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EDITORIAL : VAN DIE REDAKSIE

CURABLE HYPERTENSION

Hypertension is a very common condition and a number of its causes are eminently curable, if detected. This is now constituting an interesting diagnostic challenge to doctors in general practice and at hospitals.

The diagnosis of coarctation of the aorta may be suspected by two easy clues. A young hypertensive may have impalpable pulses in the legs. Or, if the leg pulses are palpable, the systolic pressure is lower than in the arms as measured by the simple sphygmomanometer-cuff method.

The diagnosis of renovascular hypertension remains incompletely clarified and the results of surgery are variable. Belfast workers recently reviewed a series of cases.[1] They considered the intravenous radio-hippuran renogram a safe, simple, and reliable screening test for the detection of renal artery obstruction. The only useful clinical feature in their cases was an abdominal bruit present in 63% of patients with major renal artery obstruction.

Cushing's syndrome may be extremely easy to diagnose if many typical clinical features are present—hypertension, round ruddy face, obesity, diabetes mellitus, purple striae, osteoporosis, etc. The diagnosis of such cases, and of less typical cases, may be confirmed by abnormally high levels of plasma and urine 17-hydroxycorticosteroids (17-HCS). Forsham *et al.*[2] have now described a rapid screening test for Cushing's syndrome, based on the ability of dexamethasone to suppress plasma 17-HCS levels. One mg. dexamethasone is given orally at 11 p.m. On the next day, a single morning 17-HCS-value of less than 5 μg./100 ml. should exclude Cushing's syndrome.

Phaeochromocytoma may present with clear-cut paroxysmal symptoms and 'classical' paroxysmal hypertension. Unless the possibility of phaeochromocytoma is kept in mind, some cases may inadvertently be labelled as 'menopausal' or 'neurotic' when they complain of shivering, feeling cold, warm flushes, and nervousness. Other cases present with sustained elevation of the blood pressure, resembling essential hypertension. In spite of such diagnostic techniques as peri-renal air insufflation, the newer pharmacological agents for provocative and for blocking tests, the assay of urinary pressor amines and their metabolites (VMA), the diagnosis may be established only by exploration.[3]

Conn's syndrome (primary aldosteronism) is currently the centre of a great resurgence of interest and excitement. The association of hypertension with muscular weakness and low serum potassium (hypokalaemia) has been the usual clue to the diagnosis. However, the diagnostic scope has greatly widened recently. Conn is now suggesting that some patients may have normal serum potassium and masquerade as 'essential' hypertension—normokalaemic primary aldosteronism.[4] In fact he speculates that possibly 15% of the vast population of 'essential' hypertensives may have the curable condition of primary aldosteronism.[5] He also makes the extremely interesting suggestion that large numbers of patients with apparent 'essential' hypertension combined with 'maturity-onset' diabetes mellitus have in fact got primary aldosteronism, and that they could be cured of both diseases after they have been diagnosed properly![5] The important investigations used by Conn are the demonstration of excessive aldosterone production accompanied by severe suppression of plasma renin activity.

Presently-available techniques for estimation of renin and aldosterone are sufficiently difficult and cumbersome to prevent direct knowledge of the true incidence of primary aldosteronism in our hypertensive population. Nevertheless, until such tests have been carried out, all patients with unexplained hypertension must be regarded as potentially curable by means of surgical removal of a small aldosterone-producing adrenal tumour.[4] In South African literature, the first such case due to an adrenal cortex adenoma was reported by Eales and Linder in 1956.[6] The second such case to be reported[7,8] is described by Drs. L. Stein, N. Shapiro and D. Stein on page 993 in this issue of the *Journal*. One of the values of case presentations at clinical meetings, and case reports in journals, is the stimulus they may give to colleagues to detect similar cases in their practices or hospital work.

