South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

VAN DIE REDAKSIE

HARTVERLAMMING

L. N. Katz [1] het onlangs 'n lesing aan die Kollege van Geneeshere van Philadelphia gegee wat gegaan het oor die meganisme van hartverlamming op grondslag van die opvattings van sy ,skool' wat die probleem vir amper 25 jaar al bestudeer het. Hy versmaai ,the recent trend of semantics in which the terms congestive failure, low-output failure, and high-output failure have come into vogue'. Hy is van mening dat hierdie uitdrukkings tot 'n mate van misverstand gelei het; ,low-output failure' mag aan baie faktore te wyte wees en die hart hoef in die eerste instansie nie noodwendig daarmee betrokke te wees nie, en, ,high-output failure' is 'n verkeerde benaming. Hy beskou ,congestive failure' ook as 'n verkeerde benaming aangesien kongestie kan plaasvind of die hartbloedsomloop afneem of nie en dit kan ook voorkom in ongesteldhede van die bloedvatstelsel wanneer daar met die hart niks verkeerd is nie.

Hy versuim ook nie om die losse gedagtegang i.v.m. die begrip van sogenoemde regter- en linkerhartverlamming te korrigeer nie. In dinamiese ewewig pomp die regter- en linkerhartkamers dieselfde hoeveelheid bloed; slegs wanneer 'n pasiënt se bloedsomloop begin verswak, of hy van die verswakking herstel, of as hy sterwend is, is daar wat 'n geringe volume betref enige onewewigtigheid hoegenaamd, maar daar is geen ongelykheid in die hoeveelheid wat die twee kante van die hart uitstoot nie. Ondoeltreffendheid volg wanneer die hart onbekwaam is om sy werk te doen. Somtyds lei dit tot die staking van die hartbloedsomloop (afgesien van ekstrahartbloedsomloopstaking) en somtyds tot kongestie.

Inspanning van die hart mag die gevolg wees van 'n toename in die bloed wat deur die are terugvloei, 'n toename in die groot- of longslagaarbloeddruk, of van sekere ander laste wat nie so maklik verklaar kan word nie. Die meganismes wat betrokke is by die oorkoming van die verhoogde lading is (*a*) die eenvoudige meganisme van uitsetting; (*b*) die toename in die spiermassa, deur hipertrofie, wat tyd neem; (*c*) hartversnelling, as gevolg van verminderde cholinergiese en verhoogde adrenergiese invloed; (*d*) verhoogde saamtrekkingskrag van die hartspiere wat deur hormoon- en refleksmeganismes teweeggebring word.

Hierdie is die 4 kompensasie-meganismes waarmee

EDITORIAL

CARDIAC FAILURE

In a recent lecture to the College of Physicians of Philadelphia the mechanism of cardiac failure was presented by L. N. Katz [1] based on the concepts of his 'school' which has been studying the problem for nearly 25 years. He scorns 'the recent trend of semantics in which the terms congestive failure, low-output failure, and high-output failure have come into vogue'. He feels these terms have led to some confusion; low-output failure may be due to many factors and need not be primarily cardiac, while high-output failure is a misnomer. He regards congestive failure also as a misnomer, as congestion may occur whether there is cardiac circulatory failure or not, and it may also occur in disorders of the vascular system when the heart is competent.

He does not omit to correct the loose thinking associated with the concept of so-called right and left heart failure. In dynamic equilibrium the right and left ventricles pump out equal quantities of blood; only when a patient is going into circulatory failure or recovering from it, or is moribund, is there any disequilibrium as far as minute volume is concerned, but there is no disparity in output of the two sides of the heart. Heart failure arises from incompetency of the heart. Sometimes this leads to cardiac circulatory failure (as distinct from extracardiac circulatory failure); and sometimes this leads to congestion.

Stress upon the heart may be due to an increase in the venous return, to an increase in the systemic or pulmonary arterial blood pressure, or to certain other loads not so easily demonstrated. The mechanisms that are involved for overcoming the increased load are (*a*) the simple mechanism of dilatation; (*b*) increase of the mass of muscle, by hypertrophy, which takes time; (*c*) tachycardia, through reduced cholinergic and increased adrenergic influence; (*d*) an increase in the contractile power of the myocardium brought about by hormonal and reflexogenic mechanisms.

These are the four compensatory mechanisms by

die hart sy spannings oorkom en wanneer hul almal saam in werking is, is hul onderling afhanklik. Op grond van hierdie opvattings is dit ook moontlik om hartreserwe in terme van uitsetting, oorvergroting, hartsnelheid en saamtrekking te definieer, alhoewel dit nie altyd moontlik is om hierdie 4 hartreserwemaatreëls duidelik te definieer nie. Daar is egter iets te sê vir die begrip dat daar 'n perk vir elkeen van hierdie maatreëls is waarbo verdere toename nie moontlik is nie, of indien moontlik, nadelig. Die aanpassings, wat vir 'n stygende lading op die hart vergoed, is beperk. 'n Stadium word bereik wanneer die meganismes onvoldoende is en 'n nuwe reeks gebeurtenisse vloei voort wat ingewikkeld is; baie dele van die bloedsomloop raak betrokke in 'n wisselwerking tussen menige vloeistof- en neurogeniese beheermeganismes.

