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HONDSDOLHEID IN SUID-AFRIKA

Hierdie onderwerp is nog nie weer sedert 1949[1] in hierdie *Tydskrif* bespreek nie. Tot op die huidige oomblik is daar egter sewe gevalle van menslike sterfte gedurende 1961 aangemeld by die Staatsgesondheidsdepartement. Hierdie syfer verteenwoordig 'n aansienlike verhoging van die jaarlikse aantal menslike sterftes van hierdie siekte bo dié van die afgelope tien jaar. Die verhoogde voorkoms van menslike sterftes van hondsdolheid gedurende hierdie jaar baar groot onrus, veral nadat die siekte onlangs vir die eerste keer in Piketberg, in die Kaapprovinsie, aangemeld is gedurende Oktober. Die siekte het ook op aansienlike skaal uitgebreek in Natal. Gedurende die afgelope tien jaar is slegs enkele gevalle van verdagte hondsdolheid uit hierdie Provinsie aangemeld. Die distrik van Piketberg en Natal is altwee deur die Staatsveeartsenydiens tot gebiede verklaar waarin hondsdolheid voorkom.

Die manier waarop die onlangse hondsdolheid-infeksie versprei en die redes waarom dit nou weer voorkom, is nie bekend nie. Die hoof-reservoir van hondsdolheid in Suid-Afrika behoort tot die wilde dier-soort *Viverridae*.[2,3] In die volgende tabel word die aantal verdagte menslike gevalle van hondsdolheid wat met entstof behandel is en die betrokke dier-vektore aangegee.

Jaar	*Aantal verdagte gevalle van hondsdolheid wat in Suid-Afrika met entstof behandel is*	*Verantwoordelike dier*				
		Hond	*Kat*	*Bees*	*Mierkat*	*Ander*
1959	97	46	20	26	1	4
1960	61	24	15	10	5	7
1961 (tot einde Okt.)	71	26	11	12	8	14

Uit die bogemelde syfers blyk dit dat die meeste menslike gevalle voorkom as gevolg van kontak met huisdiere. Die huisdiere is waarskynlik intermediêre virus-vektore tussen die hoof-reservoir, *Viverridae,* en die mens.

Ons sou die onlangse tendense in die patroon wat hondsdolheid in Suid-Afrika vertoon, waarskynlik kan opsom in terme van die volgende oorwegings:

1. 'n Groot verhoging van die voorkoms van virusinfeksie onder die *Viverridae,* met 'n gevolglike vermeerderde oordraging na huisdiere en mense.
2. 'n Variasie van die giftigheid van die wilde virus met verhoogde soort-patogeniteit by diere en mense.
3. Oordraging van die virus deur roofvoëls en vlermuise.

Met betrekking tot die laaste oorweging moet dit vermeld word dat vlermuis-hondsdolheid nie in Suid-Afrika beskryf is nie, waarskynlik as gevolg van die afwesigheid van die vampier-vlermuis in dié gebied. Copley het vir die W.N.N.R. en in medewerking met die Veeartsenykundige Laboratoriums van Onderstepoort ondersoek ingestel na die moontlikheid dat hondsdolheid voorkom by grotvlermuise — met negatiewe resultate. Ten spyte van hierdie negatiewe bevindings behoort ons egter gedurig op ons hoede te wees in hierdie verband.

Die patogenese van hondsdolheid by mense en diere is nog nie heeltemal duidelik nie. Dit is goed bekend dat die virus neurotropies is, maar die presiese manier van verspreiding langs die perifere senuwees na die brein toe is nog nie vasgestel nie. Onlangse bevindings insake poliomiëlitis dui aan dat die virus, nadat dit toegang gevind het tot die liggaam, in die weefsels buite die senuweestelsel vermenigvuldig en gevolg word deur viremie. Schindler,[4] in 'n eksperimentele studie met gefikseerde hondsdolheid-virus, het gevind dat ‚die virus van hondsdolheid val die sentrale senuweestelsel aan sonder 'n vermeerdering by die ingangspunt en sonder enige vasstelbare viremie'.

Sir Macfarlane Burnet[5] meen dat die probleem heroorweging moet geniet in die lig van veranderde opvattings oor die analoë proses in poliomiëlitis en eksperimentele herpes simplex.

Die meganisme van infeksie van die speekselkliere is ook nog nie heeltemal duidelik nie. Die aantasting van speekselkliere sonder infeksie van die sentrale senuweestelsel is nog nie beskryf nie.

Die Staatsgesondheidsdepartement word as lid van die Wêreld-gesondheidsorganisasie op die hoogte gehou — deur die W.G.O. — van die onlangse vooruitgang van ondersoek in verband met hondsdolheid. Die W.G.O. tree ook in adviserende hoedanigheid op oor die metodes van immunisasie wat by die behandeling van pasiënte gevolg moet word.

