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VAN DIE REDAKSIE : EDITORIAL

AAN DIE HOOF VAN DIE TAFEL

Dit is hoog tyd dat die plek van die narkotiseur in die geneeskundige beroep heroorweeg word. Sedert Morton 117 jaar gelede die eerste keer 'n demonstrasie van die bedwelmende uitwerking van eterdamp gegee het, het narkose-toediening met die ontwikkelinge in die ander vertakkinge van die geneeskunde tred gehou—aanvanklik op 'n sukkeldraffie, maar veral in die afgelope tien jaar teen 'n versnelde tempo.

Vandag staan narkose as 'n eiesoortige spesialiteitsvak sy plek vol in die geneeskundige bediening van die publiek. Die persoon aan die hoof van die tafel speel 'n sleutelrol by elke operasie. Hy is vandag baie meer as net 'n vakkundige manipuleerder van toestelle wat slegs die diepte van bewusteloosheid peil en reguleer. Hy is meer as 'n wetenskaplike wat slegs vertroud is met die eienskappe van chemiese samestellings in die laboratorium. Die koms van suksinielcholien, die ander spierverslappers en beter toerusting het ook nie soseer van hom 'n beter dokter gemaak nie. Eerder is dit die nuwer benadering om die bewustelose pasiënt in sy geheel en met die insig van 'n internis te behandel, wat hiervoor verantwoordelik is.

Soos die skrywers van 'n onlangse artikel[1] tereg aanvoer, beklee die narkotiseur die unieke plek om die fisiologiese verskynsels wat by chirurgiese pasiënte voorkom, te bestudeer, of hulle nou ook al geestelik—soos spanning en vrees en pyn—of fisiek—soos die uitwerking van belemmerde lugtevoer—is. Die vordering wat uit hierdie waarneming gevloei het, was nie net om meer gemak vir die pasiënt en groter gerief vir die chirurg te bring nie, maar ook om operasies oor die algemeen veiliger te maak en om deur die koppeling van hierdie vermoëns groter en meer ondernemende chirurgiese operasies moontlik te maak.

By die narkotiseur van vandag is daar ook nie meer beperkte kontak met die pasiënt nie, of daar behoort altans nie te wees nie. Sy plig gaan verder as net die toediening van narkosedampe en die inspuiting van slaapwekkers. Die tegniese kennis van *narkosetoediening* het plek gemaak vir die *versorging van pasiënte* wat onder narkose verkeer.[1] Dit is veral in laasgenoemde verband dat die narkotiseur sy nuwere opleiding moet gebruik en die rol van 'n interne geneesheer moet behartig. Nie net word die pasiënt se *asemhaling* vóór, gedurende en ná die operasie beheer nie, maar ook die sirkulatoriese veranderinge word waargeneem en beheer (bv. hipotensie en perifere bloedtoevoer) en die *metabolisme* word na willekeur gewysig (bv. deur verkoeling). Die belangrike vóór- en ná-operatiewe diens wat die narkotiseur aan die chirurgiese pasiënt gee, moet beklemtoon word. Veral in laasgenoemde geval word by die dag beter begryp dat sy werk nog glad nie afgehandel is op die oomblik wanneer hy die intratrageale buis uit die pasiënt se keel getrek het nie.

Daar word beweer dat die grootste verandering in die narkotiseur se benadering tot die pasiënt ingetree het toe hy die asemsak van die apparaat tussen sy hande gegryp het om *self* die respirasie te beheer pleks van die sak se bewegings soos 'n toeskouer gade te slaan. Deur hierdie beweging het die narkotiseur oorgegaan tot die beherende rol wat hy vandag by operasies speel.

Hipoteties kan die vraag nou gestel word of geneeshere oor die algemeen voldoende bewus is van die aanpassing wat daar by die toediening van narkose ingetree het. Kry die persoon aan die hoof van die tafel genoeg erkenning van sy kollegas, veral van die chirurge? Het sy status toegeneem in verhouding tot sy groter veelsydigheid en die groter diens aan sowel die pasiënt as die chirurg? Moet hy nie alte dikwels tweede viool speel wanneer daar berekening gemaak word van chirurgiese welslae nie?

Wanneer daar roeringe onder die narkotiseursgroep aan die gang is om op hoër gelde vir hul dienste aan te dring, moet hierdie feite en vrae objektief beskou word en die beeld van die narkotiseur moet in sy ware perspektief gestel word.