'n Ander aspek van hartverlamming wat deur Katz behandel is, is die meganisme van hartedeem. Sy gevolgtrekkings oor die kwessie van edeemvorming in kongestie is dat dit nie stuwing in enige besondere deel van die liggaam is of buislose klierstowwe wat die abnormale natriumchloried- en vloeistofwerking van die nier veroorsaak nie maar stuwing *per se*. Die aardrukverhoging bewerkstellig op een of ander manier 'n meganisme wat die nier natriumchloried en water laat terughou. Edeem by hartverswakking word deur die hart veroorsaak maar ongesteldheid van nier-(buis)werking is die gevolg van 'n *receptor-effector*-meganisme. Faal die hart dan reageer die liggaam asof natrium ontbreek terwyl dit in werklikheid met natriumchloried en water oorlaai is. Die mening is dat dit die herabsorpsie van hierdie stowwe is wat geraak word, maar Katz gee aan die hand dat daar inmenging is met die werklike afskeiding van natriumchloried of water uit die bloed uit na die nierbuise.

1. Katz, L. N. (1954): Trans. Coll. Phys. Philad., **22**, 53.

which the heart meets its stresses, and when all operate together they are interdependent. From these concepts too it is possible to define cardiac reserve in terms of dilatation, hypertrophy, heart rate, and contractile power, although it is not always possible to define these four measures of cardiac reserve clearly. There is, however, merit in the concept of an upper limit for each of these measures beyond which further increase is impossible or, if possible, becomes detrimental. The adjustments which compensate for an increasing load on the heart are limited. A point is reached where the mechanisms become inadequate and a new series of events ensue which are complex; many parts of the circulation become involved with an interplay of many humoral and neurogenic regulatory mechanisms.

Another aspect of heart failure considered by Katz is the mechanism of cardiac oedema. His conclusions on the question of oedema formation in congestion is that it is not stasis in any special part of the body or endocrine substances which cause sodium-chloride and water dysfunction of the kidney but stasis *per se*. Rise in venous pressure somehow sets up a mechanism that causes the kidney to retain sodium chloride and water. Oedema in heart failure is caused by the heart, but derangement of kidney (tubule) function is produced through a receptor-effector mechanism. In heart failure the body reacts as though it were depleted of sodium whereas actually it is overloaded with sodium chloride and water. It is believed that it is re-absorption of these substances that is affected, but Katz suggests there may be interference with actual secretion of sodium chloride or water out of the blood into the tubules.

1. Katz, L. N. (1954): Trans. Coll. Phys. Philad., **22**, 53.

CHEMISTRY OF MIND

The physiological background of thinking and feeling has long been a problem and a challenge to research workers. With recent advances in biochemical knowledge some progress in being made in this difficult field. In 1954 Woolley and Shaw[1] described the properties of a substance called serotonin (5-hydroxytryptamine) which is normally present in mammalian sera. Its main actions are twofold—it is a vasoconstrictor by virtue of its effect on smooth muscle and it also appears to be of importance in neural function. Large amounts of serotonin are found in brain, peripheral nerve and visceral ganglia. In an attempt to discover its precise function in cerebral metabolism attention was directed to a group of substances which are so closely related chemically to serotonin that they block its action on the brain. Some of these substances such as the ergot alkaloids, harmine etc. have been known for centuries to produce a transient abnormal mental state characterized by visual hallucinations, change in affect, feelings of unreality etc. in a setting of clear consciousness. Woolley and Shaw suggested that a deficiency of serotonin might be the cause of the spontaneously occurring psychosis, schizophrenia, because the 'experimental psychoses' resemble it so closely.

Another group of workers (Hoffer, Osmond and Smythies)[2] were also interested in the experimental psychoses. Their starting point was that mescaline, which is well known for the extraordinary mental abnormalities it produces, has a chemical structure similar to adrenaline. If they could find a 'midway' substance with the physiological effects of adrenaline and the psychological effects of mescaline they might find the answer to schizophrenia. They systematically tested substances chemically similar to mescaline and found a small number that caused psychological disturbances. They were all plant alkaloids—ibogaine (from an African bean), lysergic acid (from rye rust), hashish (from Indian hemp) and a fungus *Amanita pantherina*. Then by chance they found that a degradation product of adrenaline—called adrenochrome because of its pink colour—had a similar chemical structure to these plant alkaloids, could produce identical psychological disturbances, and could occur in the human body. They tentatively suggested that adrenochrome might be

the answer to schizophrenia, and by injecting themselves intravenously with adrenochrome they produced transient abnormalities of thinking and feeling which they claim are indistinguishable from those observed in schizophrenia.

One of the mescaline-related plant alkaloids—lysergic acid—is already being used therapeutically by some psychiatrists (Sandison [3]). They say that it can be of great use in neurotic patients because minute doses by mouth produce a transient psychosis in which the patients' unconscious fantasies and memories 'erupt' into consciousness, providing valuable information for analysis.

It may be that adrenochrome and the plant alkaloids of the mescaline group all work by acting as antagonists to serotonin, but whether there is this link between serotonin and adrenochrome has not been established. What does emerge from these interesting and stimulating experiments is the fundamental importance to progress in psychiatry of basic medical research.

1. Woolley, D. W. and Shaw, E. (1954): Brit. Med. J., **2**, 122.
2. Hoffer, A., Osmond, H. and Smythies, J. (1954): J. Ment. Sci., **100**, 29.
3. Sandison, R. A. (1954): *Ibid.*, **100**, 508.