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Wetenskaplikheid in die Praktyk

Alle pasiënte wat 'n geneesheer kom spreek is nie akuut siek nie, en veral in die geval van huisartse leer ervaring dat 'n aansienlike deel van die dokter-pasiënt kontakte die gevolg is van relatief onbelangrike probleempies en blote roetinewerk. Sommige pasiënte kom slegs om 'n aflaaiplek vir hul emosionele spannings te vind; andere kom vir roetine kontrole van bloeddruk of vir gereelde inspuitings van vitamiene B_{12} of iets dergeliks. Selfs diegene wat pas siek geword het en die dokter na hul huise ontbied het, ly nie almal aan ingewikkelde of gevaarlike sindrome nie. Die huisarts sien dag na dag talle mense met ligte keelinfeksies, verkoues, griepaanvalle en dies meer.

Die ervare geneesheer leer ook om met verloop van jare die gevaarliker toestande soos longontsteking, akute nierontsteking en middeloorabsesse sonder 'n sweem van paniek of selfondersoekende twyfel die hoof te bied. In die oë van die publiek, veral diegene wat geesdriftige lesers van populêre mediese romans is, ry die dokter van huis tot huis en van hospitaal tot hospitaal, om uur na uur lewensreddende werk te doen en briljante diagnoses te maak wat die hele toekoms van die betrokke pasiënte ingrypend verander. Dit duur nie lank voordat die nuwe geneesheer ontdek dat sulke dramatisering van die praktyk op totale wanbegrip berus nie. Spanningsoomblikke is daar wel, en juis omdat niemand kan voorspel wanneer hulle hul koppe gaan uitsteek nie, moet die dokter op sy hoede wees. Dit is 'n voortdurende tweestryd tussen die afstompende effek van sieldodende roetine en die paraatheid om ter enige oomblik gereed te wees om werklik lewensreddend op te tree, wat die lewensaard van die geneesheer kenmerk.

Almal weet dit en die meeste dra sorg dat sulke skielike lewensbedreigende toestande die dokter nie onkant vang nie. Met weinig uitsonderings kan enige ervare geneesheer, huisarts of spesialis op kort kennisgewing oordeelkundig en wetenskaplik optree. Solank ons nooit die moontlikheid van sodanige eensklapsoptredende siektes uit die oog verloor nie en kennis gereeld opknap en bybring, kan elk van ons ons plek as volwaardige dokters volstaan. Maar hoe wetenskaplik tree ons op wat betref die minder belangrike probleme? Ons keer dat die roetinewerk ons waaksaamheid nie afstomp nie, maar maak ons ook seker dat ons wetenskaplikheid gedurende die slepende roetine nie verbrokkel nie?

Indien elke ervare dokter met gereelde tussenposes ongemerk 'n bandmasjien sou aanskakel en sy eie advies aan die pasiënt opneem om dit dan later in die stilte van sy binnekamer terug te speel, en objektief na te dink of wat hy gesê het wetenskaplike kennis weerspieël, sal daar 'n paar onrusbarende ontdekkings gemaak word. Weliswaar is die dingetjies wat ons onder sulke nie-ernstige omstandighede sê, nie so belangrik nie, en die vertroue tussen dokter en pasiënt verseker boonop dat die effense onwetenskaplikheid deur beide partye onondersoekend aanvaar word. Maar die feit bly staan dat iedereen van ons met verloop van jare gewoontes ontwikkel wat met werklik wetenskaplike geneeskunde niks te maak het nie, en as ons die afgesaagde frases gereeld genoeg herhaal, of die roetine handelinkies dikwels genoeg doen sodat hulle werklik tweede natuur word, dink ons naderhand dat ons optrede in ooreenstemming met die beste mediese kennis geskied, terwyl ons in werklikheid maar net gewoontebewegings en -geluide maak.

Somatostatin (Somatotrophin Release Inhibiting Factor)

Early in 1973 the research group of Dr Roger Guillemin of the Salk Institute, La Jolla, California, published the amino acid sequence of a hypothalamic peptide which inhibits the secretion of immunoreactive growth hormone. The peptide was isolated from ovine hypothalami and was shown to have a 14 amino acid sequence with a crystine bridge.[1] This material, which has been named somatostatin or somatotrophin release inhibiting factor (SRIF), was shown to inhibit growth hormone from a number of species in pituitary monolayer cultures. It also inhibited growth hormone release following provocative stimuli in a number of whole animals, including rats. The initial observations of Guillemin and his associates have been amply confirmed in a variety of other animals and in man. At a recent International Conference on Growth Hormone, sponsored by the National Agency of the National Institutes of Health, USA, the current use of somatostatin in man and animals dominated much of the proceedings. The acute infusion of somatostatin has been shown to inhibit growth hormone release following a variety of provocative stimuli including exercise,[2] insulin-induced hypoglycaemia,[3] arginine or L-dopa[4] and the spontaneous elevations of growth hormone in acromegaly.[3,5] It is similarly effective in other animals, including non-human primates[6] and dogs.[7] In these animals somatostatin has been shown to inhibit not only the effects of stress and insulin-induced growth hormone release, but also the nocturnal peaks of growth hormone found 60 - 90 minutes after the onset of deep sleep.

Somatostatin has now been synthesised and is the subject of world-wide study at present. It has, unexpectedly, been shown to inhibit insulin release[8] and also thyrotrophin, but not the prolactin response to thyrotrophin-releasing factor.[3] The physiological implications of these rather unexpected findings are not yet clear.

Will this new and exciting polypeptide be merely of physiological interest, or is it likely to have therapeutic relevance? There may indeed be great use for this hormone in clinical endocrinology. The dramatic effect on growth hormone levels in acromegaly may make medical treatment with somatostatin the logical approach when the pituitary tumour is small, thus sparing the patient the debilitating consequences of pituitary surgery or radiotherapy, with its legacy of panhypopituitarism. This medical approach might be the more appropriate when one considers that conventional therapy in acromegaly rarely lowers growth hormone levels to normal to produce a biochemical remission of the acromegalic status.

Somatostatin may turn out to be of value in the treatment of diabetic retinopathy. There appears to be some evidence that growth hormone may be permissive in the development of diabetic angiopathy—in particular in the retinal changes. The rationale of hypophysectomy in the treatment of advanced diabetic retinopathy is based on this premise. Lundbaek and his group[2] were the first to realise the implications, and have shown that short-term administration of somatostatin dramatically lowers the typically high fluctuating growth hormone response to exercise, characteristic of juvenile diabetics. Of course it is not possible to say what effect the somatostatin might have on retinopathy—at this stage it can only be conjectural. Provided no long-term consequences of somatostatin therapy are discovered—and provided that adequate growth has already occurred, there does not appear to be any major problem to anticipate—this form of therapy may become important in the long-term management of diabetes mellitus.

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