1. Fenton, S. S. A., Lyttle, J. A. and Pantridge, J. F. (1966): Lancet, **2**, 117.
2. Pavlatos, F. C., Smilo, R. P. and Forsham, P. H. (1965): J. Amer. Med. Assoc., **193**, 720.
3. Harrison T. R. (1962): *Principles of Internal Medicine*, 4th ed., p. 1351. New York: McGraw-Hill Book Co.
4. Conn, J. W., Rovner, D. R., Cohen, E. L. and Nesbit, R. M. (1966): J. Amer. Med. Assoc., **195**, 21.
5. Conn, J. W. (1965): New Engl. J. Med., **273**, 1135.
6. Eales, L. and Linder, G. (1955): Quart. J. Med., **100**, 539.
7. Stein, L., Shapiro, N. and Stein, D. (1966): S. Afr. Med. J., **40**, 993.
8. Bickersteth Medical Society (1966): *Ibid.*, **40**, 298.

DIE WERK WAT ONS DOEN EN ONS GEESTESGESONDHEID

Die geneesheer wat sy taak benader in terme van omvattende gesinsgeneeskunde, stel uit die aard van die saak in baie meer belang as net in die siektes waaraan sy pasiënte onderhewig is. Die hele mens, en veral ook die soort werk wat hy doen—omdat dit 'n deurslaggende uitwerking het op beide sy liggaamlike en geestelike welsyn—kry dus vir die geneesheer 'n nuwe betekenis.

As ons nou dink aan al die moontlike faktore in die lewe wat kan meehelp om ons algemene welstand te bevorder, dan is die werk wat ons doen seker een van die belangrikste van hierdie faktore. Die rede waarom dit so is, is nie moeilik om te verstaan nie, aangesien die werk wat ons doen ons hele lewenstyl bepaal en omdat dit nooit ophou nie. Ons bly bewustelik of onbewustelik daarmee besig al is ons saans by die huis of al is ons ook weg met vakansie.

Ons verdien ons leeftog deur die werk wat ons doen. Ons bestee 'n groot deel van ons liggaamskragte en geesteskragte daaraan. En ons werk maak vir ons die verskil tussen selfstandigheid en afhanklikheid en tussen goeie

aanpassing en wanaanpassing. Dit kleur ons hele lewe.

Om hierdie rede is dit dan ook so jammer dat so 'n groot aantal van die mense met wie die geneesheer daagliks in aanraking kom, nie geluk en vreugde in hulle werk vind nie. Uit die aard van die lewensomstandighede waarin ons ons bevind, het skeppingsvreugde en arbeidsgenot vir baie mense uit hul werk verdwyn sodat hulle werk vir hulle 'n las in plaas van 'n lus geword het.

Nou is dit wel die geval dat dit dikwels, weens die aard van die samelewing van vandag, nie vir elkeen moontlik is om dié soort werk te doen waarvoor hy aanleg het of ook waarin hy belang stel nie. Baie soorte werk is eentonig en vervelig. Dit is 'n feit wat ons nie kan ontken nie. Maar ons kan ons nogtans nie van die waarheid losmaak nie dat die gesindheid wat ons teenoor ons werk het, 'n deurslaggewende invloed uitoefen op ons geesteswelsyn.

Waarom is die werk wat mense doen in so baie gevalle nie vir hulle bevredigend nie? Een van die eerste redes waaraan ons dink is die feit dat mense so baie van mekaar verskil wat betref hul psigiese en liggaamlike energie. Baie mense moet gedurig meer doen as waartoe hulle in staat is, en dit maak hulle ongelukkig, ontevrede en siek.

Verder is daar die aard en implikasies van die werk self wat mense moet doen. Die moderne, geïndustrialiseerde samelewing bring dit mee dat die werk wat gedoen moet word dikwels sonder diepere betekenis vir die arbeider self is, omdat hy maar net in 'n klein onderdeeltjie van die hele taakgebied ingeskakel is.

Verkeerde opvoeding en valse waardes speel ook 'n groot rol by wanaanpassing in die werk. Een van die gulde reëls van die geestesgesondheid is dat 'n mens die kans moet kry om dié soort werk te doen waarby jou talente en vermoëns aansluit. Die vermoëns van die kind word vandag nog nie genoeg in ag geneem wanneer ons kom by die keuse van sy werk of beroep nie.

Watter praktiese stappe sou ons nou in die lig van die voorgaande oorwegings kon doen om mense meer bevrediging in hul werk te laat vind? Ons kan tog nie die hele aard van die samelewing waarin ons leef eensklaps verander nie. Maar dit beteken nie dat ons niks aan die saak kan doen nie.

