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VAN DIE REDAKSIE : EDITORIAL

VERGIFTIGING DEUR INSEKDODENDE MIDDELS

Daar is al herhaalde kere in hierdie *Tydskrif* gewys op die gevare van insekdodende en peswerende middels wat in die huis gebruik word sowel as deur die gesondheidsadministrasie, die landbou, die nywerhede, en die handel.[1-4] Baie internasionale besprekings oor vergiftiging met hierdie middels het al in verskillende lande plaasgevind, byvoorbeeld in Rome in 1953 onder beskerming van die W.G.O.

Gedurende Oktober het daar ook in hierdie land so 'n bespreking plaasgevind by geleentheid van die Jaarlikse Kongres van die Gesondheidsbeamptesvereniging van Suid-Afrika. Elders in hierdie uitgawe plaas ons 'n artikel deur prof. N. Sapeika[5] wat handel oor aspekte van die moderne insekdodende middels, hulle giftigheid, en beheer. Die algemene gebruik van hierdie gifsoorte maak dit noodsaaklik dat geneeshere en mediese studente op die hoogte van sake sal bly oor die gevare en die metodes van voorkoming en behandeling van vergiftiging van hierdie aard. Die komitee van die Amerikaanse Mediese Vereniging wat die probleem van peswerende middels ondersoek het, beveel aan dat hierdie middels en verwante produkte behandel moet word in die farmakologiekursusse van mediese skole.[6] Hierdie aanbeveling word gemaak ten spyte van die ongeëwenaarde aandrang op insluiting van nuwe onderwerpe in die farmakologiekursusse van die mediese leerplan.

Daar is 'n groot aantal chemiese stowwe wat as peswerende middels gebruik word en hul giftigheid verskil baie. Onder die middels wat op die oomblik taamlik algemeen gebruik word, is die chloor-(halogeen-) koolwaterstowwe en die organiese fosfaatesters. Die organiese fosfaatesters is veral baie giftig.

Die kliniese tekens van vroeë vergiftiging deur hierdie samestellings is nie spesifiek nie en dit mag moeilik wees om hulle te onderskei van gewone siektes. Die latere tekens het egter kenmerkende eienskappe. In sommige gevalle is daar sluimerende vergiftiging wat noukeurige mediese waarneming van die slagoffer nodig maak nog lank nadat hy oorspronklik aan die vergiftiging blootgestel was. In die geval van vergiftiging met dieldrin, byvoorbeeld, moet die pasiënt nog ten minste twee jaar lank na die oorspronklike behandeling en verwydering van die blootstelling, medies ondersoek word. Na vergiftiging met parathion moet die pasiënt ook lank onder observasie gehou word en bepalings van die terugkeer van cholienesterase-aktiwiteit in die serum tot die normale moet gemaak word—indien moontlik verskeie maande lank.

Die volgende interessante nuwe feite het as gevolg van navorsing gedurende die afgelope paar jaar aan die lig gekom. In verband met dikofaan (DDT), byvoorbeeld, is dit gevind dat herhaalde dosisse wat deur vrywilligers per mond geneem is oor 'n tydperk wat geduur het tot 18 maande, en teen ongeveer 200 keer die daaglikse sterkte waarin hierdie middel gewoonlik in voedsel voorkom, nie siekte veroorsaak nie. Dit dui op 'n groot veiligheidsfaktor ten opsigte van die inneem van DDT per mond. Die absorbering van groot hoeveelhede kan egter akute vergiftiging veroorsaak.

Met betrekking tot gamma bensien-hexachloried (BHC, gammexane) kan sensitiwiteit en gestelsreaksies ontstaan. Dieldrin, wat baie gebruik word in malaria-uitwissingsveldtogte, is 'n gevaarlike samestelling. Dit word deur die vel geabsorbeer selfs in die droë vorm. In hierdie opsig verskil dit dus aansienlik van DDT. 'n ‚Onwaarneembare' sweefmengsel oor die gesig, voorarms en ander velgebiede wat nie bedek is nie, is genoeg om vergiftiging te veroorsaak.[7] Ernstige siektetoestande soos epileptivorme aanvalle wat herhaaldelik kan voorkom en na vertraging kan ontstaan, en geestesversteurings, kan veroorsaak word. Dit is dringend noodsaaklik dat dieldrin verder bestudeer moet word, veral met die oog op berging in die liggaam, bepaling van die vlak daarvan in die bloed en in die urine, en die ontdekking van vroeë tekens van vergiftiging.

Wat betref die organiese fosfaatesters, soos byvoorbeeld, parathion,[4] kan ons meld dat daar heelwat navorsingswerk gedoen word, veral met betrekking tot die uitwerking daarvan op cholienesterase en die ontwikkeling van 'n siekteteenmiddel om die ensiem weer aktief te maak nadat dit onaktief geword het. In hierdie verband is wel al vordering gemaak deur die gebruik van piridien-2-aldoksien metiodied wat die muskarienagtige sowel as die nikotienagtige uitwerking van parathion teenwerk.[8,9] Atropien word in hierdie geval gelyktydig toegedien om die opeenhoping van asetielcholien op sekere plekke in die liggaam teen te werk. Parathion is baie giftig—'n enkel druppel in die oog kan noodlottige gevolge hê.[10] Hierdie gif word al meer gebruik as 'n middel om selfmoord mee te pleeg, soos byvoorbeeld gerapporteer in Finland en in Denemarke.[10] Dit is een van die giftigste middels wat gebruik word om pestoestande te beheer.

