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KOORS—VRIEND OF VYAND?

As ons koors gedefinieer het as 'n verhoogde sentrale liggaamstemperatuur as gevolg van 'n versteurde termotaktiese meganisme, kan ons die oorsake van so 'n versteuring in vier breë groepe verdeel: (1) Skade aan die sentrale senuweestelsel, gewoonlik die gevolg van intraserebrale bloeding, (2) infeksies en infestasies, van virusse af tot wurms, (3) weefselbekadiging, bv. infarksies, maligne tumore, en jig, en (4) onsekere oorsake, bv. dié wat volg op immuniteitsreaksies, bv. lupus eritematose en rumatiekkoors, en na bloeding van die spysverteringskanaal. Hierdie vier groepe kan die termotaktiese meganisme omverwerp deur middel van (*a*) bakteriële produkte, (*b*) liggaamsel-produkte.

Bogenoemde paragraaf som dan verskeie velde op waar die navorser en sy laboratorium in staat was om in te gaan op die fyner meganisme van temperatuur-regulasie, die isolering van 'n bakteriële endotoksien uit gramnegatiewe organismes, die identifikasie daarvan as 'n polisakkaried, en endogene pirogeen afkomstig, waarskynlik, maar nie definitief nie, van die polimorfe leukosiet.[1,2]

Sydenham, die groot klinikus uit die 17e eeu, het die teleologiese gesegde: ‚Fever is a mighty engine which Nature brings into the world for the conquest of her enemies' in gebruik gebring.[3] Sedertdien het die klinikus antibiotiese middels gevind, antipiretiese middels (salisilate, steroïdes), verfynde termometers, röntgendiagnostiek, en baie biochemiese bepalings om sy terapie te lei. Die bakterioloog kan hierdie terapie doelgerig instel. Ons betrag nog koorskaarte met 'n gewyde oorgawe, en voel verlig as die inklyne die bekende rooistreep op 98·4°F, na benede verbyskiet.

Dubois[4] het by geleentheid verklaar: ‚Fever is merely a symptom and we are not sure that it is an enemy. It may be a friend'.[4] Hierdie vraag het vele werkers besig gehou. Die antwoorde wat ons kry is dubbelsinnig, dikwels teenstrydig. Die wyse waarop die vraag gestel word, bepaal die vorm van die antwoord. Ons weet byvoorbeeld dat daar tydens koors 'n verhoogde kardiale uitwerping is met 'n daling in bloeddruk as gevolg van vatverwyding in die splanchniese en renale bloedvat-bed.[2] As dié vraag gestel sou word in gevalle met kardiale inkorting, is die antwoord dat koors hier 'n vyand is, want dit is dikwels genoegsaam om so 'n hart in versaking te stort. Antipiretiese middels help ook nie hier nie, want die bloedvatveranderings geskied nogtans.[2] Andersyds mag koors 'n vriend wees in soverre dat dit moontlik 'n faktor mag wees in die hoë voorkoms van spontane remissies in sifilis,[5] en dit is selfs voorgestel dat neurosifilis en ander tersiëre stigmata voorkom in die ongelukkige indiwidue wat tydens die latente fase min tussenkomende koorssiektes gehad het![6]

Klaarblyklik moet ons dus ons toetsvraag noukeurig formuleer, want die teleologiese doelstelling van koors is tog seker gemik om 'n uitwerking te hê in 'n andersins gesonde persoon. Andersyds is daar 'n groot aantal veranderings in die liggaam tydens koors, waaronder veranderings in koolhidraat-metabolisme, plasma proteïen, ysterabsorpsie, vitamien-benodighede, lewerfunksie-proewe, elektroliet-balans, doepa-sensitiwiteit, en die reeds vermelde bloedvat-veranderings.[6] Daar is veranderings in die leukosiete en moontlik in hulle fagositiese vermoë. In ons huidige stand van kennis is dit dikwels onmoontlik om te besluit welke van hierdie veranderings die direkte gevolg of bloot sekondêre aanpassings is by 'n verhoogde liggaamstemperatuur.

In 'n baie onlangse oorsig oor die rol van koors as 'n faktor in die weerstand van die pasiënt teen infeksie,[6] word daarop gewys dat koors òf 'n effek op mikroorganismes en hul produkte kan hê, òf deur 'n wysiging van verskeie sellulêre en humorale meganismes in die pasiënt self werk. *In vitro* studies op organismes kan skynbaar nie gebruik word om verskynsels *in vivo* te verklaar nie. As afkoeling 'n sekere effek op die siekteverloop van 'n koorsige pasiënt het, kan ons nie aanneem dat koors die teenoorgestelde effek sou hê nie.

Verhoogde temperatuur beskerm tot 'n mate teen anafilaktiese skok in marmotte.

Koors verhoog die vatbaarheid vir infeksie met herpes simplex virus. Finland[6] beweer egter dat herpes labialis, wat so volop in pneumokokkale en streptokokkale pneumonieë is, uiters seldsaam, indien ooit, gevind word in stafilokokkale pneumonie. Hierdie kliniese waarneming bring ons weer by die eerste vraag uit: Aktiveer koors die virus, of word weerstand verlaag deur 'n ander faktor wat koors vergesel en net in sommige gevalle teenwoordig en in ander afwesig is? Of word 'n faktor geproduseer wat die pasiënt beskerm teen die virus in dié koorstoestande wat nie gepaard gaan met koorssere nie?