1. Beecher, H. K., Bendixen, H. H., Hallowell, P., Pontoppidan, H. en Todd, D. P. (1964): J. Amer. Med. Assoc., **188**, 49.

BRADYKININ

Bradykinin is the name given to a polypeptide formed by the action of certain enzymes on plasma proteins.[1] A number of related peptides with vasodilator and smooth-muscle stimulating properties formed in this way are referred to as plasma kinins, and of these the chemical structure of bradykinin is known. The appearance of plasma kinins in blood has been observed under various conditions. It seems that there is a very delicate balance in the blood controlling the formation of plasma kinin. It is possible that there is a continuous formation of small amounts of plasma kinin. The whole system is controlled by the presence of inhibitors of plasma enzymes, and an enzyme which rapidly destroys plasma kinin.

Bradykinin was recently isolated[2] and with the aid of synthetic chemists from Sandoz Laboratories its structure was determined. Crystalline trypsin was used as the enzyme and as substrate the pseudoglobulin fraction of the plasma proteins was prepared for the formation of the plasma kinin. The first efforts suggested incorrectly that the structure was an octapeptide, synthesis of which yielded an inactive substance. Further considerations suggested that bradykinin is a nonapeptide, and when this was synthesized it was found that the synthetic and the natural products were identical.[3]

The pharmacological actions of bradykinin include stimulation of several smooth-muscle structures, dilatation

of blood vessels, increase in capillary permeability, accumulation and migration of leukocytes, and the production of pain. Very significant is the vasodilator action in which regard it is the most potent of all substances having this action in man. Intravenous injection causes a flushing in the face and neck and, with larger doses, dilatation of peripheral vascular beds. The polypeptide is rapidly destroyed in the blood.

Bradykinin increases capillary permeability which is different from that produced by histamine. It does not produce the typical classical response.

Although the changes produced by bradykinin resemble those that occur in the early stages of an inflammatory reaction, there is no direct evidence of the part played by this agent in local tissue reactions. There is experimental evidence that the plasma-kinin forming mechanism is important in producing the vasodilatation which accompanies activation in all glands, (including salivary, lachrimal, pancreatic). It has also been suggested that bradykinin may play a role in shock, allergy, and anaphylaxis. In such circumstances the suggestion is that plasma-kinin formation takes place in the general circulation and causes generalized peripheral vasodilatation, collapse and shock.[3]

It has recently been demonstrated that a kinin peptide is released in the carcinoid syndrome.[4] The role of 5-hydroxytryptamine (serotonin) as the sole mediator of flushes in patients with the carcinoid syndrome has been questioned, since injection of the amine does not produce a typical flush, and the levels of the amine in the blood do not correlate well with attacks of flushing. Since it has been shown that catecholamines release a kinin peptide it was suggested that the latter might be responsible for the carcinoid flushes. Bradykinin was found to produce the flushes. It has been shown that carcinoid-tumour metastases contain an enzyme (kallikrein), which can be activated or released by adrenaline, alcohol, and other stimuli, and this catalyzes the formation of a vasoactive kinin peptide from the substrate (kallidinogen). The exact physiological action of the carcinoid kinin needs to be demonstrated. In addition to the flush several other features of the carcinoid syndrome, that cannot be ascribed to the action of serotonin, may be due to kinins. These include bronchoconstriction, decrease in peripheral vascular resistance producing a high-output state, altered endothelial permeability—actions which may account for the development of the heart lesions in the carcinoid syndrome. The fact that the levels of kinin fall markedly during their passage from hepatic vein to peripheral artery may explain why the most severe valvular lesions are found on the right side of the heart. Synthetic bradykinin (Sandoz) was used in the studies mentioned, a single dose of 0·1 - 1·5 microgram per kg. intravenously producing flushing episodes resembling those occurring spontaneously in the carcinoid patients.

1. Gaddum, J. H. (1955): *Polypeptides*. London: Livingstone.
2. Elliott, D. F., Horton, E. W. and Lewis, G. P. (1961): Biochem. J., **78**, 60.
3. Lewis, G. P. (1962): *The Scientific Basis of Medicine Annual Reviews*. London: Athlone Press.
4. Melmon, K. *et al.* (1964): Lancet, **1**, 514.