EDITORIAL

NEUROLOGY OF AGING

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Of all systems in the human body the nervous system is most vulnerable to aging and accounts for 50% of disabilities beyond the age of 65 years, ie. in the elderly (1). It is exclusively post-mitotic and cannot be replaced (2). The problem is compounded in Singapore in that with the aging of the post-World War II babies and a current small family nucleus, by the year 2030 a good 20% of the population will be over 65 years. However, a positive outlook should be taken by society and health planners. Dendrites and neurotransmitters could be modified and manipulated pharmacologically. Moreover, the pathogenesis of so called degenerative diseases is fast being unravelled. This article touches on the issues which distinguish normal nervous system changes from the abnormal and covers broadly the therapeutic hopes.

It is generally accepted that mental cognitive function declines with age (3). On the Wechsler Adult Intelligent Scale (WAIS) only half correct answers are required for the 75 year old individual when compared to age 21 years to achieve an age corrected IQ of 100. However, note that the depressive elderly patient may present as a cognitive disorder or pseudo-dementia.

Sleep pattern changes. They spend more wakeful time in bed and awake more frequently. Stage 4 sleep decreases and this decrease starts as early as the age of 30 years. REM sleep remains unchanged (4). An understanding of the mechanisms of the sleep cycles may unlock some of the mysteries of the biological clock within us.

Cranial sensory functions are significantly altered. Visual accommodation for near object is impaired and so is distant vision (presbyacusis). Lenses tend to yellow impairing visual acuity. Visual evoked potentials tend to delay by about 10-30 msec. Pupillary responses decrease. Hippus is no longer seen. Saccadic eye movements slow down. Upward gaze is less (normal up to 3mm shift) (5).

Critical flicker of vision frequency declines. Embedded figures are less easily extracted from confusing pictures. Hearing starts diminishing around 50 years especially for high frequency. Other cranial nerves probably remain intact. It is felt that both the attrition of visual and acoustic senses could be less if we are exposed to less noise exceeding 80db and less strain imposed to the eyes e.g. prolonged reading and TV viewing. Otolith degeneration adds on to a sense of imbalance and the elderly are prone to multisensory dizziness i.e. multiple causes for dizziness (6, 7).

The fallout of these sensory systems alone argues strongly in favour that the elderly in our build-up and vehicle intense society should undergo frequent driving tests e.g. at 3 yearly interval.

Gait is particularly vulnerable. The normal adult gait changes to a hesitant broad-based small stepped gait with some features of Parkinsonism, often including stooped posture, diminished arm swing, slight ataxia and hesitancy of steps (8-10). Rarely do individuals above 75 years walk without one of these stigmata of age that I mentioned. The differences between normal and abnormal is one of degree and social function impairment. These could be attributed to fallout of the frontal lobes beyond area 4. We under-utilise the frontal lobe as intelligence, vision, hearing, vegetative functions and posturing are all served by other areas of the brain. Diminution of muscle bulk, slowing of conduction velocities probably do not contribute to gait dysfunction. However mechanical stiffness, arthritic joints and loss of elastic tissue do contribute to clumsiness. Some of these factors are avoidable by regular stretching and aerobic exercises. Tremor should not be considered concomitant with aging as commonly felt by lay and medical circles. It is to be regarded as a dysfunction.

Ankle jerks may diminish in 10% individuals although this is now contested. 5% of the aged may have upgoing or neutral plantar response. Superficial abdominal reflexes are absent in obesity and multi-parity. Suck and grasp reflexes occur in 5% of individuals. If all or more than 2 signs are present frontal lobe disease should be suspected (11).

Male sexual function remains intact. The electrical bulbocavernosus reflex (12) remains intact throughout age in man. Overlaps are considerable. Men who are athletically active especially in racquet games maintain

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better reflexes than sedentary normal 20 year old individuals (13-15).

Pain sensation remains intact. Vibration sense fall out after age of 50 years. Cortical sensation eg. stereognosis or graphesthesia remains intact to the end (16).

Brain weight declines 5-10%. Ventricular and sulci enlargement occurs 2° to atrophy. In the absence of atrophy, ventricular enlargement with periventricular infiltrate is suggestive of normal pressure hydrocephalus. This condition is potentially treatable by ventriculo-peritoneal shunting. Nerves drop out at rate of 50,000 – 100,000/day. 30% – 50% loss of cortical nerves occur in temporal lobe. Dendritic branches fall out. Senile plaques and neurofibrally tangles appear but not in the hippocampus (always pathological). No changes occur in peripheral nerve fibres and brain stem nuclei (17-19).

There is considerable overlap and variation in opinion as to the degree of CT scan detected atrophy which would be considered as pathological. In one study 60% of demented patients had the same degree of atrophy as 15% of normal patients. Alzheimer's and dementia should not be a CT or MRI diagnosis. The cervical spine degenerates in 80% of individuals above 65 years and 100% at 75 years of age (20, 21).

Cerebral blood flow diminishes. There is increase transmission time. Evoked potentials tend to delay slightly especially the late responses e.g. P300 and other events related potentials. A decrease of cortical cholinergic receptors, diminished GABA and catecholomines are noted. Certain synthetic enzymes and monoamine oxidase are increased. Trace elements increase with age. Viruses, prions are present and may cause subtle damage. There is transcription failure of DNA to RNA. Diminished energy metabolism and accumulation of lipofusion occur.

THERAPY AND THERAPEUTICS

Dosage of drugs need to be altered as all support system declines. Drugs should be kept minimal inspite of multiple complaints. Opiates (Opium) empirically from my observation, have a protective effect on neural attrition which leads to Alzheimer's, Parkinson's and related diseases. However opium has other deleterious effects. Endogenous opiate receptors have an important undetermined role. Type B monoamine oxidase inhibitor (selegiline hydrochloride or L-deprenyl) is being tried in the prevention of brain degeneration (21). Cerebral calcium channel blockers e.g. nicardipine, nimodipine and flunarazine may retard atherosclerotic and neuronal attrition (22). Preliminary studies in rats and recent clinical studies show that if these drugs are given early in the course of atherosclerosis the process may regress by their action on endothelial lining, vasospasm and prevention of smooth muscle migration and profileration. Gingko biloba extract may exert positive effects by its action on free radicals which are involved in the pathogenesis of neural damage. The drug is remarkably safe and has significant action in patients with dizziness, chronic vascular headaches and early memory dysfunction (23).

Healthy mental state, attitude and adequate sleep are fundamental factors. Abstinence or moderation in social habits contributes e.g. not more than 1 pint of beer (15 gm of alcohol) or its equivalent per day and not more than 5 cigarettes per day. Keeping the mind active and continuing active learning encourages dendritic profileration. Careful graded exercises e.g. aerobics and light jogs improve gait, joint flexibility and agility.

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