VAN DIE REDAKSIE : EDITORIAL

AGADIR

Kort voor middernag op Maandag 29 Februarie 1960, is die stad Agadir binne 'n paar oomblikke feitlik totaal vernietig deur 'n aardbewing. Die daaropvolgende geneeskundige ondervindings is nou so pas, veral in die Franse literatuur, geopenbaar.[1,2] Verskeie nuwe aspekte van 'n toestand wat slegs 20 jaar gelede die eerste keer duidelik erken is, verdien nou die aandag. In 1941 is Bywaters se meesterlike en reeds klassieke beskrywing van spiervergruising sonder oop wonde (‚crush syndrome') onderwerp aan groot belangstelling; maar om lonende waarnemings te maak, is dit nodig om groot getalle van post-troumatiese nieraandoenings te bestudeer. Dit strek tot groot eer van Bywaters dat sy oorspronklike waarnemings in Londen gedurende die laaste oorlog so noukeurig bevestig is in Korea deur die Amerikaners, en nou in Agadir deur die Franse.

Uitgebreide spiervergruisende beserings as gevolg van ondergrondse verpersing is in 87 van die gevalle van Crosnier en sy kollegas[1] behandel in Casablanca; dié beserings is 62 keer gevolg deur 'n mate van nierskade. Eenvoudige mioglobinurie het 21 keer voorgekom, en in 19 pasiënte was daar die meer ernstige mioglobinuriese nefrose, gekenmerk deur totale anurie wat meer as 48 uur lank geduur het, met spontane herstel daarna. In die derde Franse groepering kom sterfgevalle eers voor in die sogenaamde kwaadaardige nefrose. Elf pasiënte wat behandel is sonder die kunsmatige nier, is almal oorlede; uit 11 pasiënte wat behandel is met die kunsmatige nier, was daar slegs 7 sterfgevalle. Dit is duidelik dat aanhoudende post-troumatiese anurie 'n baie swak prognose het.

Nadat die slagoffers in Agadir dae lank onder die grond vasgeknel was, was die beste behandeling dreinasie van die uitgesproke spieredeem deur spleetformasie in fascia-omhulsels, chirurgiese verwydering van duidelik nekrotiese weefsel, en daarna noukeurige kontrole van die bloed-kalium konsentrasie (op sy beste deur herhaalde elektrokardiografiese ondersoek), sodat gevalle vir die kunsnier vroegtydig erken kon word. Kalium-ophoping in die bloed was die grootste bedreiging van die Agadir-oorlewendes. Normaalweg is daar slegs sowat 3 gram kalium in die ekstra-sellulêre vog, terwyl daar intrasellulêr 147 gram aanwesig is in die gemiddelde volwassene, en die klem het deesdae verskuif van die hemoglobinurie of mioglobinurie af tot die verlies van kalium uit die selle en die daaropvolgende miokardiale prikkelbaarheid en ineenstorting.

Inwoners van Johannesburg voel elke week of twee 'n aardbewing. Baie dokters in hierdie stad het ten tyde van die Agadir treurspel gewonder wat sou gebeur as 'n stad soos Johannesburg deur 'n ernstige aardbewing getref sou word.

In Agadir is na 3 dae van reddingswerk die bevel uitgevoer om alle reddingswerk te vervang deur ontsmettingswerk. Daar was nog groot getalle lewendes vasgevang in die ruïenes, maar dit was baie warm, en die stank van die dooies en die oop rioel-stelsels was afskrikwekkend.[3] Volgens geneeskundige raadgewing was hierdie beslissing om verdere reddingswerk te staak sodat doeltreffende ontsmetting deur Duitse leërmanskappe ingestel kon word, absoluut noodsaaklik om grootskaalse epidemies te verhoed. Lt. Fordham, 'n brandweer-offisier, wonder tereg of ons westerlinge so doeltreffend sal werk aan 'n onvoorsiene natuurramp. Die eenheid en lojaliteit wat geskep is deur Kroonprins Moulay Hassan is nie maklik te voorsien in Engeland of Suid-Afrika nie, soos dit ook die geval is met die spontane wêreldwye hulp in Agadir, veral deur Franse, Italiaanse, Duitse, en Amerikaanse soldate, insluitende hul geneeskundige eenhede. Ontstellende gedagtes van hierdie aard word ondersteun deur die pleidooi van Prof. Ian Aird wat glo dat hulp in 'n nasionale natuurramp beperk behoort te word tot burgerlike persone wat verbonde is aan die Internasionale Rooikruis-organisasie, en geen ander hoegenaamd nie.[4] Gelukkig is daar ten minste die spesiale komitee van die Internasionale Federasie van Chirurgiese Kolleges en Akademies wat tans hierdie probleem van spoedige hulp ten tyde van nasionale rampe bestudeer.