Die behandeling van menslike kontakte by hondsdolheid hoort by die Staatsgesondheidsdepartement met sy netwerk van distriksgeneeshere tuis. Daar is geen praktiese tegniek beskikbaar wat geskik is vir die definitiewe diagnose van hondsdolheid in pasiënte net nadat hulle aan die siekte blootgestel is nie. Vinnige mediese optrede is dus absoluut noodsaaklik in persone van wie dit gemeen word dat hulle na blootstelling die gevaar loop om hondsdolheid te mag kry. Noodhulpbehandeling gevolg deur immunisasie-prosedures met serum en entstof teen hondsdolheid moet dus dadelik in werking gestel word.

Ensefalitis wat volg op die toediening van entstof vir hondsdolheid kom soms voor as 'n komplikasie by die behandeling met Semple se konynbrein-entstof (wat met ultraviolet-lig bestraal is en deur die gesondheidsdepartement uitgereik is). Indien moontlik, word hospitalisasie van die pasiënt aanbeveel.

Die behandeling van entstof-ensefalitis met steroïdes is ook beskryf. In die geval van een pasiënt is herstel gerapporteer na die toediening van hierdie middels.[6] Daar word egter ook gedink dat hierdie middels verantwoordelik is vir die belemmering van die immunologiese respons tot hondsdolheid-entstof wat aan 'n persoon toegedien is.[7] Ver-

dere ondervinding met steroïdes in die behandeling van ensefalitis wat volg op die toediening van entstof vir hondsdolheid skyn nodig te wees om hul nuttigheid vas te stel.

Die voorkoming van hondsdolheid in persone wat gereeld blootgestel word aan die risiko van infeksie is 'n onderwerp wat heelwat aandag geniet. 'n Onlangse aanbeveling van Greenberg en Childress[8] gee aan die hand dat eend-embrio-entstof gebruik moet word in plaas van brein-weefsel-entstof by die voorkomende behandeling van hondsdolheid.

Die maatreëls wat toegepas moet word vir die beheer van uitbreiding van hondsdolheid, is in die V.S.A. beskryf.[9,10] Sulke maatreëls veronderstel die hoogste graad van samewerking tussen mediese en veeartsenykundige dienste. Beheermaatreëls kan soos volg opgesom word:

1. Die vorming van aksiekomitees in die betrokke gebiede.

2. Die voer van aktiewe publisiteitskampanjes oor die radio en in die pers.

3. Die oprigting van nood-klinieke vir die inenting van honde.

4. Die invoer van streng afsonderingsregulasies.

5. Die aanhou van diere wat rondloop, in geskikte plekke, vir afsonderingsperiodes van ten minste ses maande.

6. Toesig oor wilde diere in epidemiese of ensoötiese gebiede, en die beheer van wilde roofdiere en vektorbevolkings.

7. Spoedige mediese behandeling van menslike kontakte deur verantwoordelike mediese outoriteite.

1. Van die Redaksie (1949): S. Afr. T. Geneesk., **23**, 337.
2. Snyman, P. S. (1937): J. S. Afr. Vet. Med. Assoc., **8**, 126.
3. Henning, M. W. (1949): *Animal Diseases in South Africa*. Kaapstad: C.N.A.
4. Schindler, R. (1959): Wld Hlth Org. Techn. Rep. Ser. 108/Rabies.
5. Burnet, F. M. (1960): *Animal Virology*. New York en Londen: Academic Press.
6. Briggs, G. W. en Brown, W. M. (1960): J. Amer. Med. Assoc., **173**, 7.
7. Burns, K. F., Shelton, D. F., Lukeman, J. M. en Grogan, E. W. (1960): Publ. Hlth Rep. (Wash.), **75**, 5.
8. Greenberg, M. en Childress, J. (1960): J. Amer. Med. Assoc., **173**, 4.
9. Schnurrenberger, P. R. en Russell, J. H. (1961): Publ. Hlth Rep. (Wash.), **76**, 4
10. Herbert, H. J. en Humphrey, G. L. (1961): *Ibid.*, **76**, 5.

RABIES IN SOUTH AFRICA

This is the first discussion on rabies that has appeared in this *Journal* since 1949.[1] On going to press, seven human deaths from rabies infection have been notified to the State Department of Health during 1961. This figure represents a marked increase over the preceding ten years in the annual number of deaths from this disease, and is regarded with concern. More particularly so, since the first recorded outbreak of rabies in the North West Cape occurred at Piquetberg during October. Another extensive outbreak of rabies has been reported from Natal, while over the ten previous years only an occasional case of suspected rabies has been notified from this Province. Both Piquetberg and Natal have been declared rabies-infected areas by the State Veterinary Service.

The mode of spread of recent rabies infection to the North West Cape and reasons for the increased prevalence of the disease in Natal are not known. The main reservoir of rabies infection in South Africa is the wild-life animal species *Viverridae*.[2,3] Notifications of the number of suspected human rabies cases treated with vaccine over the last three years and the animal vectors responsible are reflected in the following table:

Year	*Notifications of suspected humans treated with vaccine in South Africa*	*Responsible animal*				
		Dog	*Cat*	*Bovine*	*Meerkat*	*Other*
1959	97	46	20	26	1	4
1960	61	24	15	10	5	7
1961 (to 1 Oct.)	71	26	11	12	8	14

The abovementioned figures indicate that most human cases occur as a result of contact with domestic animals, the latter probably being intermediate virus vectors between the main reservoir in *Viverridae* and man.

