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VAN DIE REDAKSIE

SPANNING VAN DIE MAAGMOND

Onlangse studies het veel aan die dag gebring aangaande die aard van die funksieversaking by die toestand bekend as kramp of spanning van die maagmond, maar voordat die verslae bestudeer word, moet ons eers die huidige menings insake slukdermsluitspiere uiteensit. Nadat dit lank die onderwerp van meningsverskil was, is dit definitief bewys dat daar 'n intrinsieke sluitspier onder in die slukderm is, omtrent 2-4 cm. bokant die maagmond; dit is bewys deur die meet van drukverval oor die streek waar die slukderm by die maag aansluit.[1] Die normale slukderm besit twee sulke streke van hoë druk—daar is nog een aan die bo-ent; en hierdie twee streke verhoud respektiewelik dat die maaginhoud en lug in die ingewand versluk word.[2] Dié deel van die esofaag tussen die onderste sluitspier en die maagmond is van groot belang by maagmondkramp; Lerche (aangehaal deur Gould en Bernhard[3]) noem dit die ‚voorhof'.

By maagmondkramp word die voorhof vernou, en daar is 'n wisselende, maar gewoonlik aansienlike, vergroting van die liggaam. Dit was eers gemeen dat die vernouing deur kramp veroorsaak was, maar hierdie verduideliking was maklik verwerp; die vernoude streek bied geen weerstand teen die passeer van stawe nie. Dit is dan as 'n groot stap vorentoe beskou toe Hurst en Rake,[4] in die loop van studies van outopsie-materiaal van gevalle van maagmondkramp, gevind het dat daar 'n vermindering in die aantal senuweeknoopselle in die vernoude deel was. Met dié feit as grondslag het hulle die teorie geopper dat die mondgedeelte van die slukderm nie kon ontspan nie (maagmondkramp). Hierdie teorie is egter nie sonder meer aanvaar nie; en dit blyk dat onlangse navorsing aan die Guy-hospitaal waar Hurst, voormalig werksaam was, hierdie stelling omvergewerp het.[2] Gedurende operasies vir verskillende slukdermafwykings, o.a. maagmondkramp, is spierstrokies verkry uit die voorhof; wat gevalle van maagmondkramp sowel as beheergevalle betref het, kon doeltreffende aantalle senuweeknoopselle gewoonlik histologies gedemonstreer word, en biochemiese toetse het bewys dat hierdie selle wel bekwaam was. Trounce *et al.*[2] vestig die aandag daarop dat ná afsterwe die voorhof nie so duidelik uitkenbaar is nie, en stel voor dat die materiaal van Hurst se gevalle miskien in werklikheid uit die vergrootte gedeelte afkomstig was. Volgens hulle is die liggaam van die slukderm abnormaal by gevalle van maagmondkramp en kan daar 'n gebrek aan senuweeknoopselle wees. Dit is nie duidelik wat hierdie vermindering veroorsaak nie, maar dit is *nie* te wyte aan sekondêre inflammasieveranderings nie.

Die werk van Kramer en Ingelfinger[5] lewer verdere bewyse van 'n verspreide stoornis in die senuweevoorsiening en beweeglikheid van die slukderm by gevalle van maagmondkramp; hulle het naamlik 'n abnormale reaksie op cholinergiese prikkeling aangetoon. Anders as normale mense of lyers aan ander slukdermafwykings, ondervind pasiënte met maagmondkramp geweldige en dikwels pynlike sametrekkings van die slukderm ná 'n spierinspuiting van 3 -6 mg. metacholien (Mecholyl). Hierdie krampe kan deurligtings-

EDITORIAL

ACHALASIA OF THE CARDIA

Recent studies have shed much light on the nature of the dysfunction in the condition known as achalasia of the cardia or cardiospasm, but their consideration should follow a brief statement of current views about oesophageal sphincters. After prolonged controversy, firm evidence of the existence of an intrinsic lower oesophageal sphincter about 2–4 cm. above the cardia has been provided by the measurement of pressure gradients across the region of the gastro-oesophageal junction.[1] The normal oesophagus possesses two of these high-pressure zones, there being another at the upper end; these respectively prevent the aspiration of stomach contents and air into the viscus.[2] The part of the oesophagus between the inferior sphincter and the cardia is of great importance in achalasia; it has been called the 'vestibule' by Lerche (quoted by Gould and Barnhard[3]).