1. Crosnier, J. *et al.* (1960): J. Urol. méd. chir., **66**, 636.
2. Mirouze, J., Faure, H. en Teilhaud, G. (1960): Presse méd., **55**, 721.
3. Fordham, J. H. (1960): Postgrad. Med. J., **36**, 652.
4. *The Times* (London): 8 August 1960.

THE PYLORIC ANTRUM

It was Edkins[1] who in 1906 first postulated that the antrum of the stomach might elaborate a hormone which mediates the gastric phase of gastric secretion. He was impressed with the distinctive histological structure of the pyloric antrum, and as a result of various experiments (including the intravenous injection of extracts and the placing of food in the antrum), he considered there might be a hormonal mechanism involved. He coined the word gastrin to indicate the antral hormone.

Subsequent work cast doubt on this hypothesis. Conflicting results were obtained in animal investigations by different workers. It became apparent that histamine was present in all the extracts used, but in recent times histamine-free extracts with definite gastric secretory activity have been prepared. Studies in dogs have shown[2] that resection of the gastric antrum causes a marked decrease in the secretion of acid gastric juice when antrum resection has been carried out and when Pavlov and Heidenhain pouches have been made. This shows that the secretion of acid juice in the isolated pouches is in some way related to the presence of an antrum. Further experiments showed that, with complete interruption of the extrinsic nerve supply to the antrum and separation of vascular and neuromuscular connections to the rest of the stomach, the antrum stimulates the body of the stomach to

secrete acid gastric juice when it (the antrum) is exposed to the contents of the duodenum. Edkins' original hypothesis was in this way confirmed.

Marked increase in gastric secretion was observed after transplantation of the antrum into the colon. With this technique it was demonstrated that chronic peptic ulcers can be induced in dogs. This strengthens the view that excessive acid secretion, induced by a normal physiological stimulus (gastrin), will cause peptic ulcer.

There is also the hypothesis that acid plays an important regulatory rôle in inhibiting the hormonal mechanism of the antrum. In some way or other acid-secreting tissue inhibits the mechanism. Other physiological processes may also be involved, and although many different approaches have been made to this problem, the complete answer is not yet available. Mechanical factors may be important in the gastrin mechanism.[3] Stimulation of the isolated antrum with an inflated balloon and perfusion with an alkaline solution will cause prompt secretion of highly acid gastric juice;[4] acid will block the mechanical stimulus. Unlike a chemical stimulus, mechanical stimulation produces a constant increase in motility of the antrum. Small doses of an anticholinergic drug block the increase in motility and also the release of gastrin. Incidentally, the cytological origin of gastrin is not known. The intramural nervous tissue of the antrum would appear to be important in the endocrine mechanism.

Much work has been done to elucidate the inhibitory effect of acid on the production of gastrin. There would appear to be some local chemical interference with the formation or release of gastrin. There are studies which suggest that an antisecretory hormone may be produced by the antrum. The evidence for this has been both indirect[5] and direct.[6] Other evidence is against the concept that the antrum produces an antisecretory hormone.[7] The information which is available at present does not provide a clear answer on whether the antrum does produce an antisecretory hormone.

In summary, the pyloric antrum functions as an endocrine organ. It secretes no hydrochloric acid, but it produces gastrin which mediates the gastric or antral phase of gastric secretion. The exact nature of the chemical stimulus which releases gastrin is not known. Mechanical stimulation also releases gastrin. The formation of gastrin is inhibited by acid, thus establishing a homeostatic mechanism which terminates the gastric phase of gastric secretion. Acid also inhibits other gastric secretory stimuli. The cell origin of gastrin is unknown. Intramural nerve plexuses appear to play a rôle in the release of gastrin. Fuller details of this important physiological mechanism, which has practical implications, are given by Woodward and Dragstedt.[8]

1. Edkins, J. S. (1906): J. Physiol., **34**, 133.
2. Woodward, E. R. *et al.* (1950): Amer. J. Physiol., **162**, 99.
3. Grossman, M. I. *et al.* (1948): *Ibid.*, **153**, 1.
4. Woodward, E. R. *et al.* (1957): Gastroenterology, **32**, 868.
5. Harrison, R. C. *et al.* (1956): Ann. Surg., **144**, 441.
6. Jordan, P. H. and Sand, B. F. (1957): Proc. Soc. Exp. Biol. (N.Y.), **94**, 471.
7. Longhi, E. H. *et al.* (1957): Amer. J. Physiol., **191**, 64.
8. Woodward, E. R. and Dragstedt, L. R. (1960): Physiol. Rev., **40**, 490.