**Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal**

Kaapstad, 16 Maart 1963 Deel 37 No. 11 Volume 37 Cape Town, 16 March 1963

VAN DIE REDAKSIE : EDITORIAL

## HOEKOM ONS NIE GEWOONLIK NABEELDE SIEN NIE

As u na die horlosie op die kaggel kyk en dan na die muurskildery direk bokant, sien u nie enige aspek van die horlosie gesuperimponeer op die skildery nie. Hoekom nie? Om die geheue te verfris, Brink[1] definieer 'n nabeeld as volg: ‚die beeld van 'n voorwerp wat in die bewussyn bly voortbestaan nadat die werklike beeld van die voorwerp self nie meer op die retina val nie; met ander woorde, 'n subjektiewe verskynsel wat optree nadat die objektiewe prikkel opgehou het om op die oog in te werk'. Hoekom sien ons hulle dan nie altyd nie, soos elkeen self hierdie fundamentele feit van die visuele proses sal kan bevestig? Hierdie vraag is onlangs deur Daw ondersoek,[2] maar is reeds deur Goethe verklaar op grond van gebrekkige aandag en deur Helmholtz op grond van 'n stadige, onwillekeurige ronddwaal van die oog. Behalwe vir Joegoslawiese werk in 1952, word Goethe en Helmholtz se verklarings aanvaar as die enigste waarskynlike verklarings, hoewel Daw 'n ander aspek kon beklemtoon.

As twee skyfies geprojekteer word, een in rooi en die ander in wit lig, ontstaan sensasies van rooi, wit, pienk, grys, geel, blou, groen, bruin, oranje en swart.[3] As verklaring is aangevoer dat die verskynsel te wyte is aan opvolgende kontraste oftewel, nabeelde. Dit maak nie saak waar die waarnemer kyk op die oorspronklike beeld nie, die kleure verskyn heeltemal onafhanklik daarvan. Die kleure wat in 'n flitsbeligting gesien word, is baie soos dié in aanhoudende lig. Die vraag is dus of ons waarneming werklik verward raak met nabeelde en opeenvolgende kontras.

Daw beskryf eksperimente waarin 'n gekleurde toneel in rooi lig geprojekteer word, en gesuperimponeer word op 'n groenkleurige skyfie in wit lig. Die waarnemer konsentreer op een punt in die toneel vir twaalf sekondes en dan word die rooi lig afgeskakel. Die sekondêre toneel was 'n swart-en-wit foto. As die waarnemer nog aanhou kyk na dieselfde punt sien hy 'n gekleurde nabeeld. Die kleure bly dieselfde as ander fiksasiepunte gekies word, mits dieselfde punt as fiksasiepunt in die sekondêre beeld gebruik is. As hy nie aanhou kyk na die fiksasiepunt nie, verdwyn die nabeeld en die hele toneel word swart-en-wit. As hy wegkyk van die fiksasiepunt af, en dan weer terugkyk, verdwyn die nabeeld solank hy wegkyk maar keer terug as hy weer op die bepaalde punt fikseer. 'n Geringe beweging vanaf fiksasiepunt laat die nabeeld verdwyn. Die nabeeld neem ongeveer 12 sekondes om heeltemal te verdwyn. Deur die prosedure hierbo genoem, kon nabeelde herhaaldelik terug geroep word, maar elke keer swakker. Eksperimente is ook gedoen met skerp en swak kontrasbeelde en in 'n swak beligte vertrek (1 voet-lambert) tot redelik helder daglig (500 voet-lamberts). Die resultate was dieselfde behalwe dat nabeelde gouer vervaag het in helderder lig as in dowwe lig.

Hieruit word afgelei:

(*i*) Dat nabeelde eerder geïnhibeer as vernietig word — hulle keer terug as die oog weer op dieselfde punt fikseer. 'n Ander eksperiment het getoon dat as die oog na 'n nuwe punt beweeg, die nabeeld herverskyn waar die toneel die nabeeld pas. Soos die oog rondbeweeg, bly die nabeeld daarin, onsigbaar en intakt, ten spyte van alle ander indrukke wat op die retina gemaak word.

(*ii*) As die kontoere van die nabeeld geïnhibeer word, word sy kleure ook geïnhibeer, en

(*iii*) Vir doeltreffende inhibisie is kontoere en detail in die sekondêre toneel belangrik. As die sekondêre toneel uit swak en vae kontoere bestaan, verskyn die nabeeld swak. As daar geen kontoere is nie, is die nabeeld redelik volledig.

Die skrywer besluit dan dat die drie verklarings vir nabeelde elk tot 'n mate waar is: Goethe se teorie is nie soseer 'n verklaring nie, as 'n voorskrif van hoe om nabeeld uit te lok, naamlik die konsentrasie. Helmholtz beweer dat ons nie gewoonlik nabeelde sien nie omdat ons nie lank genoeg op een punt fikseer om 'n nabeeld te sien nie, terwyl Daw se studies toon dat 'n nabeeld vorm na 1 - 2 sekondes van fiksasie en dat dit heeltemal algemeen voorkom, maar dat die nabeeld geïnhibeer word deur enige daaropvolgende stimulasiepatroon waarvan die kontoere in stryd is met dié van die nabeeld.