Dat koors op die een of ander wyse tot voordeel mag strek, is deur die eeue heen versigtig aangeneem en het soms die status van dogma verkry.[6]

In die oorsig waarna reeds herhaaldelik verwys is,[6] word die volgende voorbeeld van die voortplanting van gevathede en aforismes omtrent hierdie vraag gegee: In 'n uitstekende artikel waarsku 'n kinderarts tot versigtigheid by die voorskryf van antipiretiese middels. Hy grond sy stelling op 'n versigtige argument, maar sluit af met die aanhaling van Dubois se reeds genoemde aforisme.[4] Kort daarna word die artikel in 'n weekblad opgesom onder die titel ‚Friendly fever' en 'n tydjie daarna verskyn 'n artikel deur 'n geneesheer in 'n maandblad ‚Fever, the heat that heals'.

Is dit nie so dikwels waar dat ons nie glo wat ons bewys nie, maar liewers ‚bewys' wat ons glo? Een ding is seker — ons het geen antwoord op die vraag of koors ons vriend of vyand is nie. Met die ontwikkeling van antibioties-weerstandige organismes en die isolasie van gesuiwerde pirogene waarmee ons koors kan verwek sonder infeksie, wil dit vir ons voorkom of 'n antwoord op hierdie eeue-oue vraag dalk binnekort nodig mag wees.

1. Pickering, G. W. (1958): Lancet, **1**, 59.
2. Atkins, E. (1960): Physiol. Rev. **40**, 580.
3. De Kruif, P. en Simpson, W. M. (1940): J. Lab. Clin. Med., **26**, 125.
4. Dubois, E. F. (1946): *Fever and the Regulation of Body temperature*. Springfield: Charles C. Thomas.
5. Stokes, J. H., Beerman, H. en Ingraham, N. R. (1945): *Modern Clinical Syphilology*, 3e ed. Philadelphia: Saunders.
6. Bennett, J. L. en Nicastri, A. (1960): Bact. Rev., **24**, 16.

THE CONQUEST OF MALARIA

Unique and sensational advances have been made during recent decades in our knowledge of the diseases of warm climates. Among these malaria probably occupies the first place as one of the scourges of mankind, a disease which has affected the course of history itself. The struggle against this widespread disease has taken place on many fronts, and the names of the pioneers who contributed to the knowledge and treatment of malaria will always be remembered. The student of medical history will know that a great deal has been written on this subject. For a balanced and fair presentation, the account by Jaramillo-Arango[1] should, however, be consulted. His scientific record has also considerable literary merit.

The mosquito is the most deadly of enemies in tropical countries. In the course of days or weeks it can spread death to thousands of human beings. It transmits a number of tropical diseases, including malaria and yellow fever. The victims of malaria throughout the world have been calculated at three million a year. There is evidence that malaria was known in the ancient world, and it was thought that it probably contributed in some degree to the fall of the Greek and Roman Empires. It is one of the oldest diseases known to man.

Until the beginning of this century the precise cause of malaria was not established and its mode of spread was one of the intricate problems confronting medical science. In 1880 Alphonse Laveran discovered the haematozoon, but it was only in 1898 that Ronald Ross definitely established that the parasite is transmitted through the mosquito. For centuries there had been suspicion that malaria and other diseases were transmitted by insects. The connection between mosquitoes and this disease was demonstrated by Patrick Manson in his studies on elephantiasis due to filaria organisms, and suggested by Carlos Finlay in his considerations of the transmission of yellow fever.

Space does not permit a full discussion of the details of all the information on which the mosquito-malaria theory came to be based. The demonstration of its truth, urged by Manson, was undertaken with brilliant success by Ronald Ross. This eminent investigator was a poet, writer, philosopher, and mathematician. When on furlough in London from the Indian Medical Service, he was shown the parasite of malaria by Manson and was so intrigued that he was determined to prove the theory of its propagation. Three years later, in 1897 in Calcutta, he discovered in the stomach-wall of a species of mosquito the öocysts of the sexual cycle of the parasite. After this momentous discovery Ross spent the evening composing a poem of thanksgiving. This poem was subsequently printed and published with his other poems.

Further work on the haematozoon of birds enabled Ross to demonstrate the equation mosquito: malaria in all its phases. Manson was in constant correspondence with Ross, advising and encouraging him, and it was he who announced the great news to the scientific world at the meeting of the British Medical Association in Edinburgh in 1898. Two distinguished Italian scientists, Grassi and Bignami, then made the practical demonstration of the malaria-mosquito postulate in man. In Rome, in 1898-1899, they exposed four persons to the bite of mosquitoes incubated from larvae and fed only on malarial blood, and all four contracted the disease. Grassi also had the distinction of showing that it is only the genus *Anopheles* that transmits the disease, and only the female of the species that has the power to infect. The male does not bite the human being. To counter all possible arguments against these newly acquired concepts, Manson decided to demonstrate the experiment in London — a city free from any suspicion of malaria. Infected mosquitoes were sent from Rome and two volunteers, his son and his assistant, both contracted malaria after being bitten by these mosquitoes. The question was solved and all doubts were removed.

There followed a campaign of direct mosquito control which aimed at depriving the mosquito of its breeding places and destroying the larvae wherever the female has succeeded in laying her eggs. Drainage and reclamation of swamps and marshes, the spraying of standing water, and the protection of individuals against mosquitoes have been important methods used. The discovery of insecticides such as dicophane (DDT), gamma benzene hexachloride and others has enhanced the scope of these activities. New antimalarial drugs are nowadays available to give protection to visitors to a malaria zone, and radical cure of infection can be effected easily and economically. Dr. Jaramillo-Arango's account of the mosquito-malaria theory, the history of cinchona, and the progress achieved in the knowledge and treatment of malaria, should be read by everybody who is interested in this fascinating problem.

1. Jaramillo-Arango, J. (1950): *The Conquest of Malaria*. London: William Heinemann