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College of Syntonic Optometry

NEUROGENIC HYPERTENSION

Classically we were taught that a hyper tensive retinopathy was:

(a) Very serious, especially in young individuals.
(b) Etiology – unknown.
(c) Associated with it is diffuse arteriosclerosis.
(d) Retinal lesions caused by angiospasm leading to angiosclerosis.
(e) Ophthalmoscopic signs.
    1. Slight narrowing of arteries.
    2. If progressive there is sclerosing of arteries with indentation of veins at points of crossing.
    3. Engorgement of veins.
    4. Flame shaped and rounded retinal hemorrhages.
    5. Edema – grayish haziness of nerve head.
    6. Cotton-wool patches of exudate.
    7. Star shaped pattern around macula due to edematous condition.
(f) Visual disturbance depends largely upon location of hemorrhages in retina and amount of edema.
(g) Visual disturbance may be first symptom which brings patient to practitioner.
(h) Prognosis is poor if neuro retinopathy is advanced. Most patients dying of renal insufficiency.

"But as I wondered about this problem, I thought, if I ever see a patient like that, there will be little I or anyone else can do.

What constitutes an essential hypertension? Stedman's Medical Dictionary defines hypertension as supertension and a synonym for hyperpiesis, meaning high blood pressure. But will this ever justifiably give an insight into a so-called hypertensive patient. No insight will actually be gained by anyone using a sphygmomanometer and saying, "your blood pressure is a little high." Why? Because hypertension, like any other disease or disturbance of a human being affects that organism totally: not just in his renal system or cardio-vascular system but in his total organismic makeup. True the systolic and diastolic pressures are findings, when persistent, but they alone can no more diagnose a hypertension than our No.7 can diagnose a patient's visual problem.

Since retinal arteriosclerosis seldom precedes hypertension and most generally follows it, its basic cause, or at least a co-ordinate cause must be neurogenic. The aim here in treatment is # 1. To find the emotional cause of elevated pressure and # 2. To provide normal hygienic measures to prevent the disease from progressing further causing an accelerated arteriosclerosis and all of its morbid signs. Among all patients and in fact among all people, there is a certain anxiety connected with the heart, that is to say, most all patients will accept a diagnosis of a liver dysfunction, a kidney involvement or a stomach disorder, but not the heart. This stem from the fact that for all time in the lives of men the heart has had literary and romantic reference which denoted its normal function as the very essence of life itself. Therefore, the primary step is psychotherapy. In psychotherapy the disease is explained to the patient so that there will be an emotional acceptance of their plight. Failing this may be enough to cancel the effect of any or all other treatments. Secondly, the patient's physical condition must be made as good as possible. This can be accomplished by prescribing a proper diet and a way of life compatible with this patient's state. This "way of life" can be highly ramified and depend upon each individual's emotions and financial abilities. It is best practice to lean toward moderation instead of stopping completely some form of work or play which the patient may be used to and enjoy. Diets are a point I even hesitate to mention because I realize many of you are far more proficient than I, in this field. However, two schools of thought do exit: #1, low protein and #2, salt free. Low protein may have some action in lowering arterial tension. Here again the diet must be balanced because the hypoproteinemia induced by negative nitrogen balance will advance edema in

imminent congestive heart failure. Salt free diets are common place among hypertensives but their value is uncertain. Fisberg believes that the impalatability of salt free diets voluntarily reduces caloric intake. Alcohol and tobacco again introduce much controversy but within reasonable limits there is little effect they can have. In fact, if either relieves psychic tension, they can be considered therapeutic.

In examining a hypertensive, the prognosis is determined by discovering what adjustments the major vessels and the heart have made. Cardiac hypertrophy can be determined by inspection and palpation of the chest wall in order to find the apex beat. In ventricular hypertrophy the ventricle moves laterally and posteriorly causing the heart to be tipped toward the chest wall. Finding of apex beat at the sixth intercostal space will provide evidence of moderate hypertension. Auscultation of the ventricular contraction will in hypertension produce a loud low-pitched prolonged sound: contrasted with the slapping short character sound of mitral stenosis. Protodiastolic gallop is heard in the hypertensive in the left lateral decubitus position and is significant of myocardial weakening. If the pulse is 100 or more there will be overlapping of the normal auricular sound and it is the summation of sounds that produce the gallop rhythm. At the base of the heart the loud aortic second sound is contrasted with the pulmonic second sound and is an indicator of increased diastolic pressure. As adjustment is lost the pulmonic second sound increases in intensity and indicates increasing failure of the left ventricle.