In achalasia there is constriction of the vestibule surmounted by variable, but usually considerable, dilatation of the body. At first the narrowing was attributed to spasm, but this explanation was easily disproved; the contracted area offers no resistance to the passage of bougies. It then appeared to be a great advance when Hurst and Rake,[4] using material obtained at autopsy from cases of achalasia, stated that there was a diminution in the number of ganglion cells in the narrowed part. On this basis they postulated that there was a failure of relaxation (achalasia) of the cardiac portion of the oesophagus. However, this thesis did not achieve unquestioned acceptance; and recent work from Hurst's old hospital, Guy's, appears to have disproved it.[2] At operations for various oesophageal disorders including achalasia, strips of muscle were obtained from the vestibule; in both achalasia and the control cases adequate numbers of ganglion cells could usually be demonstrated by histological means, and biochemical tests showed that these cells were functioning. Trounce *et al.*[2] point out that after death the vestibule may become less obvious, and suggest that in Hurst's cases the material may really have come from the dilated part. According to them, the body of the oesophagus is abnormal in achalasia and may be deficient in ganglion cells. The reason for this diminution is not known, but it is not due to secondary inflammatory changes.

Further evidence of a widespread disorder of oesophageal innervation and motility in achalasia comes from the studies of Kramer and Ingelfinger,[5] who have demonstrated an abnormal response to cholinergic stimulation. Unlike normal people or patients with other oesophageal disorders, sufferers from achalasia experience violent and often painful contractions of the oesophagus after an intramuscular injection of 3–6 mg. of methacholine (Mecholyl). This can be observed fluoroscopically and is held to be of use in the diagnosis of

gewys waargeneem word en dit word beweer dat dit nuttig is by die diagnose van maagmondkramp. Daar word voorgestel dat hierdie spieraksie 'n voorbeeld van Cannon se wet is, nl. dat 'n orgaan wat gebrekkig is aan senuwee buitengewoon gevoelig is vir sekere chemiese prikkels.[6]

By maagmondkramp is die funksionele afwyking blykbaar dat daar by die slukbeweging nie 'n ware peristaltiese golf ontwikkel en langs die slukderm af beweeg nie; gevolglik ontvang die slukderm nie sy normale prikkeling om te ontspan nie. Daar is dus ‚spanning' by die maagmond, maar die gebrek aan ontspanning is ondergeskik aan verspreide siekte van die liggaam van die slukderm.

Daar is vandag nog geen definitiewe behandeling nie. Hoewel die nitriete soms nuttig is, help ander kramp-teenmiddels nie en anticholinergiese middels kan erger dan nutteloos wees; 'n spierinspuiting van 25-50 mg. methantelien bromied (Banthine) by normale gevalle kan 'n verbygaande toestand, wat na maagmondkramp lyk, in die hand werk.[7] Hierdie feit ondersteun die idee van senuweegebrek by die etiologie van hierdie siekte. Dit kan by baie gevalle nodig wees om die spiervesels van die voorhof te skeur sodat die voedsel deur swaartekrag, en met die hulp van sametrekkings van die keelholte en die boonste gedeelte van die slukderm, in die maag kan kom. Dit kan gewoonlik bewerkstelling word deur skerp rekking met 'n hidrostatiese sakkie onder X-straal-beheer; indien hierdie metode nie slaag nie, of as die slukderm té verrek en kronkelend is om die sakkie sekuur in posisie te plaas, is snykundige ingreep moontlik nodig (Heller se operasie). Dit beantwoord gewoonlik die doel, maar as die verwyding té verregaande is, kan maagslukdermterugvloeiing plaasvind, met gevolglike peptiese slukdermontsteking.

Hoewel die behandeling van maagmondkramp dus maar 'n moeilike saak bly, is daar nietemin vooruitgang, en al is die etiologie nog duister, is die patogenese tog al bekend. Maar daar moet nog baie aan hierdie probleem gewerk word voor ons aanspraak daarop kan maak dat hierdie seldsame maar belemmerende siekte oorwin is.

achalasia. It is suggested that this behaviour is an example of Cannon's law, that a denervated structure is abnormally sensitive to certain chemical stimuli.[6]

In achalasia the dysfunction appears to be that with swallowing a true peristaltic wave does not develop and move down the oesophagus; consequently the vestibule does not receive its normal stimulus to relax. There is thus 'achalasia' at the cardia, but the failure to relax is secondary to diffuse disease of the body of the oesophagus.

At present there is no definitive therapy. Although nitrites are occasionally of some use, other antispasmodics do not help, and anticholinergic drugs may be worse than useless; the intramuscular administration of 25–50 mg. of methantheline bromide (Banthine) to normal subjects may induce a transient state resembling achalasia.[7] This is another point supporting the denervation concept of the aetiology of this disease. Many cases will need to have the muscle fibres of the vestibule ruptured, so that food can enter the stomach by gravity and with the aid of pharyngeal and upper oesophageal contractions. This can usually be accomplished by brusque dilatation with a hydrostatic bag under fluoroscopic control; if this fails, or if the oesophagus is too dilated and tortuous to permit of accurate placement of the bag, operative section (Heller's operation) may be required. This usually works, but if the dilatation is too extensive, gastro-oesophageal regurgitation may result, with consequent peptic oesophagitis.

Thus, while the treatment of achalasia remains difficult, there have been advances, and the pathogenesis, if not the aetiology, is now known. But much further work is required before we can say that this uncommon but disabling malady has been mastered.

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