Abstracts

Editors:
C. Fieschi, Roma
C. W. Loeb, Genova

27 figures, 19 tables, 1983
Drug Dosage

The authors and the publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accord with current recommendations and practice at the time of publication. However, in view of ongoing research, changes in government regulations, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions. This is particularly important when the recommended agent is a new and/or infrequently employed drug.
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Quantitative determination of cerebral blood flow (CBF) and energy metabolism have been exploited to study brain aging since [Scheinberg and Stead](1949). The hypotheses to verify were:

(a) normal aging is accompanied by a slow decline of CBF and CMRO2, which is parallel, and coupled, to reduced functional activity;

(b) chronic diseases of the cerebral vasculature in aging are characterized by a reduction in CBF and an increase in arterovenous oxygen differences - that is, an increased extraction ratio;

(c) chronic brain diseases - such as degenerative dementia - show a decline in oxygen consumption that precedes the decline in CBF; particularly, in this latter condition, cerebral vascular reactivity is fully normal; while in chronic vascular diseases reserves of vasoregulation are reduced.

Neither of these points has received clear experimental support, thus contributing to cast doubt on the whole concept of ‘chronic brain arterial disease’ for example.

Overall, these studies lacked an anatomo-clinical correlation, and now are completely displaced by emission computerized tomographic studies. Two points which were discussed at the Xth Cerebral Blood Flow and Metabolism Symposium in Paris a few days before this Satellite deserve mentioning, since these papers are not being presented here in San Remo.
the still preliminary phase of the neuropsychological correlates of the PET metabolic findings, and
the limitation still inherent to the new astonishing technologies.
Perhaps due to these, it is not entirely surprising that Rapoport, in normal subjects whose eyes were covered and ears plugged to minimize sensory inputs, found no fall in brain oxidative metabolism with age in healthy individuals, while Metter mostly noted a different pattern of intercorrelational distribution of LCMR-glu between young and old (normal) subjects which may reflect ‘a better ability of the older brain to focus regional interaction in response to functional demands, with a more selective arousal pattern’. In other words, perhaps the older participants among us are less energetic, but more ‘astute’ (selective) to compensate. True (!), there is still a long way to go, but these findings cast some doubts on reported ‘subtle’ changes associated with pathology, and much more so than on supposed prevention and treatment, when based on such variable data even in the normal. Furthermore, it seems more unlikely that from animal studies we may learn much on how the complex human brain ages. Difficult as it is, this seems to be a most promising area of basic and applicative investigations for the forthcoming years, and we are therefore proud and thankful for having gathered for two days in
San Remo such a qualified group of experts and original scientific contributions. Besides thanking our friends and collaborators, among and above all the members of the Scientific Advisory Board and Maria Paola Gerini, the National Research Council, the University of Rome, the Administration of San Remo and the pharmaceutical companies with their helpful contributions, we wish
to express our gratitude and encouragement to the International Society of Cerebral Blood Flow and Metabolism, and its President, Louis Sokoloff, and to the Italian Group of Aging for their support and for their effort in the frontier of this area of research.

Cesare Fieschi Carlo Walter Loeb