All treatment of early essential hypertension is prophylactic. That is to say the aim is to attempt to stop or arrest arteriosclerosis. This can be done by diet and psychotherapy as mentioned previously and will reduce the strain on the acute branchings and unsupported arterial points. If hyperlipemia is found to be present in persistently greater amounts than 25-mg. per 100cc. its treatment is needed to aid in stopping arteriosclerosis. This may mean insulin will be enough to arrest hyper-arteriosclerosis in diabetes: however, it may not all be that simple to control plasma lipids. It might be well to mention that experiments have shown dietary fats not to be a relation of plasma lipid. The time-honored standby of treating atherosclerosis is iodine. However, except in the formative stages the aid is doubtful. Early dietary measures are taken to decrease metabolism and reduce excess weight in order to maintain cardiac efficiency and halt cardiac hypertrophy. Cardiac efficiency is increased by digitalis by maintaining chemical constituents of myocardium. However, the administration of digitalis may in some cases provoke angina in patients not in cardiac failure.

In the brain itself, hypertension causes consequences of hemorrhage, thrombosis, vertigo and headache. These consequences are mediated by the cerebral arteries, which are weak and thin walled. The more vulnerable vessels are the posterior communicating, anterior or middle cerebral arteries, near the circle of willis. Since cerebral vessels are weak when constriction is found elsewhere in the body dilatation is found in these arteries.

Vertigo may result from a large variety of stimuli but one applicable to us is stimulation of ocular-muscles. Spontaneous vertical nystagmus may result from a lesion of the brain stern.

The relationship of headache and increased pressure is not a direct one in that it only occurs in about 50% of hypertensives. Also, headaches do not result from increased intercranial pressure. The seat of the pain is thought to be located in the blood throbbing on pain end organs in the dilated vessel walls. This pain originates in branches of the external carotid hence manual pressure on the temporal, frontal, supraorbital, postauricular or occipital arteries will decrease headache.

Renal circulatory activity has for a century been the seat of the primary causal unit of hypertension by some authorities. But since the investigation of an endocrine function to the kidney the picture has changed.

Angiotonin is formed by the action of renin, a proteolytic enzyme present in the renal cortex and substate which is produced in the liver. This polypeptide constricts smooth muscle and acts especially on the arterioles, arteries, and myocardium. In the normotensive injection of angiotonin will produce clinical symptoms; however, in injection of angiotonin will produce clinical symptoms; however, in hypertensives this is not a rapid change and adjustment is made to it. Therefore, it is now advanced that a beginning "neurogenic" hypertension will eventually result in renal dysfunction, due to either prolonged vasoconstriction or more probably of arteriosclerosis.

Where does all of this lead us? I have endeavored to put across the idea that hypertension, in the very beginning, is due to a disturbed balance of the nervous system. Now, syntonically, we can provide syntony of the autonomics. Therefore, we have in our power to decrease the rapid increase of neurogenic hypertension.

In our offices we have used syntonics for several years now, and feel that we do aid the hypertensive. The research in these cases, we feel, will be greatly advanced with the purchase of a B & L. ophthalmoscopic camera. With it, we will be able to follow advance retinal change, regardless of how minute they might be. This will also assist the several physicians with whom we consult and are consulted by in hypertensive cases. There will be no more guessing as to whether this or that vessel has changed or if a hemorrhage has increased in size.

In conclusion I wish to express my thanks to Dr. Gallagher and D. Scott for their insistence that I prepare a paper for presentation at this meeting. Also, in particular I express appreciation to my father, Dr. G. K. Miller for his aid during many discussions relative to neurogenic hypertension.

To the College of Syntonic Optometry, I am deeply indebted for the welcome shown to me during the 1950 National Assembly at Chicago. This experience afforded me a broader view of optometry and the opportunity to be of greater service in my community.

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