THE TROPHO-NEUROSES OF PARETIC DEMENTIA.*

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In presenting for your consideration a brief review of the vaso-motor abnormalities exhibited in the clinical history of paretic dementia, I do so, not that I am able to offer anything new, but to accentuate the important part which neuro-angio-paralysis plays in the production of many of the most interesting phenomena of this dreaded malady.

The vaso-motor and trophic changes, which are observed in both the early and late stages of the disease, are characteristic and important. Glancing hastily over the clinical picture presented by the early stage of paretic dementia, we find an array of symptoms, some of which, if not all, are found in every case. Among these may be mentioned vertigo, flushing of the face, and other evidences of transient anæmia and hyperæmia of the brain; alterations in the tension of the pulse, it being high in the more active forms of the disease and low in the depressive form (Spitzka), frequent attacks of headache, associated with a sense of pressure in the head; occasional slight apoplectiform attacks, and many other symptoms familiar to all who have observed cases of paretic dementia in their incipency. These symptoms are, however, evanescent in character, failing to furnish satisfactory evidence of structural changes in the interstitial or parenchymatous tissues of the brain, they are explicable only on the hypothesis of vaso-motor paralysis. The mental symptoms at this stage of the disease also bear evidence of very slight cerebral disturbance, and are accounted for more satisfactorily by the theory of functional circulatory disturbances than by the belief that real structural changes in the cerebral tissues have occurred.

The slight impairment of the mind, which makes its appearance and progresses so slowly; the sudden attacks of mental confusion, synchronous with the flushing of the face and the attacks of cerebral congestion; the blunting of the finer sensibilities, the mild exhilaration, the abnormal development of the Ego, the unnatural changeableness of the moods and temper of a formerly well-balanced

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individual; the irritability and the frequent attacks of excitement or melancholy, are suggestive of vascular abnormalities and of vaso-motor disturbances. It is in this prodromal stage that Meynert, Folsom, and others believe that the line of demarcation between functional and organic diseases can be drawn; and by proper treatment and regimen the malady be stayed in its progress, if not actually cured. Later in the history of paretic dementia, we have the episodic attacks of epileptiform and apoplectiform seizures which not infrequently, after a few days of mental and physical impairment, result in the patient emerging from the attack with apparent rejuvenation of mental power. These attacks are often recovered from too quickly to have been due to any inflammatory process, as is claimed by some writers.

As the disease advances, the progressive impairment of mental vigor, the frequency of profound apoplectiform and epileptiform attacks, the marked ataxic and other symptoms of progressive paralysis of all striated muscles, mark the era of more serious brain changes. We now find the vaso-motor and trophic abnormalities presenting themselves in more tangible form. The vaso-motor paralysis is evidenced by changes in every tissue of the body. The oedema of the feet and limbs, hyperidrosis, malnutrition of the cutaneous surfaces, localized anæsthesias and hyperæsthesias, herpetic eruptions following the course of branches of the trigeminus, brittleness of the hair, sponginess of the gums, ulceration of the mucosa of the cheeks, acute decubitus, neuro-paralytic congestion of the lungs, othæmatoma, sub-dural hæmatoma duræ matris, etc., all are consequent upon trophic changes. Post-mortem, the findings are still indicative of grave vaso-motor changes, which, by long-continued disturbances, have produced organic lesions of the cerebral structures. These may be briefly summarized as follows: An atrophy of the brain as a whole or in part, degeneration of the cells and neuroglia, of so pronounced a character as to lead Clouston to speak of general paresis as "essentially a death of that tissue, and equivalent to a premature and sudden senile condition; senility being the slow physiological process of ending, general paralysis the quick pathological one."

The vascular changes are as important as constant, the vessels of the pia and cortex being tortuous, looped, varicose, their walls thinned, the peri-vascular lymph spaces dilated. The membranes of the brain, especially the pia-mater, are almost constantly changed, being injected, infiltrated, thickened and opaque, the vessels giving

ocular evidences of their inability to bear the strain put upon them. The *ependyma* of the ventricles is granular, softened, and often detached. These abnormalities may with reason be attributed to a primary vaso-motor paralysis, which resulted in weakened vascular walls, and later, structural disease of the many vessels of the cerebral cortex. The condition known as "*pachymeningitis interna hemorrhagica chronica*," so frequently observed in advanced general paresis, is an interesting phenomenon. Many authors hold that this condition is primarily and essentially an inflammation of the internal layer of the *dura-mater*.

From a series of investigations which I have carried on for a number of years, I have become convinced that this condition is caused by a neuro-angio paralysis of the vessels of the *pia-arachnoid*, and that the hemorrhage which produces the sub-dural *hæmatoma* is due to a rupture of these vessels; and that any inflammation of the internal surface of the *dura-mater* is secondary to the hemorrhage.

To be brief, it appears to me that after a painstaking clinical and pathological study of the many vaso-motor abnormalities and tropho-neuroses to be observed in a case of parietic dementia, the observer is almost forced to adopt the advanced views of Meynert, Folsom, and Spitzka. These writers hold, in the main, that the symptoms in the primary stage of parietic dementia are largely due to functional or vaso-motor disease of the cerebral vessels, and that this disease begins in the vaso-motor paralysis of the cerebral vessels; and that the cortical encephalitis and degenerative processes, seen in the later stages, are secondary to the neuro-angio paralysis.

Folsom, who has written an able monograph on this disease, adopts Meynert's doctrine; and Spitzka regards the progressive deterioration of the brain as being chiefly the result of neuro-angio paralysis, which is the essential element in the production of the chronic inflammation of the cerebral cortex, so characteristic of the disease.

Taking into consideration, therefore, the preponderance of evidence in favor of the vaso-motor origin of parietic dementia, we should direct our researches toward the vaso-motor center on the floor of the fourth ventricle, hoping there to discover the primary lesion which produces this very fatal and distressing disease. When we add to this incentive the possibility of cure in the prodromal stage of the disease, as held by Meynert, the subject becomes still more important, and is worthy the treatment of abler pens than mine.