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Ons Mediese Armamentarium

Dokters moet gedurig studeer om by te bly met al die moderne ontdekkings op die gebied van geneeskunde. Daar is feitlik daagliks nuwe en belangrike kennis beskikbaar wat geassimileer moet word. Hierdie gedagte van die voortdurend studerende geneesheer is nie nuut nie—dit is die beeld wat iedere pasiënt van sy hardwerkende dokter het en die ideaal wat alle dosente aan opleidingshospitale aan hul studente voorhou. Dit klink maklik, maar slegs die besige praktisyn weet hoeveel geestesenergie dit verg om na 'n lang dag nog wetenskaplike materiaal te probeer verteer.

Afgesien van die praktiese probleme wat die hoof gebied moet word wat betref nagraadse studie, is dit ewe belangrik dat die dokter nie verstrik raak nie in 'n warboel van nuwe gegewens wat soms nog nie voldoende bewys is nie. Bo en behalwe die plig wat hy teenoor sy pasiënte het om homself op hoogte te hou met nuwe kennis in sy vakgebied, moet hy versigtig kies en keur. Daar is drie hoofaspekte wat oorweging vereis.

Die geneesheer moet besluit of die nuwe navorsingsresultate wat aan hom opgedis word finaal aanvaar kan word. Ons weet almal uit dure ervaring dat bevindings wat skynbaar op die suiwerste wetenskaplike grondslag onweerlegbaar bewys is, op die ou end in die praktyk geblyk het onjuis te wees. Die ervare dokter weet naderhand instinkmatig welke ‚feite' hy as sulks kan aanvaar en welke nog maar met 'n knippie sout geneem moet word. Hierdie oordeelsvermoë onderskei die minder suksesvolle geneesheer van sy briljante kollega. Dit berus op 'n bykans onbewuste sifting van vorige ervaring en sekere waarskynlikheidsfaktore. Sommige kan dit doen; ander nie. Die wat nie oor die vermoë beskik nie moet sorg dra dat hulle slegs nuwe ontdekkings wat die goedkeuring van 'n leermeester of leerskool dra as werklike vooruitgang aanvaar, anders loop hulle gevaar om op 'n dwaalspoor verlore te raak of die prooi te word van sommige onetiese verkoopslui.

Verder moet die nagraadse studerende dokter besluit of die nuwe kennis, al is dit oënskynlik feitelik korrek, wel enige kliniese waarde het. 'n Groot deel van die navorsing wat vandag in die wêreld onderneem word, is van suiwer akademies-wetenskaplike aard en het nie direk op die praktiserende geneesheer enige betrekking nie. Daarmee bedoel ons geensins dat die navorsing onbelangrik is of nie gedoen hoef te word nie. Ons wil slegs beweer dat dit nie vir die besige dokter nodig is om sy reeds karige vrye tyd te bestee aan die bestudering van sulke esoteriese kennis nie. Hy kan dan liefs sy tyd gebruik om kliniese siektebeelde weer na te lees. Gedurende simposiums waar daar referate gelewer word deur laboratoriumpersoneel asook deur klinici, is dit altyd interessant om te merk welke verskil daar bestaan tussen die klinikus wat steeds pragmaties in staat is om sy ewewig te behou, en diegene wat in 'n warboel van halfverteerde feitekennis verstrik geraak het. Dit is laasgenoemde groep wat eers die laboratoriumondersoeke aanvra, en daarna besluit welke kliniese nut die resultate kan dien.

Derdens moet die dokter ook seker maak dat sy agtergrondskennis nie verkwyn nie. Al is sekere nuwe ontdekkings nie dadelik op sy pasiënte van toepassing nie, moet hy tog besluit welke feite miskien op 'n latere stadium as agtergrondskennis kan dien om voorsienbare of onvoorsienbare ontwikkeling te kan verstaan. In hierdie opsig is dit gewoonlik nie slegs nodig dat nuwe navorsing bestudeer moet word nie—reeds voorheen geassimileerde kennis moet van tyd tot tyd opgeknap word. Dit geld veral vir die basiese begrippe waarop ons hele kliniese praktyk berus. Dit is die moeite werd vir selfs die mees ervare geneesheer om nou en dan 'n slag terug te gaan en seker te maak dat hy presies weet wat die uitdrukking pH beteken of wat bedoel word met milli-ekwivalent per liter. Dat dit nie gedoen word nie, spreek uit ons algemene aanvaarding dat die soortlike massa van urine in die omtrek van 1 010 lê. As dit waar is sou 500 ml. urine ongeveer soveel as 'n middelslag-gesinsmotor geweeg het. Wat presies beteken soortlike massa ?

