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AGING FROM CEREBRAL VASCULAR DISEASE SEEN FROM THE PSYCHIATRIC POINT OF VIEW*

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It would be too big a task to discuss all the possible psychiatric complications of cerebral vascular disease. Fortunately our subject is coupled with the problem of aging and I can therefore confine myself to the psychiatry of the second half of life.

I propose to deal with this matter under the headings: (1) Involutional psychoses, (2) Presenile psychoses, (3) Arteriosclerotic psychoses and (4) Senile psychoses with special reference to cerebral vascular disease.

INVOLUTIONAL PSYCHOSES

Involutional psychoses are ill-defined, for under this heading various mental diseases of different aetiology, endogenous, endocrinal and arteriosclerotic are grouped together. The meaning of the word involution is not clear either. I suspect that it is one of those Latin words that have changed their meaning after they have slipped somehow into the medical dictionary. The Germans have a better word for it; they speak of *Rückbildungsalter*. Decline would perhaps be the best translation. However, *verba valent usu*. When does this involution, this decline start? It is an arbitrary matter and depends on which function is referred to. As regards heart and muscles, one is on the decline at 30 when one is a rugby player; as regards learning by heart and photographic memory one may have started one's decline earlier; as regards adaptability to new circumstances, in other words as regards mental elasticity, involution may be considered to start in the early forties.

One can certainly not equate involution with the menopause as is so often done. That would in the first place exclude the male sex, but it would also ignore the fact that women need not be on the decline apart from their productiveness, and are in fact often in the prime of life, after their menses have ceased. It is of course true that many women have nervous complaints during the menopausal period and also that psychoses which occur at that time often show exogenous symptoms like schizoid traits, peculiar paranoid pictures and hallucinations. This simply shows that these psychoses have not only a temperamental but also an exogenous, *in casu* a hormonic aetiology.

It is obvious that the involutional age is different for different organs and for different functions; it certainly varies in different persons. As we are dealing with psychiatry, I want to stress the middle-age feature of decreased mental elasticity and increased secondary function (i.e. the influences of past experiences on our thinking and feeling). It explains why, for instance, true paranoia, previously latent, becomes manifest at the age of 40, why attacks of depression become more persistent and of longer duration at middle-age. It is the chief cause of so-called involutional melancholia.

Those of us who have learned their psychiatry in the later stages of the Kraepelinian era know the endless controversy concerning involutional melancholia being a separate entity or belonging to the manic-depressive psychoses. Whereas depressive phases of the manic-depressive psychosis were curable and followed by a completely normal interval, the involutional melancholia (usually precipitated by a personal loss) presented a somewhat different picture and was more fixed and protracted and could end in permanent mental sickness and dementia. One usually assumed that in the latter case cerebral arteriosclerosis had supervened.

This problem has assumed a new aspect since the advent of electro-convulsive therapy, for it is exactly in involutional melancholia that E.C.T. has recorded its greatest successes. The results of electroconvulsive therapy have in my opinion proved that involutional melancholia is essentially endogenous, not based on organic changes (hormonic or arteriosclerotic) but psychologically explainable, that it is in fact the middle-age reaction in certain predisposed people to personal, seemingly catastrophic changes in life. The loss of mental elasticity prevents the middle-aged person from adapting himself to the altered circumstances and starting a new life.

It must, however, be admitted that some cases of involutional melancholia are complicated by cerebral arteriosclerosis. The diagnosis of early cerebral arteriosclerosis is by no

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means an easy matter. It is a well-known fact that the abscence of arteriosclerosis in other parts of the body does not exclude cerebral arteriosclerosis and *vice versa.* The so-called neurasthenic stage of early cerebral arteriosclerosis, the lack of concentration, the increased mental fatiguability, the slight loss of memory, may well be reversible melancholic signs. The interpretation of retinal vascular changes is not easy either, and in my experience often misleading.

I should like to make a plea for careful E.C.T. in these cases of involutional melancholia with suspected early cerebral arteriosclerosis. It has been my experience that with the newer *Glissando* method and with generous spacing of one's treatments confusion and gross memory defects can be avoided and excellent results achieved. Obviously I would never advocate any form of electrical treatment in cases of pronounced cerebral arterioclerosis and arteriosclerotic dementia.

PRESENILE PHYCHOSES

This leads us to cerebral arteriosclerotic psychoses as such. But before discussing them and the senile dementias proper, I should say a few words about the so-called *presenile psychoses*, which as regards age lie between the involutional psychoses and the old-age group of psychoses.

