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DISCUSSION

Used in dosage equivalent on a weight basis to the one used with reserpine, recanescine has shown a slight hypotensive action, but this hypotensive action seems far inferior to the one that can be produced by *Rauwolfia serpentina* or its main alkaloid reserpine. We agree that the number of our cases is small and that our observation period is too short to permit a definite conclusion, but it appears that recanescine, while having all the inconveniences of reserpine, is a less potent alkaloid.

SUMMARY

Recanescine, a new alkaloid of *Rauwolfia canescens*, was given orally to 17 patients for a mean period of 19 weeks. Only four patients showed a significant decrease in blood pressure.

Recanescine has most of the side-effects of reserpine and with the same incidence.

On an equivalent dose, recanescine appears to be inferior in hypotensive action to reserpine.

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INTERNAL CAROTID INSUFFICIENCY*

A USEFUL PHYSICAL SIGN

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THE TEST DESCRIBED HERE is an exceedingly simple one and no doubt has been thought of and employed by many people. However, it does not appear to be recorded in any of the literature pertaining to the diagnosis of carotid insufficiency.

The differential diagnosis between vascular insufficiency and a brain tumour remains difficult in many instances. Age is no criterion.[1, 4] An abrupt onset of a complete hemiplegia in a previously well individual leaves little doubt. A history of partial hemiplegia or intermittent and transient deficiencies is very suggestive[5] but still poses a very considerable problem in the differential diagnosis. A tumour of the hemisphere may simulate the entire picture. In our experience palpation of the carotids themselves has never revealed the diagnosis even in known cases of complete internal carotid thrombosis. The early appearance of a transient ipsilateral monocular blindness is a very helpful diagnostic symptom.[3] Many, however, do not have this feature. Lowered ipsilateral intraocular tension is reported to be of great diagnostic aid.[7]

The cerebral hemisphere will function through a wide variation in arterial pressure.[8] Voluntary movement is preserved almost down to the point of shock level of blood pressure. Anything below the minimal requirement to a hemisphere will result in a complete hemiplegia but not necessarily death to the cerebral tissue. In any one patient the clinical picture will depend on the amount of collateral circulation available. With a complete carotid occlusion and no collateral circulation the patient is completely hemiplegic. With an adequate collateral circulation there may be no signs whatsoever. It is apparent that a complete thrombosis may exist with a history of intermittent signs and complete recovery between attacks (Case 2). Such a patient must have a collateral circulation which is adequate but barely so. Any manœuvre which tends to reduce the total circulation to the head and hence reduce the available collateral circulation may produce transient symptoms referable to the deprived hemisphere, as has been demonstrated in animals.[2] The seemingly premonitory signs may actually indicate the presence of complete occlusion with barely adequate collateral circulation. There are many instances during the normal day when a person's cerebral blood flow is physiologically reduced. Prolonged immobility at a desk, sudden arising, large food intake, or standing still in a waiting line are everyday examples. In such circumstances a hemisphere functioning only on a low collateral circulation may become further deprived to the point of transient dysfunction.

These features were apparent in three patients originally suspected of a brain tumour with intermittent signs referable to one hemisphere. In each there was angiographic evidence of complete occlusion of the internal carotid artery.

In *Case 1*, in a male aged 54, there was a three-week history of intermittent right-sided hemiparesis and aphasia. Neurological examination revealed right arm weakness and a marked aphasia. Angiography revealed a complete obstruction of the internal carotid at its point of departure from the common carotid. Exposure of the carotid revealed a palpable mass about one-half inch (1.25 cm.) long at this junction. This was removed surgically and verified as a thrombus. There was no significant back flow of blood from the cranial end, although a catheter passed with ease up to a distance estimated to be at the syphon. There was no significant improvement following surgery. The *second case* was in a male aged 63, with intermittent aphasia and right-sided hemiparesis of one week's duration. Angiography revealed a complete block of the internal carotid at its point of

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departure from the common carotid. Contralateral angiography showed irrigation of both hemispheres from the good carotid artery. An oxygen-encephalogram revealed no evidence of a neoplasm and the man was dismissed from the hospital symptom-free at that time. *Case* 3 was in a man aged 39, with a two-week history of intermittently progressive right hemiparesis. There was a history of visual loss in the left eye at the onset. Physical examination revealed a nearly complete right hemiparesis and aphasia and a very pale left disc. Angiography revealed a complete obstruction to the internal carotid just beyond its point of departure from the common carotid. The patient became progressively worse and died one week later. Thrombosis of the internal carotid on the left was verified at autopsy.

