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EDITORIAL

CHLORDANE POISONING

The Council on Pharmacy and Chemistry of the American Medical Association performs many useful services for the medical and allied professions. Through its committee on pesticides information is made available from time to time on insecticidal agents of immediate medical interest. A recent addition to the series of reports of this nature is one dealing with chlordane,[1,2] a domestic and agricultural insecticide widely used in America.

The insecticidal properties of this chlorinated hydrocarbon were discovered in the United States and in Germany about the same time, but it was in America that chlordane was first made available—shortly after World War II. The compound is a heavy, dark brown, oily liquid soluble in the common organic solvents. The commercial product contains an admixture of 25-40% of related compounds consisting of isomers that have been separated by chromatography, and other substances of different chemical nature; these additional compounds contribute in some degree to the insecticidal action of the final product and also to its harmful effects.

The lethal action is achieved by contact, ingestion or exposure to the vapour of this viscous pungent agent. In man absorption can take place through the skin, the gastro-intestinal tract, or the respiratory tract; rapid penetration can occur, and the higher motor cortex and the cerebellum are then affected. The symptoms of acute chlordane poisoning are similar to those occurring with dicophane (chlorophenothane; DDT) and other chlorinated hydrocarbon insecticides. Irritability, salivation, dyspnoea, tremors and convulsions, followed by death, are the features that have been observed experimentally in animals, and also in human beings as the result of carelessness in handling the commercial mixture or from accidental ingestion or inhalation of chlordane. Other symptoms that have been reported in these cases are nausea, vomiting, diarrhoea, abdominal pain, cough, blurred vision, ataxia, confusion, delirium and mania.

Acute signs of poisoning may appear as early as

VAN DIE REDAKSIE

CHLOORDAAN-VERGIFTIGING

Die Raad vir Farmasie en Chemie van die American Medical Association lewer waardevolle dienste aan mediese en verwante professies. Die Raad se komitee vir plaagbestryding stel van tyd tot tyd verslae oor insekdodende middels beskikbaar wat van onmiddellike belang vir die mediese professie, is. Onlangs het 'n verslag verskyn oor chloordaan[1,2] 'n insektedoder wat algemeen in Amerika in die huis en op plase gebruik word.

Die insekdodende eienskappe van hierdie chloorkoolwaterstof is amper gelyktydig in die Verenigde State en Duitsland ontdek maar dit was in Amerika dat chloordaan die eerste keer beskikbaar gestel is—kort na Wêreldoorlog II. Dit is 'n swaar, donkerbruin, olierige vloeistof wat in gewone organiese oplosmiddels oplosbaar is. 25-40% van die handelsproduk is verwante verbindings (wat bestaan uit isomere wat chromatografies geskei is) en ander stowwe wat verskillend van chemiese aard is; hierdie addisionele mengsels verhoog tot 'n mate die insekdodende werking van die middel maar terselfdertyd ook die skadelike gevolge daarvan.

Kontak met, ingestie van of blootstelling aan die wasem van hierdie klewerige skerp middel kan noodlottig wees. Absorbering vind by die mens plaas deur die vel, in die maagdermkanaal en die lugweë; deurdringing kan snel geskied en dit tas dan die hoër motoriese skors en die cerebellum aan. Die simptome van akute chloordaanvergiftiging is soortgelyk as dié van dikofaanvergiftiging (kloorfenotaan, DDT) en ander chloorkoolwaterstof-insektedoders. Prikkelbaarheid, kwyling, kortasemigheid, bewing en stuiptrekkings gevolg deur die dood is kenmerke wat waargeneem is by eksperimente met diere en ook by die mens te wyte aan nalatigheid in die gebruik van die handelsartikel of as chloordaan per ongeluk ingeasem of ingeneem word. Ander simptome wat by hierdie gevalle aangetref word is mislikheid, vomering, diarree, maagpyn, hoes, dofsien, koördinasiesteuring, verwarring, en ylhoofdigheid.

Akute tekens van vergiftiging kan 45 minute na ingestie voorkom, en die dood mag binne 24 uur intree, maar dit kan ook daelank vertraag word na 'n enkel

45 minutes after ingestion, and death may occur within 24 hours but it may be delayed for many days after a single oral toxic dose. In fatal cases the dominant pathological signs that have been observed in human beings are generalized congestion, oedema, and signs of gastro-intestinal irritation.

In chronic chlordane poisoning symptoms indicate disturbances in the central nervous system and the optic nerve, while post-mortem examination has revealed degenerative cell-changes in the liver and kidneys.

A few deaths in human beings are already on record.[1] In one case a young woman died within a few minutes after spilling a suspension of chlordane on her dress; in another, death occurred 9½ days after suicidal ingestion of 6 g. of chlordane. Several instances of non-fatal poisoning have been noted. Contact dermatitis has occurred in a number of cases among exterminators and others handling this irritant poison.

