternative approach to the management of convulsions that is being re-explored is the more usual one, viz. sedation. Here the ideal to be aimed at is the drug that will abolish, or at any rate control, muscle spasm without impairing consciousness or respiration. Whilst this combination, like most ideals, is probably unattainable, substantial success has been encountered with the barbiturates, with mephenesin, and more recently with chlorpromazine. Parenteral or intravenous use of the barbiturates has been standard treatment for many years, but respiration and the level of consciousness are inevitably depressed and the incidence of respiration complications, even with antibiotic coverage and strict nursing, is high. Mephenesin and chlorpromazine, both of which act by depressing conduction of impulses through the internuncial neurones, are known to be effective against the muscular spasm of tetanus, and good use of the latter drug has been made in controlling tetanus convulsions in a child.[7] Perhaps the answer lies in combinations of these drugs, such as the successful use of continuous intravenous thiopentone and neuromuscular blocking agents, reported by Forbes and Auld.[8]

1. Editorial (1954): S. Afr. Med. J., **28**, 584.
2. Annotation (1956): Lancet, **1**, 493.
3. Scheibel, I. (1955): Bull. Wld. Hlth. Org., **13**, 381.
4. Brooks, V. B., Curtis, D. R. and Eccles, J. C. (1955): Nature (Lond.), **175**, 120.
5. Lassen, H. C. A., Bjorneboe, M., Ibsen, B. and Neukirch, F. (1954): Lancet, **2**, 1040.
6. Lassen, H. C. A., Henriksen, E., Neukirch, F. and Kristensen, H. S. (1956): *Ibid.*, **1**, 527.
7. Kelly, R. E. and Laurence, D. R. (1956): *Ibid.*, **1**, 119.
8. Forbes, G. B. and Auld, M. (1955): Amer. J. Med., **18**, 947.