It seems reasonable to postulate that recent trends in the pattern followed by rabies infection in South Africa could be assessed in terms of the following considerations:

1. A marked increase in the incidence of virus infection among the wild-life species *Viverridae*, with a consequent increased transmission to domestic animals and man.

2. A virulence variation of the wild virus with increased species pathogenicity in domestic animals and man.

3. Transmission of virus by predatory birds and bats.

With regard to the third consideration it must be stated that bat rabies has not been described in South Africa, probably because of the absence of the vampire bat species in this region. Copley, on behalf of the Council for Scientific and Industrial Research, and in collaboration with the Onderstepoort Veterinary Laboratories, has investigated the possibility of rabies infection in cave bats with negative results. Nevertheless, despite these negative findings, constant vigilance should be maintained and encouraged in this field.

The pathogenesis of rabies infection in man and animals is not clearly understood. That the virus is neurotropic in activity is well known; however, the precise manner of spread along the peripheral nerves to the brain has not been established. Recent findings in relation to poliomyelitis virus indicate that this virus, after gaining access to the body, multiplies in tissues outside the nervous system and is followed by viraemia. Schindler,[4] in experimental studies with fixed rabies virus, has found that 'the rabies virus attacks the central nervous system without an increase at the portal of entry and without any detectable viraemia'.

It is the opinion of Sir Macfarlane Burnet[5] that 'the problem needs re-examination in the light of changing views about the analogous process in poliomyelitis and experimental herpes simplex'.

The mechanism of salivary-gland infection is likewise not fully explained. Salivary-gland involvement in the

absence of central-nervous-system infection has not been described.

The State Department of Health, as a member of the World Health Organization, is kept informed by this body of recent advances in rabies research. The WHO also acts in an advisory capacity on the methods of immunization to be adopted for the treatment of patients.

Treatment of human rabies contacts is the responsibility of the State Department of Health through its network of District Surgeons. For practical purposes no laboratory technique is at present available whereby a definite diagnosis of rabies infection in patients can be established immediately following exposure to the risk of the disease. Therefore prompt medical action is absolutely imperative in persons who have been assessed as a definite exposure risk to rabies virus infection. First-aid treatment followed by immunization procedures with antirabies serum and vaccine should be instituted immediately.

Post-rabic vaccinal encephalitis does occur as a complication in treatment with the Semple-type rabbit-brain vaccine (ultraviolet-light irradiated) issued by the Department of Health. In order to counteract this complication it is recommended that patients should be carefully observed throughout the period of treatment. If possible, hospitalization of patients is advised. The treatment of post-rabic vaccinal encephalitis with steroid drugs has been described, and in one patient dramatic recovery is reported to have followed the administration of these agents.[6] On the other hand, these agents are also believed to have been responsible for impairment of immunological response to the administration of rabies vaccine in a reported human case.[7] Further experience with steroid drugs in the treatment of post-rabic vaccinal encephalitis seems necessary in order to ascertain their usefulness.

The prophylaxis of rabies in persons regularly exposed to the risk of infection is a subject which has received considerable attention. A recent recommendation advanced by Greenberg and Childress suggests that duck-embryo vaccine should supplant the use of brain-tissue vaccine for the prophylaxis of rabies infection.[8]

Measures to be adopted for the control of rabies outbreaks have been described in the USA.[9,10] Such measures call for the highest order of cooperation between the medical and veterinary services.

Control measures can be summarized as follows:

1. The formation of action committees in the affected areas. Such committees to be advised by medical and veterinary staff.
2. The promotion of energetic publicity campaigns by radio and press organizations.
3. The siting of emergency dog-vaccination clinics.
4. The introduction of strict quarantine regulations.
5. Control of stray animals by isolation in suitable premises for quarantine periods of at least six months.
6. Wild-life surveillance of epidemic and enzootic areas with control of predatory wild-life and vector populations.
7. Prompt medical treatment of human contacts by the responsible medical authorities.

1. Editorial (1949): S. Afr. Med. J., **23,** 337.
2. Snyman, P. S. (1937): J. S. Afr. Vet. Med. Assoc., **8,** 126.
3. Henning, M. W. (1949): *Animal Diseases in South Africa.* Cape Town: Central News Agency.
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6. Briggs, G. W. and Brown, W. M. (1960): J. Amer. Med. Assoc., **173,** 7.
7. Burns, K. F., Shelton, D. F., Lukeman, J. M. and Grogan, E. W. (1960): Publ. Hlth Rep. (Wash.), **75,** 5.
8. Greenberg, M. and Childress, J. (1960): J. Amer. Med. Assoc., **173,** 4.
9. Schnurrenberger, P. R. and Russell, J. H. (1961): Publ. Hlth Rep. (Wash.), **76,** 4.
10. Herbert, H. J. and Humphrey, G. L. (1961): *Ibid.*, **76,** 5.