Gesien uit die chemiese, farmakologiese, kliniese en administratiewe aspekte, sowel as uit die oogpunt van moontlike vergiftiging deur hierdie middels, is daar baie interessante fasette verbonde aan die studie van kiemdodende en peswerende middels.

1. Aantekening (1952): S. Afr. T. Geneesk., **26**, 684.
2. Van die Redaksie (1953): *Ibid.*, **27**, 1149.
3. *Idem* (1954): *Ibid.*, **28**, 389.
4. Klugman, H. B. (1959): *Ibid.*, **33**, 899.
5. Sapeika, N. (1959): *Ibid.*, **33**, 1063.
6. Aantekening (1953): J. Amer. Med. Assoc., **152**, 709.
7. Hayes, W. J. (1959): Bull. Wld Hlth Org., **20**, 891.
8. Steyn, D. G. (1958): S. Afr. T. Geneesk., **32**, 894.
9. Aantekening (1959): Lancet, **2**, 166.
10. Toivonen, T. *et al.* (1959): *Ibid.*, **2**, 175.

INSECTICIDE POISONING

Attention has been drawn on a number of occasions in the *Journal* to the dangers associated with the use of insecticides and other pesticides used in the home, in health administration, agriculture, industry, and commerce.[1-4] Many discussions on poisoning with these agents have taken place in different countries and also at an international level, as for instance in Rome in 1953 under the suspices of the WHO.

The latest discussion in this country took place in Cape Town recently at the Annual Congress of the Health Officials' Association of Southern Africa. Elsewhere in this issue we publish an article by Prof. N. Sapeika[5] in which some details are given of modern insecticides and their toxicity and control. The widespread use of these poisons has made it necessary that physicians and medical students should be adequately informed about their dangers and the methods used for the prevention and treatment of insecticide poisoning. The Committee on Pesticides of the American Medical Association has urged the introduction of a study of pesticides and related products into the pharmacology courses of medical schools.[6] This is being done notwithstanding the unprecedented demand for including additional subjects in pharmacology in the medical curriculum.

A great variety of chemical agents have been used as pesticides, and their toxicity varies a good deal. Among those widely used at present are the chlorinated (halogenated) hydrocarbons and the organic phosphate esters, the latter being particularly toxic.

The clinical signs of early poisoning by these compounds are not specific and may be difficult to distinguish from the signs of an ordinary illness. The late signs have characteristic features. In certain instances there is a latent intoxication which necessitates close medical observation of the victim of poisoning for a long time after the original exposure. For example, in dieldrin poisoning the patient should be examined medically for at least two years after the initial treatment and removal from exposure. Also, after parathion poisoning prolonged observation and estimations of the return of serum cholinesterase activity to normal should be made, possibly for several months.

Some interesting newer facts that have emerged from studies in recent years may briefly be cited here. In experiments with dicophane (DDT), for instance, it has been found that repeated oral doses taken by volunteers for periods up to 18 months, at about 200 times the daily rate at which a man might receive this insecticide in his diet, does not produce illness, indicating a large safety factor as far as this mode of entry is concerned. Acute poisoning from the absorption of large amounts of DDT can, of course, produce serious symptoms.

With regard to gamma benzene hexachloride (BHC, gammexane), sensitivity and systemic effects can be acquired. Dieldrin, which has been much used, for example in malaira campaigns, is a dangerous compound. It is absorbed through the skin, even in the dry form, in this respect differing considerably from dicophane. An 'imperceptible' mist of the suspension on the unprotected face, lower arms, and other skin areas, is sufficient to cause poisoning.[7] Serious illness may be produced, including epileptiform seizures, which may be recurrent and delayed in onset, and mental disorder. There is urgent need for further study of dieldrin, its storage in the body, its estimation in blood and urine, and the detection of early signs of intoxiaction.

In the organic phosphate esters, for example parathion, much research work has been in progress especially with regard to its action on cholinesterase, and the development of a suitable antidote to reactivate the enzyme when it has been inactivated. In this connection an advance has been made with the introduction of pyridine-2-aldoxime methiodide which counteracts the muscarinic as well as the nicotinic effects of parathion,[8,9] atropine being given concurrently to counteract the accumulation of acetylcholine at certain sites in the body. Parathion is a very dangerous poison; a single drop in the eye may have a fatal effect.[10] It has been used to an increasing extent as a means of suicide, for example in Finland and Denmark.[10] It is one of the most toxic agents used for pest control.

There is much of interest and of practical importance in the field of insecticide poisoning from the chemical, pharmacological, toxicological, clinical, and administrative points of view.

1. Annotation (1952): S. Afr. Med. J., **26**, 684.
2. Editorial (1953): *Ibid.*, **27**, 1149.
3. *Idem* (1954): *Ibid.*, **28**, 389.
4. Klugman, H. B. (1959): *Ibid.*, **33**, 899.
5. Sapeika, N. (1959): *Ibid.*, **33**, 1063.
6. Annotation (1953): J. Amer. Med. Assoc., **152**, 709.
7. Hayes, W. J. (1959): Bull. Wld Hlth Org., **20**, 891.
8. Steyn, D. G. (1958): S. Afr. Med. J., **32**, 894.
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