Letters To The Editor

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Prospagnosia, A Transient Ischemic Attack

To the Editor:

A 57-year-old man was admitted to the hospital with a specific inability to recognize familiar faces or their photographs. However, he knew persons when he heard their voices or saw any special feature of their clothing. This episode lasted 20 hours and was not accompanied by any other neurological deficiency; reading, writing, color distinction and visual function remaining normal.

This neurological deficiency episode was preceded by 2 transient ischemic attacks, of 15 minutes each, declared by right brachio-crural paresis and hypalgesia in the same area.

The neurological examination, neuropsychological study, tests for visual function and memory, were normal. Vital signs, blood and chemistry were normal. The skull and thorax x-rays, electrocardiogram, echocardiogram, aortocranial angiography and computerized tomography were normal.

We believe that the clinical data of this patient, because of its semiological characteristics and the two previous neurological deficiency episodes, corresponds to a transient ischemic attack, a sudden beginning episode consisting of a subjective or objective neurological dysfunction with complete regaining of the neurological function over 24 hours.

From the anatomical point of view bilateral damage to the inferomedial occipital and temporal cortex is the accepted localization for producing prosopagnosia.

To our knowledge this case is the first in which prosopagnosia has been reported as a transient ischemic attack.

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Reasons Are Given
For OPG-Gee Results

To the Editor:

In the recent paper by Lynch et al. (Stroke 12: 325-330, May-June, 1981), the stated accuracy of the OPG-Gee was considerably lower than that reported by several other groups of investigators. There are 2 definite reasons and one possible explanation for this.

1. One definite reason was the first criterion for abnormality of the study, an ophthalmic artery pressure less than two-thirds of the brachial systolic pressure. This is contrary to my report1 involving data derived with the instrument with a 500 mm Hg vacuum manometer. This is contrary to my report1 in which I observe that the ophthalmic systolic pressure should not be less than 39 plus 0.42 times the brachial systolic pressure (OSP = 39 + 0.42 BSP). The latter interpretation will reduce considerably the incidence of false-negative results.

2. We measure the brachial blood pressure by arm cuff and auscultation. Bilateral determinations are made in the sitting position, prior to the test. The test is completed in the supine position, and, immediately upon completion of the test, with the patient remaining in the supine position, the brachial blood pressure is redetermined in the arm in which the higher systolic pressure was noted in the sitting position. The supine brachial systolic pressure is the figure used for correlation with the ophthalmic systolic pressures. The latter brachial systolic pressure most accurately reflects that encountered during the test.2 The criteria that I have established do not apply if systolic pressures from arterial lines or Doppler systolic pressures are used for correlation.

3. The paper stated that biplane angiography was obtained, and a hemodynamically significant lesion was defined as a residual stenosis lumen which was 50% or less than that of the least transverse diameter of the normal distal internal carotid artery. I presume that this applies, even if this severity is seen in only one plane of the angiogram. However, this degree of stenosis must be seen in both planes of the angiogram before a cross-sectional area stenosis of 75% can be determined. Carotid stenoses do not become hemodynamically or pressure-significant until they have reduced the cross-sectional area by 75% or more, as defined by the instrument.3 It puzzles me why the authors categorize the results regarding varying degrees of stenosis, when no claim has ever been made by me in this regard.

The principal purpose of the technique is the physiologic definition of the brachiocephalic arterial system. A secondary application is its use as a screening device for the non-invasive detection of hemodynamically significant carotid lesions. Multiple reports confirm a high degree of accuracy, when the instrument is used for its intended purpose. The simplicity and speed of its application and interpretation are its principal attributes. In no way does it compete with any method of anatomic definition of the brachiocephalic arterial system, invasive or noninvasive. I encourage the authors, and other investigators, to duplicate the technique as I have presented it, if they wish to duplicate the accuracy reported by several groups of investigators.

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References
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