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ASMA

Die woord ‚asma' beteken vir die pasiënt fluitende asemhaling van welke oorsaak ook al. Die allergoloog sien dit as brongospasme as gevolg van 'n spesifieke hipersensitiwiteitsreaksie. Die psigiater is geneig om dit as 'n psigosomatiese siekte te beskou. Ons onderskei ook nog kardiale asma waar longedeem met brongospasme gepaard gaan.

Die woordjie ‚brongospasme' kan egter onder 'n vergrootglas geplaas word. As ons in kliniese sin praat van brongospasme, bedoel ons dat ons rongusse hoor, en lei daarvan af dat daar vernouing van brongusse en/of brongiole is, hetsy die gevolg van swelling van die slymvlies of spasme van die spierwand van die brongus. Daar word egter twyfel gewerp op die bestaan van ware spasme van brongiale spiere, en dit is veral onder torakale chirurge dat hierdie twyfel sterk op die voorgrond kom. Gilfillan[1] het twaalf pasiënte tydens segmentele longreseksie vir tuberkulose bestudeer. Die helfte het 'n geskiedenis van brongospasme gehad. Die brongiolêre bewegings is met 'n handlens bestudeer tydens operasie en ook 'n paar histologiese studies van brongiole is gedoen. Daar was geen spierhipertrofie in die asmalyers se brongiole nie. Vanuit 'n ander rigting het Wells[2] die probleem benader deur die meganika van asemhaling tydens asma-aanvalle te bestudeer. Die inspiratoriese en ekspiratoriese weerstand van die lugweg is albei vermeerder en na terapie is daar dikwels 'n groter verbetering in die inspiratoriese weerstand as in die ekspiratoriese weerstand. Dit weerspreek die opvatting van verhoogde ekspiratoriese weerstand as die hooffaktor in brongiale asma. Die werk van asemhaling is baie verhoog in asma, en dit is die geval byna uitsluitlik as gevolg van werk om lugweg-weerstand te oorkom. Hierdie bevindings sou meer ten gunste van edeem en afsluiting van brongusse deur afskeidings wees, hoewel brongospasme nie uitgesluit is nie.

So dikwels is mense met brongitis geneig tot asma aanvalle, veral in die winter, dat waar 'n geskiedenis van asma na 'n episode van infeksie van die boonste lugweg plaasvind, mens kan praat van brongitiese asma. Die dramatiese gevolge van antibiotiese terapie ter verligting van hierdie asma kan miskien ook gesien word as 'n verbetering van die lugweerstand deur afskeiding en edeem te verminder eerder as die opheffing van spierspasme om die brongusse. Geensins wil mens die brongospastiese element wegpraat nie, maar die klem net verskuif na die belangrike rol van edeem van die slymvlies tydens so 'n aanval.

Dat daar veelvuldige faktore vir hierdie edeem van die slymvlies is, is welbekend. Die volgende is voorbeelde hiervan: vogretensie van allergie (soos die nasale slymvliesswelling van hooikoors), inflammatoriese edeem (soos reeds genoem), en langdurige hipoksie met verhoogde kapillêre deurlaatbaarheid. Die dramatiese effek van kortisoon kan miskien in hoofsaak die vermindering van inflammatoriese edeem beteken, terwyl hipertoniese glukose ook op hierdie edeem deur dehidrasie sy effek mag hê. Vogtige klimaatstoestande wat verdamping van vog belemmer, is nadelig terwyl droë klimaatsomstandighede voordelig mag wees.[3]

Die ‚honger' na lug, 'n primitiewe behoefte in mens en dier, kan selde sonder emosionele reaksie geskied. Vrees is dus sowel 'n gevolg as 'n oorsaak van asma.[3] Die pogings van die pasiënt om lug deur hierdie vernoude buisie te stoot teen 'n hoër tempo verg meer werk en dus verdere uitputting, 'n groter behoefte aan suurstof vir spierwerking, en 'n verergering van die edeem as gevolg van hipoksie.