Neonatal Necrotizing Enterocolitis

Neonatal enterocolitis is a dangerous and life-threatening problem in the management of the premature baby. The aetiology of enterocolitis is still in some doubt and apart from its occurrence in premature babies it may be seen in association with aganglionic megacolon, following exchange transfusion, associated with severe infections and with respiratory distress. The presence of abdominal distension and radiological evidence of intraluminal gas in a sick neonate is sufficient for the diagnosis.

Recently several publications have outlined the importance of this condition and emphasized the need for careful and planned management. Early diagnosis and correct treatment is often rewarding and as the long-term sequelae of this condition are related almost entirely to the extent of bowel damage, a vigorous and enthusiastic approach is always warranted.

There is no doubt that bowel ischaemia with subsequent tissue damage and secondary bacterial invasion is the final common pathway in the pathogenesis of this condition. The preceding events are often difficult to determine and are to a large extent unknown.

Two factors are probably important in considering the aetiology of this condition in the premature baby: (i) susceptibility to infection, and (ii) susceptibility to changes in cardiac output and splanchnic blood flow. It is probable that other conditions associated with enterocolitis produce similar changes allowing these two problems to become manifest.

The pathologic changes in the bowel are a result of systemic and local changes in perfusion. The bowel wall of the premature baby is particularly vulnerable because of its thinness and because of the virtual absence of mucosal lymphoid tissue, but it undoubtedly is also immunologically less able to cope with infection. Ischaemia may destroy the mucosal barrier, thereby providing entry into the richly vascular submucosa for virulent organisms or lethal toxins, which would perpetuate local and systemic perfusion inadequacies or perhaps initiate a Shwartzman-type reaction.

Stevenson **et al.**[1] were unable to show any evidence of major vascular thrombosis in their cases and it would seem likely from the clinical progress that transient vascular spasm or splanchnic hypotension is probably the underlying cause of the bowel ischaemia. Once established, the structural damage to the bowel wall is perpetuated by gaseous distension.

The frequent distribution of this disease in the colon may be a result of its bacterial environment and susceptibility to gaseous distension.

The nature of the bowel content has also been stressed as an important consideration. Dehydration of the meconium mass results in an extremely viscous material which may be difficult for the colon to evacuate if bowel function is compromised. Portal hypotension, shock and septicaemia may possibly alter the bowel contents so that they contribute to progression of the disease once the mucosal protection is lost.

The radiological appearance of intraluminal gas results from the loss of mucosal integrity, with distension further contributing to extension of the intraluminal air. Mesenteric vein gas or portal gas represents a later stage in the progression of this process.

If acceptable salvage is to be attained one should be constantly aware of the very early signs of ill health in a premature or any other susceptible infant. Temperature instability, inactivity, vomiting, and only later distension, should all be sufficient to warrant further investigation and vigorous treatment.

Investigation and progress should be assessed clinically and radiologically and acidosis and metabolic deficits corrected. Two-hourly assessment in this way will indicate the trend in the disease and if it is progressive despite antibiotics, fluids and nasogastric suction, surgery is indicated.

This problem is best considered as an end-organ response to ischaemic distension and infection. The host's immunologic, haemodynamic and metabolic status influences the rate of onset and progress.

Treatment must be thorough and if instituted early will often be limited to nasogastric suction, antibiotics and intravenous therapy. Late institution of therapy is frequently associated with progression requiring surgical exploration, resection and colostomy. Careful and frequent examination is always rewarding. There is no place for conservatism in the face of deterioration.

1. Stevenson, J. K., Bell, R. S. and Graham, C. B. (1971): Amer. J. Roentgenol., **112**, 123.