In the treatment of poisoning the symptomatic measures used are the same as for other chlorinated hydrocarbon insecticides; removal of poison from the skin by *immediate* washing with soap and water or, if the chlordane has been swallowed, gastric lavage followed by a saline purgative. Milk and oils must be avoided. Barbiturates may be indicated when there is overstimulation of the central nervous system, but should be used with care.

The high toxicity of chlordane has militated against its use in the treatment of pediculosis and scabies, as compared with dicophane (DDT) and gamma benzene hexachloride. In its use for the control of agricultural and household pests it is dangerous to persons exposed to it. Even when applied to selected areas care is required to avoid deposits on surfaces played on by children. The slow liberation of fumes when it is applied indoors constitutes a danger. Its use on food crops is only permissible at certain stages of their development.

1. The Present Status of Chlordane (1955): J. Amer. Med. Assoc., **158,** 1364.
2. Fatal Chlordane Poisoning (1955): *Ibid.*, **158,** 1367.

mondelingse toksiese dosis. In noodlottige gevalle by die mens is die uitstaande patologiese tekens algemene kongestie, edeem en tekens van maagdermirritasie.

Simptome by kroniese chloordaanvergiftiging dui op stoornisse in die sentrale senuweestelsel en die gesigsenuwee, terwyl lykskouings degeneratiewe selveranderings in die lewer en niere aantoon.

'n Paar sterfgevalle is alreeds noteer.[1] In een geval het 'n jong vrou binne 'n paar minute beswyk nadat sy 'n chloordaan suspensie op haar rok uitgestort het; in 'n geval van selfmoord het die dood ingetree 9½ dae na die ingesie van 6 g. chloordaan. Verskeie gevalle van vergiftiging sonder noodlottige gevolge is aangeteken. In 'n aantal gevalle het huidontsteking (as gevolg van kontak) voorgekom by uitroeiers wat met hierdie prikkelgif werk.

Die simptomatiese behandeling is dieselfde as in die geval van ander chloorkoolwaterstof-insektedoders; verwyder die gif van die vel deur *onmiddellik* met seep en water te was. As chloordaan ingesluk is moet die maag gespoel word en 'n soutpurgasie toegedien word. Melk en olies moet vermy word. In geval van oorstimulasie van die sentrale senuweestelsel kan barbiturate nuttig wees maar hul moet in oorleg en met sorg gebruik word.

Te wyte aan die hoë toksisiteit van chloordaan moet dit nie vir die behandeling van pedekulose en skurfte gebruik word nie en is dikofaan (DDT) en gamma benseenheksachloried verkieslik.

Die gebruik daarvan in die huis en op die plase stel diegene wat daaraan blootgestel word in gevaar. Selfs as dit net in uitgesoekte gebiede gebruik word moet voorsorg getref word om te verhoed dat dit neerslaan op plekke waar kinders mag speel. Die stadige vrystelling van dampe binnenshuis is gevaarlik. Dit moet slegs vir voedselgewasse op sekere stadia van hul groei gebruik word.

1. The Present Status of Chlordane (1955): J. Amer. Med. Assoc., **158,** 1364.
2. Fatal Chlordane Poisoning (1955): *Ibid.*, **158,** 1367.

'VANISHING TOES'

A *Lancet* annotation[1] makes reference to the case which Dr. D. M. Krikler[2] recently reported in the *South African Medical Journal* in an article entitled 'The Case of the Vanishing Toes'. The writer of the annotation agrees with Dr. Krikler that the case is probably an example of 'idiopathic osteolysis'. The patient (seen in January 1955) was a 35-year-old Coloured male, in whom, 5 years before, certain toes (the 1st and 4th of the right foot and the 4th and 5th of the left) had disappeared wholly, or almost wholly, in the course of a few months without any history of pain or trauma or exposure to cold.

On X-ray examination, all the phalangeal bones of the missing toes were seen to have disappeared, except a small piece of the proximal phalangeal bone of two of them; and the distal end of one of the metacarpals was also missing and there were signs of erosion of the distal phalanges of two of the remaining toes. The lesion consisted of erosion with no evidence of reaction. Further, there was fusion of the cuneiforms and synostosis of the 4th and 5th metatarsals on the right; the evidence suggested that these last conditions were congenital. X-ray revealed no other lesions in the rest of the skeletal system (apart from the absence of the right arm, which was missing from the level of the shoulder joint; the patient said that the loss had occurred in childhood, when he was mauled by a tiger in a circus); and the history and examination revealed nothing suggesting a cause for the loss of the toes. The patient was a 'steady' drinker and malnourished, with hepatomegaly and pellagrinous changes in the arms and the dorsa of the feet.