In contemplating situations such as existed in these three patients, it became apparent that they must be experiencing variations in the available collateral circulation. It was also apparent that if a transient decrease in the collateral circulation could be provoked deliberately with safe control, it should be of great diagnostic value. Thus any manœuvre which would cause such a transient decrease in the collateral to the already impoverished hemisphere should provoke a reappearance of, or aggravation of, the original signs and symptoms. Gradual digital occlusion of the good carotid artery does just that. This deprives both hemispheres of blood, but the normal has a range of reserve as opposed to the previously impoverished hemisphere which becomes suddenly embarrassed to the point of dysfunction.

In each of the three patients mentioned gradual digital compression of the uninvolved carotid produced paræsthesiæ and weakness of the ipsilateral extremities within a matter of seconds. In other words, a patient with thrombosis of the *left* carotid artery with transient signs of numbness and tingling and weakness of the *right* hand exhibits immediate exaggeration of the symptoms when the *right* carotid is partially occluded. The diagnostic value of this manœuvre has been substantiated by Dr. Ross on a patient of his with an angiographically verified thrombosis of the internal carotid artery. It is realized of course that such a manœuvre would give a false negative response in a patient with adequate collateral through the ipsilateral external carotid via the ophthalmic artery or through the vertebral system.

This has been tried in numerous control patients with proven hemispheral tumours who had minimal or transient signs and symptoms referable to the opposite extremity. In none of these did compression of the carotid to the uninvolved hemisphere produce ipsilateral signs and symptoms even when the vessel was compressed to complete occlusion and held. In some there was the expected weakness on the side opposite the compressed carotid as in the usual Matas test.

No patient with middle cerebral thrombosis has been available for this test since the phenomena were noted. It would not be expected to have any effect in such a condition unless the anterior cerebral in the particular patient were supplied predominantly or exclusively from the opposite carotid.

In summary, the picture of carotid insufficiency may produce signs and symptoms often difficult to distinguish from those of an expanding intracranial lesion. When thrombosis of one carotid is suspected, gradual digital compression of the other carotid will produce an almost instantaneous aggravation of the signs and symptoms if the suspicion is correct. False negative responses may be obtained when an adequate collateral circulation exists via the vertebral or the external carotid system but false positive responses seem unlikely.

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EFFECT OF THYROID ABLATION UPON SERUM CHOLESTEROL AND β-LIPOPROTEIN SPECTRUM

The thyroid gland has been consistently implicated in the control of both lipid metabolism and atherogenesis, and it has been known for many years that serum cholesterol levels are influenced by thyroid function. In recent years, several workers have proposed thyroid ablation with radioiodine for the relief of anginal pain due to cardiac insufficiency. The objection has been raised repeatedly that this might accelerate the atherosclerotic process in these patients, particularly of that group in which cardiac insufficiency was caused by coronary sclerosis.

In an attempt to clarify this point, Florsheim, Morton and Goodman (*Am. J. M. Sc.,* 233: 16, 1957) studied the possible effect of reduction of thyroid activity upon atherogenesis by following Gofman's "atherogenic index" and the total serum cholesterol in patients treated with radioiodine. In hyperthyroid patients rendered euthyroid, mild rises in serum cholesterol and atherogenic index were observed. It was considered, however, that these involved only a minimal added risk of atherogenesis by Gofman's calculations. In euthyroid patients *without* atherosclerotic heart disease, the biochemical changes described above following therapeutic thyroid ablation were also of small magnitude. However, in euthyroid atherosclerotic patients, in whom thyroid ablation was carried out, there were generally greater rises in both serum total cholesterol and atherogenic index. This would suggest that therapeutic thyroid ablation in euthyroid patients with atherosclerosis is a potentially dangerous procedure. However, the writers feel that the excellent results obtained by many workers in rehabilitating patients through the relief of cardiac decompensation, congestive failure and anginal pain by this method, certainly seem worth any added small risk of increased atherogenesis.