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VAN DIE REDAKSIE : EDITORIAL

DIE REGULERING VAN ASEMHALING

Minder as 'n jaar na die vorige simposium wat ter herdenking van J. S. Haldane gehou is,[1] is weer 'n simposium oor die regulering van asemhaling gehou. Waar die eerste simposium veral gerig was op die chemiese kontrole van asemhaling, was die tweede meer toegespits op die neurologiese kontrole-meganismes. Trouens, in die woorde van W. O. Fenn,[2] kan asemhaling nie langer as 'n blote refleksmeganisme beskou word nie, maar eerder as 'n gedragsprobleem. Ritmiese asemhaling is so natuurlik soos die ritmiese beweging van arms en bene tydens stap. Die omvang van ventilasie word in 'n ommesientjie reggestel volgens die liggaamsbehoeftes. Een van die veelvuldige afferente bane moet CO_2-produksie of algemene metaboliese spoed toets om 'n aanpassing teweeg te bring.

Onder normale omstandighede bly die CO_2-spanning merkwaardig konstant. Die aanpassing van asemhaling, sover dit CO_2 betref, word deur Fenn[2] met 'n elektries-verwarmde kombers vergelyk. As mens koud kry, stel jy die temperatuur hoër, en as jy na 'n rukkie te warm word, stel jy dit laer. As jy die eerste maal reg geraai het, ondervind jy geen abnormale sensasie nie en verdere verstelling is nie nodig nie. Hierdie konsep word gesteun deur die werk van Loeschke en Gertz,[3] wat geen genoegsame stimulasie vir ventilasie by rustende omstandighede vind nie— nie CO_2, lae O_2 of pH nie.

By die aanvang van oefening verhoog ventilasie dadelik min of meer volgens die ‚geskatte' graad van oefening, en slegs as hierdie skatting foutief blyk te wees, stel boodskappe vanaf CO_2-, O_2-, en pH-reseptore die ventilasie reg.[2]

Hierdie konsep is klaarblyklik oorvereenvoudig, en 'n mens kom tot die gevolgtrekking dat die endogene CO_2 (of metaboliese CO_2) anders gehanteer word as die eksogene (of ingeasemde) CO_2. Dit skyn die enigste logiese verklaring te wees vir die feit dat alveolêre CO_2 merkwaardig konstant bly by rus en 'n groot spektrum van spieraktiwiteit. Andersyds, as CO_2 ingeasem word, neem ventilasie met 1·5 liter toe vir elke 1 mm.Hg toename in alveolêre pCO_2, en keer eers weer na normaal terug as die alveolêre pCO_2 normaal is.[2]

In the eerste sewe referate van die simposium is die fisiologie van neurone ten opsigte van die regulasiemeganismes bespreek. Dit lewer bewyse dat neuronale metabolisme nóu saamhang met die eienskappe van die selmembraan en dat hierdie faktore sy funksionele aktiwiteit bepaal.[4] Die funksionele organisasie van neurone is die resultaat van eksitasie en inhibisie op verskillende dele van die neuronale bevolking, en hulle is weer sinapties verbonde aan die retikulêre projeksiebane wat die wisseling in die wakende-slapende asemhalingsiklus beïnvloed.[5]

Vervolgens word die sentrale faktore in neurale regulasie in agt referate oorweeg en die kontroversiële aspekte van die koördinasiemeganismes in die breinstam uitgepluis;[6] en daarna toon Redgate[7] dat die hipotalamus, by onderdrukking van sy funksie, die Hering-Breuer refleksinhibisie bevorder; ook dat daar ander gevolge is wat bes verklaar kan word deur te aanvaar dat die hipotalamus verbind is met neurone in die medullêre retikulêre formasie wat inspirasie sal bevorder. Hierdie afdeling sluit af met 'n stimulerende referaat deur Barchilon,[8] professor in die psigiatrie aan die Albert Einstein Kollege, Nieu-York, oor die ‚Emosies en respirasies'. Vir die respiratoriese fisioloog is die psigoanalitiese, en dikwels Freudiaanse, benadering tot hiperventilasie iets nuuts, maar dit is stimulerend vanweë die klinies-bekende verskynsels.

Almal sal sekerlik nie saamstem met die patogenese van asma soos in 'n geval-beskrywing hier uiteengesit nie,[8] maar almal sal kan getuig van 'n sterk psigiese faktor by die meeste asmalyers. Ten slotte is sy verklaring van sterftes-sonder-oorsaak, byvoorbeeld van die persoon wat oënskynlik gesond is, maar wat hom gereed maak om te sterwe en dit dan doen, na ons wete, die aanneemlikste tot dusver.