Gorlam *et al.*[3] were only able to find 16 cases of disappearing bones reported in the literature. They discount trauma as a possible cause, and state that biopsies indicate that a common feature is an over-

growth of thin-walled blood-vessels, and this haemangiomatosis may be an aetiological factor. Progress of the condition is slow and variable; it commonly stops after a time but occasionally ends fatally.

1. Annotation (1956): Lancet, **1**, 93.
2. Krikler, D. M. (1955): S. Afr. Med. J., **29**, 1050.
3. Gorham, L. W., Wright, A. W., Shultz, H. H. and Maxon, F. C. jun. (1954): Amer. J. Med., **17**, 674

SURGERY OF THE GALL-BLADDER AND THE COMMON BILE-DUCT*

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I. SURGERY OF THE GALL-BLADDER

The surgical treatment of diseases of the gall-bladder consists largely of the treatment of stones and their complications. There is a voluminous literature on gallstones, but its analysis would serve little purpose in this paper. It is of greater value to analyse one's own experience, to discuss how we can diagnose lesions of the biliary tract earlier, with greater accuracy, and how we can treat our patients more efficiently and with greater safety.

Cholecystectomy is one of the commonest of abdominal operations, usually associated with low mortality and morbidity; but this very fact has led to the mistaken view that it is also a simple operation. This light-hearted attitude is to be deplored for, not only may removal of the gall-bladder be an extremely difficult and dangerous operation, but it may be attended with the most serious accidents, such as damage to the common bile-duct.

I present for analysis a consecutive series of 250 cases (260 operations) of surgery of the biliary tract in private practice. This figure could have been more than doubled if one had included free cases operated on in hospital but, because clerking facilities are limited hospital cases are largely lost to follow-up. But although the analysis is confined to the more limited series the opinions expressed in this paper are based on the total experience.

This series of 250, being consecutive cases, is of more value than a selected group of cases of, say, calculous disease only, as it shows a fair cross-section of the type of biliary surgery which may be encountered in practice.

Table I shows an analysis of the type of biliary surgery performed in this series.

TABLE I

Cholecystectomy	230
Cholecystostomy	10
Stricture of common duct	3
Cholecysto-jejunostomy	2
Cystic duct, remnant excision	4
Secondary choledochostomy	4
Laparotomy, infective hepatitis	3
Carcinoma of gall-bladder, biopsy	1
Gallstone ileus	1
Sphincterotomy	2
Total operations	260

* Papers presented at the South African Medical Congress, Pretoria, 1955.

Fig. 1 indicates the age incidence in 230 cases of cholecystectomy. There were 160 females and 70 males. In both sexes the highest incidence occured in the 5th decade.

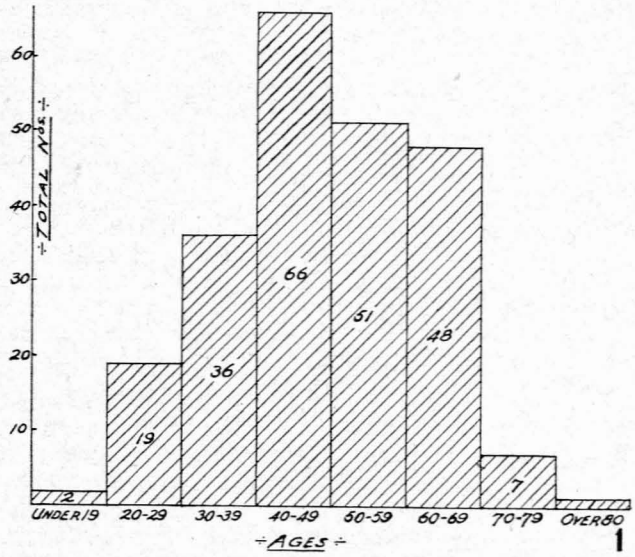

Fig. 1. Age incidence in 230 cases of cholecystestomy.

Table II shows an important analysis of the presence or absence of stones in the common bile-duct. This aspect of surgery of the biliary tract is discussed in Part II. The table indicates that, in all cases of cholecystectomy, it was considered necessary to explore the common duct in 29·1% of cases, that stones were present in 14·8% of all cases, and that, of all common ducts opened, stones were present in 50·7%.

TABLE II. STONES IN THE COMMON DUCT

Cases	*Common duct opened* No.	%	*Stones present* No.	%	*Common duct opened, found to contain stones*
230	67	29·1%	34	14·8%	50·7%

Table III analyses mortality rate. In cholecystectomy only, the mortality was *nil*. Where the common duct was explored, the mortality rate was 8·9%, making an average mortality rate in all cases of cholecystectomy,