In die eerste plaas is dit belangrik om die rol van toewyding en ywer te beklemtoon. Dit maak nie saak wat ons doen of waar ons werk nie; die vraag is: hoe goed doen ons ons werk? Aan goeie en betroubare arbeiders het die wêreld altyd behoefte.

In die tweede plek moet die gesindheid van verantwoordelikheid en selfrespek aangekweek word. Dit kan begin word deur ouers en onderwysers van kinders en later voortgesit word deur hul mediese raadgewers.

In die derde plek moet daar 'n poging aangewend word om, in 'n nog groter mate as wat dit wel gedoen word, aandag te gee aan betroubare en wetenskaplike beroepsleiding. Veral in 'n land soos dié waarin ons leef, is elke geskoolde en goed-aangepaste werker 'n belangrike belegging vir die toekoms.

By al dié praktiese oorwegings, is daar egter ook nog 'n addisionele oorweging wat van die grootste belang is. Die groot humanis, wyle dr. Albert Schweitzer, het eenmaal gesê dat elke mens, behalwe die werk wat hy doen waardeur hy sy bestaan voer, ook nog 'n tweede taak moet beoefen, nl. daardie bydrae wat 'n mens bo en behalwe jou gewone werk lewer in terme van belangelose diens aan mense persoonlik of aan die gemeenskap in die algemeen. Want die gelukkige en werklik gesonde mense is die doeners en die gewers. Dit is hulle wat die lewe en mense met welwillendheid tegemoet gaan, wat die beste kans het op die verwerwing van daardie onaantasbaarheid van gees wat die sleutel is tot nie alleen liggaamlike welstand nie, maar ook geestesgesondheid.

Dit is die soort werk wat ons op onbaatsugtige wyse in diens van die mensdom verrig—en waarvoor ons nie betaal kan word nie—wat op die duur die grootste bevrediging vir ons bring. En in die mate wat ons daarin slaag om hierdie tweede taak met welslae en toewyding te volvoer, sal ons in staat wees om die werklike betekenis te peil van dié ou spreuk: .Die arbeid waarin ons 'n lus het, heel die pyn.'

ABNORMALITIES OF SACRUM AND FEMUR IN INFANTS OF DIABETIC MOTHERS

It must be considered as reasonably certain that congenital anomalies are more common in infants of diabetic mothers than infants of normal mothers. Only recently has it been noticed that a particular kind of anomaly is seen with especial frequency.

Pedersen *et al.*[1] from Copenhagen, in 1964, suggested that defects of long bones were commoner in children of diabetic mothers than in controls. These authors found seven such cases in their series; hypoplasia of one or both lower limbs was present in all, with or without sacral and other defects. Passarge and Lenz[2] have now collected 43 cases from the literature in which the affected infants have an agenesis of the sacrum and coccyx and/or malformation of the lower limbs (most commonly hypoplastic femora). Malformations of the upper limbs were seen in only two instances, renal agenesis in four, and other occasional defects included abnormalities of tibia and fibula, dislocated hips, cleft palate and congenital heart lesions.

This clinical picture has been termed the 'syndrome of caudal regression', and may perhaps be related to the sireniform monsters, which have hypoplasia of the lower limbs and ano-urogenital anomalies. The syndrome is not common, but may occur in about one percent of infants born to diabetic mothers. On the other hand not all affected infants have mothers who are diabetic, in fact the incidence of maternal diabetes is probably around sixteen percent.[2] This figure is based on seventy-two cases of sacral agenesis, ten of whose mothers were known to be diabetic at the time of their pregnancies. The connection with diabetes may well be closer, since in a few instances the mothers only developed diabetes some time later.

Passarge and Lenz[2] point out that insulin has been shown to induce defective development of the tail skeleton in chickens, but not all the diabetic mothers had received insulin so that this certainly cannot be generally incriminated in the human syndrome. In one family both a father and his daughter had no sacrum, but there appears to be no other evidence of familial incidence.

1. Pedersen, L. M., Tygstrup, I. and Pedersen, J. (1964): Lancet, **1**, 1124.
2. Passarge, E. and Lenz, W. (1966): Pediatrics, **37**, 672.