REFERENCES

Blessed, G., Tomlinson, B., and Roth, M. (1968). Personal communication.
Corsellis, J. A. N. (1962). *Mental illness and the ageing brain.* London. Oxford University Press.
Fine, W. (1966). *Geront. Clin.* **8,** 121.
Goldstein, K. (1930). *The organism.* New York. 420, 535.
Goldstein, K. (1942). *After effects of brain injury in war.* London. Pp. 420, 435, 440.
Kay, D. W. K., Beamish, P., and Roth, M. (1964). *Brit. J. Psychiat.* **110,** 146.
Krapf, E. E. (1936). *Mental disturbances in high blood pressure.* Leipzig. 520.
Mayer Gross, W., Slater, E., and Roth, M. (1960). *Clinical Psychiatry.* London.
Pickering, G. (1951). *Lancet.* **2,** 345.
Roth, M., and Morrisey, J. D. (1952). *J. ment. Sci.* **98,** 66.
Roth, M. (1955). *J. ment. Sci.* **101,** 281.
Rothschild, D. (1941). *Amer. J. Psychiat.* **98,** 324.
Rothschild, D. (1956). In chapter xi of *Mental disorders in later life.* Editor, Kaplan, O. London. Oxford University Press.

Discussion

Dr Irvine: What is the rôle of the mental hospital in the inpatient care of patients with the syndrome just described?

Dr Bergmann: Dr Irvine, speaking as a geriatric physician, is on the other side of the fence and has to bear a much greater burden than we do. The rôle of the mental hospital is in a state of flux, but perhaps our main rôle is with the associated severe behaviour disturbances, severe emotional reactions in the mildly or moderately impaired patients who do not need nursing and are not bedridden. It is important to distinguish between the arteriosclerotic and the senile psychotic, because of long static periods that may be found in the former group. Where dementia is minimal and depression is maximal, I have given ECT with great success, although sometimes in fear and trembling.

Dr Rao (*Dulwich*): Is there not a significant amount of metabolic psychosis in the aged?

Dr Bergmann: I agree that metabolic insults, for a variety of reasons, aggravate arteriosclerotic psychosis.

Dr Wollner: An attempt at defining the various causes of dementia is academically important, but I would like to ask the speaker whether he feels that we have as yet sufficient evidence to differentiate these groups on clinical grounds. Some of the examples given almost make it appear that the clinical criteria were made to fit a simple classification between the groups rather than to find evidence for it. Would he perhaps agree that at this moment it would be better to confine our diagnosis to organic brain disease or organic state, look for all possible causes for this and treat whatever is treatable.

Dr Bergmann: No! But Dr Wollner obviously has a point. The prospective studies that were done and the work of Roth in 1955 tested the concepts of unitary organic psychosis in old age, which at that time included depression and schizophrenia, and there were men like Dr Wollner who said, 'It's all part of the same thing but we'll treat the dominant symptom as best we can'. I think there can be little argument that functional psychoses of old age have now been clearly separated off. This is mainly due to the advent of antidepressant drugs and of ECT and the very different outcomes of treatment. There is a great deal of important work going on at the moment regarding RNA metabolism and vitamin B_{12} deficiency, particular attention being paid to the degenerative brain disorders of the senile dementia group. I agree that this work is not conclusive but it may be that in the next generation a great deal of light will be thrown on this subject and that differentiation will become important and far more specific.

Dr Wollner: Is the speaker including under senile dementia the affective disorders, or do we mean by dementia the conditions associated with organic brain disease?

Dr Bergmann: What I was saying was that in the early 1950's and up until quite recently people were prepared to include the affective disorders under a unitary concept of psychosis in old age, such as you are suggesting we should keep for the organic disorders. I believe that the unitary concept of psychosis inside both the organic and the functional groups has been harmful to the psychiatric care of the aged.

Dr Dawe (*Caterham*): Would you give us your diagnostic criteria for arteriosclerotic dementia and would you indicate how we should tackle it as a clinical problem? Does the administration of cyclandelate or isoxsuprine, for example, help the condition or not?

Dr Bergmann: Blessed's work, which has perhaps been the most accurate prospective neuropathologically checked study, showed that there was about an 80 per cent chance of being right if you diagnosed arteriosclerotic dementia in those cases with a history of stroke, localized neurological signs plus any two of the following—hypertension, a variability in course (as opposed to senile psychosis which tends to become steadily worse), epileptic fits, the preservation of insight and personality, and the presence of emotional incontinence. These are the criteria for diagnosing it, and I think a reasonable case has been made for recognizing this as a group. As to the management, I agree entirely with Dr Rao who says that the minor and asymptomatic insults to the brain that may come from associated physical disease are of the greatest importance. Conditions that in non-brain-damaged people you might consider insignificant and not worth treating, such as the secondary infection of an upper respiratory tract infection, might well require antibiotics for a person with an early arteriosclerotic psychosis who is still existing at home. I believe that vasodilators are no good at all, I think it is very deceptive to look at a disease with a fluctuant and remittant course, give your favourite pill, and claim to have done a great deal of good in the last 10 or 12 cases. Probably you are also treating a urinary infection, heart failure, or hypertension at the same time. I see no convincing evidence of the value of vasodilators. And as Dr Jacobs at the Whittington Hospital once said, 'ischaemic tissue contains some of the best vasodilators acting at the point where they are needed'.

Big and little strokes

Dr J. C. Brocklehurst, M.D., M.R.C.P. (Ed.), M.R.C.P. (Glasg.) (*consultant physician*)

Since stroke is primarily a disease of blood vessels a moment's thought must be given to the blood supply of the brain, and the effects of ageing upon it. The internal carotid arteries are uncomplicated, dividing within the cranium into anterior and middle cerebral arteries. The other pair of arteries concerned—the vertebrals—are however more complex, and their course tortuous. They arise from the subclavian arteries, pass back at the root of the neck to enter the transverse foramina of the sixth cervical vertebra, and then pass upward through the foramina in the vertebrae above this. They are thus particularly exposed to pathological changes affecting the upper spine. In old age intervertebral discs become dehydrated and squashed—and vertebral bodies porotic and collapsed. The vertebral arteries may thus become tortuous—and liable to kink on flexion or extension of the neck.

The vertebral arteries pass through the foramen magnum and join together to form the basilar artery, overlying the pons. This supplies blood to the hind-brain and cerebellum. The basilar artery finally divides into the two posterior cerebral arteries.

The anterior cerebral arteries are joined by the anterior communicating artery: the middle and posterior cerebral arteries by the posterior communicating artery. Thus is formed the Circle of Willis.