Waar onlangse werk dus meer die klem op die edeem en swelling van die brongiale slymvlies as op spasme van brongiale spiere laat val, moet mens miskien ook dienooreenkomstig jou begrip van asma effens wysig.

1. Gilfillan, R. R. (1958): J. Thorac. Surg., **36**, 63.
2. Wells, R. E. (1959): Amer. J. Med., **26**, 384.
3. Hinshaw, H. C. en Garland, L. H. (1956): *Diseases of the Chest*, Londen: Saunders.

RHEUMATOID ARTHRITIS

Steroid therapy in rheumatoid arthritis was first instituted about ten years ago by Hench *et al.*[1,2] at the Mayo Clinic. During these years many investigations have been carried out on the use of the adrenocortical steroids and their synthetic analogues in this disease, and there is now a fair understanding of the indications and contra-indications for the use of these potent agents. How they produce their beneficial effects is still virtually unexplained. Their benefits are only palliative or suppressive, not curative. They need to be given for an indefinite period, and there is always the risk of side-effects occurring, some of which are serious. Some patients become relatively refractory to these drugs after prolonged administration. The progress of the rheumatic process may not be halted, even when symptomatic relief is being maintained. In view of these difficulties certain policies have been adopted for the employment of the steroids. An authoritative review of this subject has recently been published by Boland.[3]

Steroid therapy should only be used in carefully selected cases where there is active and potentially reversible disease. Conservative treatment should first be employed, especially in mild disease with good chances of natural reversal. The optimum dosage for selected patients is an individual matter; and complete inhibition of the disease should not generally be sought. In discontinuing steroid therapy, the drug should not be abruptly withdrawn, but the dosage should be reduced gradually. Certain emergency conditions, such as severe infections, surgical operations, or injuries, necessitate the administration of additional amounts to cope with the stress.

Reports in the literature indicate great variations in the success obtained with cortisone and hydrocortisone therapy. It would appear that adequate improvement has been maintained in more than fifty per cent of patients. Variable results are to be expected in a disease such as rheumatoid arthritis, with its varying severity and with the various

schedules of dosage used in its management. The most frequent reason for inferior results has been the development of side-reactions. During the past five years synthetic steroid analogues have been tested. Of the hundreds that have been examined only a few have been marketed.

In 1954 prednisone and prednisolone became available. These drugs are more potent in anti-inflammatory activity than the natural hormones, and their administration does not lead to an increase in electrolyte activity. They are capable of producing improvement in patients no longer satisfactorily controlled by hydrocortisone or cortisone. The general incidence of side-reactions is essentially the same as with hydrocortisone, with a difference in the proclivity to induce certain effects. They are more prone to produce peptic ulcers and cutaneous ecchymoses, but less prone to cause salt and water retention and a rise in blood pressure.

Triamcinolone was manufactured in 1956. It is peculiarly liable to produce anorexia, muscle weakness, nausea, fatigue, and erythema. Methylprednisolone is satisfactory in antirheumatic activity, but would not appear to have advantages or disadvantages when compared with prednisolone. Its potency is somewhat greater.

The latest steroid whose therapeutic efficiency has been tested is dexamethasone.[3,4] The general incidence of untoward reactions with this antirheumatic drug is essentially the same as with prednisolone when comparable doses are given. Relatively common side-effects are excessive weight gain and ecchymotic skin lesions. This drug also has ulcerogenic properties, as revealed by routine radiographic studies of the upper gastro-intestinal tract.

The ideal suppressive agent for rheumatoid arthritis remains to be found. Great advances have of course been made, and better steroids may still be produced. There is of course always the possibility that some other type of chemical compound, or indeed some other type of treatment, may yet be found.

1. Hench, P. S., Kendall, E. C., Slocumb, C. H. and Polley, H. F. (1949): Proc. Mayo Clin., **24**, 181.
2. *Idem* (1950): Arch. Intern. Med., **85**, 545.
3. Boland, E. W. (1959): Ann. N.Y. Acad. Sci., **82**, 887.
4. Neustadt, D. H. (1959): J. Amer. Med. Assoc., **170**, 1253.