In die laaste aantal afdelings van die simposium word die perifere kontrolemeganismes bespreek, asook die meer bekende gebied van die humorale meganismes, en ten slotte word 'n integrale benadering gebruik om bekende regulasiepatrone te omskryf.

Na ons mening is hierdie simposium, so kort na die vorige, tog tydig, aangesien chemiese kontrole van respirasie en neurologiese kontrole aanvullende aspekte dek— en hiervan is die laasgenoemde meganisme miskien tot dusver die mees verwaarloosde.

1. Cunningham, D. J. C. en Lloyd, B. B., reds. (1963): *The Regulation of Human Respiration*. Oxford: Blackwell.
2. Fenn, W. O. (1963): Ann. N. Y. Acad. Sci., **109**, 415.
3. Loeschke, H. H. en Gertz, K. H. (1958): Arch. ges. Physiol., **276**, 460.
4. Quastel, J. H. (1963): Ann. N. Y. Acad. Sci., **109**, 436.
5. Purpura, D. P. (1963): *Ibid.*, **109**, 505.
6. Wang, S. C. en Ngai, S. H. (1963): *Ibid.*, **109**, 550.
7. Redgate, E. S. (1963): *Ibid.*, **109**, 606.
8. Barchilon, J. (1963): *Ibid.*, **109**, 619.

THE URAEMIC SYNDROME

Uraemia is a complex of symptoms and signs which result from failure of renal regulation of the composition and the volume of the body fluids.[1] The increase in urea concentration is quantitatively the most striking abnormality of the body fluids in uraemia, but it is certainly not the most important. Urea can be administered to normal subjects to produce blood levels comparable to those in uraemic patients and will cause thirst and polyuria, but none of the other manifestations of uraemia. In uraemic patients undergoing haemodialysis for the treatment of the condition, the blood urea can be kept unchanged by the addition of urea to the dialysis bath; even though the

blood urea is maintained at a high level, relief of the signs and symptoms occurs.

Much more important are the development of hyperkalaemia, elevation of plasma phosphate and sulphate, decrease of plasma bicarbonate and sodium, and the retention of toxic products of protein catabolism that may be responsible for some of the central nervous manifestations of uraemia. Potassium intoxication is an important lethal factor in acute renal failure and in the terminal stages of chronic renal disease. The intoxication is hastened by extensive trauma, severe infections, and haemorrhage into the alimentary canal and body cavities or tissues. The heart is especially affected by hyperkalaemia; impulse formation, conduction, and contractility are affected, and electrocardiographic changes are demonstrable that may be pathognomonic (high peaked T-waves, broad QRS complexes, conduction defects). Central nervous symptoms and muscle weakness are also associated with the hyperkalaemia.

Diminished glomerular filtration and failure of regulation of acid-base balance by the kidneys cause other changes. A reduction in filtration rate results in an increase in phosphate and sulphate in the blood plasma. Muscular twitching, and less commonly tetany, develop as highplasma-phosphate depresses the solubility and ionized fraction of calcium, which is to some extent counteracted by the development of acidosis. It can thus happen that in correcting acidosis by alkali therapy, tetany may occur if calcium is not administered at the same time. Osteomalacia may be caused by the acidosis, but also to some extent by parathyroid overactivity. Acidosis also increases loss of potassium from cells, so that hyperkalaemia develops. In chronic renal disease acidosis is common in the uraemic stage and it develops slowly and insidiously.

The excessive administration of water in acute renal failure causes the development of hyponatraemia, and strict restriction of salt intake may be the cause of chronic renal failure. Restriction of sodium intake to correct the associated hypertension may deplete the body stores of sodium and cause dehydration. Renal excretory and regulatory functions become further decreased, and uraemia is increased; water is retained to restore the volume of the body fluids, and hyponatraemia results. The effects of hyperkalaemia are intensified by hyponatraemia and by hypocalcaemia, and the converse also applies.

Much has yet to be learned about the cause of many of the symptoms and signs of uraemia.[1] General metabolic disturbances probably account for most of the manifestations. Some of the abnormalities in the body fluids that are significant have been briefly indicated, but the control mechanisms of erythropoeiesis, blood pressure, and volume of the body fluids are also disturbed. Treatment of acute or chronic renal failure is directed first to factors that can be promptly corrected, such as obstruction, infection, circulatory insufficiency, and the action of toxic substances. Further measures are aimed at reducing the excretory load, utilization of extrarenal routes of excretion, and compensation for regulatory inadequacies. The basic mechanisms of renal function, the consequences of renal diseases, and the principles involved in therapy are clearly and authoritatively described by Pitts in an attractive volume that has recently become available.[1]

1. Pitts, R. F. (1963): *Physiology of the Kidney and Body Fluids*. Chicago: Year Book Medical Publishers.