There are several histological varieties of these presenile psychoses but the best known are Alzheimer's and Pick's diseases. I shall only mention them in passing, because vascular disease does not seem to play an important role in these conditions, although Alzheimer thought that Pick's disease probably had an arteriosclerotic aetiology.

Pick's disease is based on a selective atrophy of parts of the brain (mainly frontal or temporal) due to death of ganglion cells. In Alzheimer's disease the cerebral atrophy is more generalized. Its histology, with its abundance of senile plaques and fibrillary tangles, places it much nearer to the senile psychoses proper. Both diseases cause progressive gross dementia, usually occur in the presenium, but may make their appearance much earlier.

ARTERIOSCLEROTIC PSYCHOSES

As regards *Cerebral Arteriosclerosis* the old division into arteriosclerosis of the large vessels, causing neurological syndromes, and the generalized arteriosclerosis of the smaller cortical vessels, causing psychiatric disease, is still a useful one. It must not be forgotten that cerebral arteriosclerosis can remain symptomless for a long time.

It stands to reason that neurological disease caused by haemorrhage or thrombosis also has its psychiatric aspect, especially in the case of aphasia, but its discussion would lead us too far.

As regards the general cerebral arteriosclerosis one can distinguish between a mild (beginning ?) form causing a neurasthenic syndrome (mentioned above) and a more intense affection of the cortex causing arteriosclerotic dementia.

The prognosis of the mild form need not be too bad, at least not for quite a time; it shows moreover a marked tendency to remission. We must have all treated cases of this kind where with wise handling life remained tolerably normal and active for many years. It would of course be unwise not to take certain precautionary measures but it would be equally unwise to prescribe complete rest or idleness in these cases; in fact one should advise, if possible, continuation at a slower *tempo* of the patient's original work or activities with a mental interest such as creative hobbies. What should be avoided are efforts to live above one's reserve and vascular age and above all undertaking new responsibilities.

The severe form of progressive arteriosclerotic dementia is of course another matter. Its prognosis is poor, its treatment symptomatic.

SENILE PSYCHOSES

It is often impossible to distinguish these cases from *senile dementia;* in fact in post-mortems one usually finds combinations of cerebral arteriosclerotic disease and senile changes. Cases of typical clinical presbyophrenia have been described which later histological examination proved to be chiefly arteriosclerotic and the opposite also holds good.

That does of course not mean that there are no senile dementias without or with very little arteriosclerotic changes. One must not forget that many mild senile dementias have gradually developed *via* the physiological senile mental changes of loss of mental elasticity, of fixation on the past and lack of interest in the present and in consequence of fixation amnesia. Such patients, if one can call them that, may have soft arteries and become centenarians.

Recapitulating, one can therefore say that cerebral vascular disease can play some part in some of the involutional psychoses, little or none in the presenile psychoses and a deciding role in arteriosclerotic psychoses and dementia, and that it probably is also concerned in many cases that have been diagnosed as senile dementia during life.

How far then does cerebral vascular disease play a part in the process of aging?

There is a great deal of truth in the saying: '*On n'a que l'âge de ses artères,*' but that does not mean that the condition of one's blood-vessels is the deciding factor in aging, although it may be a deciding factor in growing old prematurely. After all, we also grow old without arteriosclerosis. It will probably remain a mystery why life's parabola must come to an end in any case, and what causes its curve to go downhill so much later in some people than in others. I suspect that, if we could preserve the harmonious functioning of all our endocrinal glands, life would continue for ever, which is just another way of expressing an insoluble riddle.

One thing is certain, the arteriosclerotic does not make old bones. Atheroma is not a disease of old age, it is a disease of middle and early old age. It would be nice if we could prevent or cure it, we might prolong the life of our patients. But unfortunately it is a sign of old age of the arterial system itself which has come to the end of its span of life and it is to a marked degree hereditary. Again unfortunately, we cannot choose our ancestors, otherwise we might all reach our century, for very old people are almost invariably descended from long-living parents.

But if we cannot help our patient by preventing early arteriosclerosis or by choosing his ancestors for him, we can give him good advice: To remain functioning and not to retire into inactivity. Applied to his mind that means to continue with mental work as long as possible, preferably in his own sphere and otherwise in creative hobbies, which naturally will be different in people of different educational levels, tastes or talents but which should be mental all the same, keeping his mind interested and alive and therefore keeping it young.