Ons vorm dus gedurig nabeelde, maar ons sien hulle nie. Sommige is sterk, ander swak, afhanklik van hoe lank die oog op 'n punt rus voordat dit die res van die toneel verken. Hulle word saamgedra met die oog en tree te voorskyn as die oog weer rus op 'n beeld wat geometries ooreenkom met die nabeeld. Dit gebeur selde in die gewone lewe want ons het nie te doen met geometries-eenvoudige patrone nie, maar wel met bome en huise, gesigte en rokke, en met helder en dowwe ligte wat die nabeelde voortdurend inhibeer.

Was dit Prediker wat gesê het: ‚Die oog word nie moeg van sien nie'?

1. Brink, H. E. (1957): *Menslike Fisiologie*, deel II, p. 132. Stellenbosch: Universiteitsuitgewers.
2. Daw, N. W (1962): Nature (Lond.), **196**, 1143.
3. Land, E. H. (1959): Proc. Nat. Acad. Sci. (Wash.), **45**, 115 en 636.

## ACTIVE SUBSTANCES IN THE CENTRAL NERVOUS SYSTEM

A number of substances which have powerful pharmacological actions (on other organs) have been identified in the central nervous system. Acetylcholine, adrenaline, noradrenaline, 5-hydroxytryptamine, gamma aminobutyric acid, and substance P are present in nervous tissue and would appear to be concerned in the transmission of impulses at synapses. Other techniques will, no doubt, reveal other active substances in the nervous system.

The complexity of the nervous system makes investigation of the action of drugs and of naturally-occurring substances at central synapses a difficult problem. Yet considerable progress in this field has been made in recent years. A useful review of the subject has appeared in the latest edition of *Recent Advances in Pharmacology*.[1]

Some parts of the central nervous system contain considerable quantities of acetylcholine and others very little. The concentration in different parts has been determined. Evidence makes it likely that there is an alternation of cholinergic and non-cholinergic (but cholinoceptive) neurones in some pathways. Experiments have also shown an association between cerebral activity and an increased turnover in acetylcholine. The evidence for cholinergic transmission at central synapses is nevertheless not yet so well established as that for the autonomic ganglia and the neuromuscular junctions.

The catechol amines found in the brain are dopamine, noradrenaline and adrenaline; they have much in common with 5-hydroxytryptamine (serotonin) in their synthesis, inactivation, storage and distribution, and certain drugs tend to modify the concentrations of all these amines. Dopamine and noradrenaline are found in the mammalian brain in considerable amounts, but the amount of adrenaline is much less. The amount of 5-hydroxytryptamine is about the same as that of dopamine and noradrenaline. The concentrations of these amines in different parts of the brain show considerable variations; a table with full details is given in the reference article.[1] No conclusions can be drawn about the functions of the amines from the data provided. The amines do not pass easily through the blood-brain barrier and their central actions are slight after systemic injection as compared with their well-known peripheral actions. Amine oxidase inhibitors cause a rise in the concentration of the amines and evidence of cerebral stimulation. Reserpine, which releases the amines and reduces their concentrations in the brain, and alpha methyldopa ('aldomet'), which also decreases the amines, have a sedative action.

Gamma aminobutyric acid (GABA) has been known to be present in living matter, including mammalian brain, for a number of years. Pyridoxal deficiency tends to depress its synthesis, the site of which is at present little known. This organic acid is found throughout the brain, the amounts in grey matter being generally higher than those in white matter. The actions of GABA, and of factor 1, make these the most likely of the known substances in the brain to have an inhibitory action. Substance P, which is a polypeptide of unknown composition found in high concentration in sensory nerves and their continuation in the posterior columns, may be a transmitter. Further investigation of this substance is worth while, but will not be easy because of the lack of pure preparations of high potency.

Histamine is present in considerable amounts in some peripheral nerves, but in the central nervous system the amounts are relatively low. There are high concentrations in the pituitary gland, especially in the stalk and the posterior lobe, and it is here that there are considerable numbers of mast cells which elsewhere in the body are able to synthesize and store histamine. There is no evidence at present that histamine plays a role in nervous transmission.

Considerable amounts of adenosine compounds are present in the brain in the bodies of neurones and in both myelinated and non-myelinated fibres. It has been suggested that adenosine triphosphate (ATP) may be concerned in the binding of noradrenaline in adrenergic nerves.

Most of the substances that have been mentioned as occurring in the central nervous system have been identified and estimated because of powerful pharmacological actions they exert on an isolated organ or on the peripheral circulation of an experimental animal, actions which appear to have little in common with any possible function they may have in the brain. However, acetylcholine, adrenaline, noradrenaline, 5-hydroxytryptamine, gamma aminobutyric acid, and substance P all probably exert actions on receptors on cell membranes. The peripheral nerve transmitters acetylcholine and noradrenaline are also rapidly liberated in the central nervous system from sources where they are held inactive and they are rapidly inactivated by enzymes that are easily available. However, although certain neurones in the central nervous system can be classified as cholinergic, identification of adrenergic neurones is much less certain.

1. Robson, J. M. and Stacey, R. S. (1962): *Recent Advances in Pharmacology*, 3rd ed. London: J. & A. Churchill.

## 44th SOUTH AFRICAN MEDICAL CONGRESS, JOHANNESBURG 21—27 JULY 1963

**As already advised to those who have submitted titles of papers they wish presented at Congress, the latest date for the receipt of synopses is 31 March 1963. The full papers must reach the Congress Office not later than 30 April 1963.**

**These dates are very important.**