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VAN DIE REDAKSIE *EDITORIAL*

Opleiding in die Etiek

Op die keper beskou, is dit eintlik jammer dat hoegenaamd enige opleiding ten opsigte van etiese optrede in die geneeskunde nodig is. Korrekte gedrag kan in 'n groot mate beskou word as 'n samesmelting van gesonde verstand en goeie maniere, en enigeen wat hom dit werklik eerlik en ernstig ten doel stel om gesonde, streng etiese geneeskunde te praktiseer, behoort min moeilikheid te ondervind met die fyner detail van korrekte optrede. Iemand het trouens eenkeer gesê dat as 'n dokter aan die Geneeskundige Raad skryf om vir 'n eksemplaar van die etiese reëls te vra, moet hy streng dopgehou word—miskien soek hy na skuiwergate.

Nogtans is dit waar dat daar sekere aspekte van praktykvoering is wat miskien nie so voor-die-handliggend is nie, en wat 'n mate van toeligting vereis. Dit is die dure plig van ons mediese skole om toe te sien dat sulke opleiding nie agterweë bly nie. Dit is waar dat kursusse in mediese etiek wel by die meeste fakulteite aangebied word, maar in sommige gevalle is hulle werklik maar skamel en ontoereikend wat al die aspekte van die gedragskode en etiese slaggate betref.

Die Geneeskundige en Tandheelkundige Raad het gedurende sy vergadering van 27 tot 29 Mei in Kaapstad besluit dat die saak weer onder die aandag van die mediese beroep gebring moet word, aangesien die Raad met kommer bewus is van tekortkominge in die etiese onderrig aan sommige van ons sentrums. Ons voldoen met graagte aan die versoek van die Mediese Raad om hierdie aangeleentheid aan ons lesers te stel.

Wat behoort sulke opleiding in die etiek te behels? Daar is twee groot afdelings waarin die toeligtingsmateriaal gegroepeer kan word: ten eerste inligting aangaande die vereistes van die Wet soos vervat in die Regulasies, en ten tweede 'n meer algemene motiverende uiteensetting van die hoë ideale en standaarde van die mediese beroep. Om so 'n kursus betekenisvol te maak, is beslis meer as net 'n handjievol lesings nodig, en dit is waarskynlik aan te beveel dat die lesings deur meer as een persoon aangebied word, anders word onwillekeurig die indruk geskep dat die streng standaarde wat verkondig word net vanuit een bron ontstaan, eerder as die breë siening van die hele beroep te wees.

Wat die algemene samesnoerende motivering tot hoë standaarde betref, kan mens seker nie beter vaar as om die Eed van Hippokrates as uitgangspunt te gebruik nie. Die bepalinge het gedurende die meer as tweeduisend jaar nie verander nie, want, soos N. P. van Wyk Louw dit gestel het: 'Die waarheid is enkeld en oud'. Die indruk wat hierdie onderrig op die voorgraadse student gaan maak, sal afhang van die persoonlikheid van die lektor, en daarom het hy 'n werklik uiters verantwoordelike taak, want faal hy, sal hy aandadig wees aan die skepping van 'n groep studente wat vir die res van hul akademiese loopbane mank sal gaan aan 'n onewewigte oordeel oor die kernbelangrikheid van etiese optrede.

Dit is altyd gevaarlik om kategoriese uitsprake te lewer, maar dit skyn tog asof 'n kursus wat meerdere jare duur, aangewese is ten einde te verseker dat die studente stap vir stap, soos hul ontwikkeling in die mediese wetenskap vorder, ook die meegaande ontluiking van hul etiek kan ervaar.

Drugs Used in Hyperuricaemia

Drugs are available to decrease hyperuricaemia. They include the uricosuric drugs such as probenecid and benzbromarone, which increase the excretion of uric acid, and allopurinol, which diminishes the production of uric acid. They decrease the urate content of the body.

Allopurinol inhibits xanthine oxidase, the enzyme which catalyses the conversion of hypoxanthine and xanthine to uric acid.[1-4] The serum urate levels and the output of urates in the urine are decreased, but the drug can be used with a uricosuric agent in refractory hyperuricaemia,[4] and a high output of neutral or alkaline urine should be maintained to keep the xanthines in solution.

Allopurinol is useful in primary gout where uricosuric drugs have not produced adequate control, and in patients with damaged renal function and uratic calculus or gravel. It may be used in gout secondary to treatment of malignant disease.

Acute attacks of gout may be precipitated in some patients at the beginning of treatment. Nausea, vomiting, diarrhoea, skin rashes and fever sometimes occur, as well as leucopenia and jaundice, which disappear when treatment is stopped. Allopurinol interferes with production of nucleotides, and is incorporated into nucleic acids. The long-term effect is not yet established, and the selection of this agent for initial hypo-uricaemic therapy is still controversial.[3]

Probenecid is a uricosuric agent,[1-4] which increases the excretion of urate by blocking reabsorption of urate by the renal tubules. Since the action is blocked by salicylates, it is of no value in acute gout. Probenecid produces a fall in serum urate within 48 hours, joint movement is progessively improved, pain subsides, and tophi may regress. The urine should be kept alkaline with potassium citrate, 1 g 3-6 times daily, to prevent the formation of uric acid stones.

Sulphinpyrazone is a derivative of phenylbutazone which diminishes the reabsorption of urate by the renal tubules.[1-4] The action is impaired by salicylates and citrates and, as with probenecid, it has no place in the treatment of acute gout. The potential toxicity of this drug is high. It may produce acute gout, epigastric discomfort, and it may activate a peptic ulcer. Leucopenia and thrombocytopenia may occur, so that blood counts are advisable. These effects are not unexpected since the drug is related to phenylbutazone. It is contra-indicated when there is impaired renal function.

Salicylates interfere with uric acid secretion at low dosages (less than 3 g/day) and with both secretion and absorption at high dosages (more than 4 g/day), i.e. they reduce urinary uric acid and increase serum uric acid at low doses, and have the opposite effect at high doses. They are not important uricosuric drugs and can block the action of probenecid and sulphinpyrazone.

Benzbromarone is a new uricosuric agent which produces a rapid and prolonged decrease in serum uric acid levels, which is demonstrable within a few hours after oral administration of the drug.[5,6] A reduction of 50% of the initial blood urate level is generally obtained during the first 10 days of treatment; the effect continues for some days after withdrawal of the drug. Gouty tophi and pain are diminished and mobility improved, and the action of this agent is not antagonised by salicylates. This uricosuric drug is indicated in the treatment of chronic (primary) gout, and in hyperuricaemia occurring in malignant disease or during administration of oral diuretics or certain other drugs. Adequate diuresis and alkalinisation of the urine must be maintained during administration. As with other uricosuric agents it is contra-indicated when there is marked renal insufficiency or in the presence of renal calculi.

The acute arthritis of gout is treated conventionally with rest, local measures and the anti-inflammatory drugs. Colchicine or another suitable anti-inflammatory drug may be given concomitantly with one of the hypo-uricaemic drugs during the first few days of treatment to avoid an attack of acute gout, which is always possible at the beginning of treatment of hyperuricaemia and gout. This risk can be reduced by initiating therapy with large quantities of fluid and alkalinisation of the urine.

The metabolic and inflammatory aspects of gout are distinct processes; the former may or may not be accompanied by the latter. Treatment must in fact be focused independently on these two aspects; in many instances therapeutic agents for controlling one process influence the other as well.

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