
The Terminology of Transient Visual Loss Due to Vascular Insufficiency

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SUMMARY Transient visual loss due to cerebro-ocular vascular disease is a common symptom. The purpose of this paper is to present a unified terminology of the monocular vs. binocular or homonymous types. Lack of proper identification may lead to mis-diagnosis and improper management of these entities.

Monocular blurred vision must be investigated since its origin is so commonly due to arteriosclerosis of the carotid system. Binocular blurred vision due to vertebro-basilar insufficiency is managed conservatively in almost all instances.

IT IS UNIVERSALLY acknowledged that the most reliable indicator of impending ischemic stroke is a transient ischemic attack.1 The semantics of the ocular symptomatology have become confusing and are often merely referred to as transient visual obscurations. It is the purpose of this communication to attempt to clarify this matter.

The first distinction one must make in describing the ocular symptoms is whether they are monocular or binocular. The monocular attacks are designated as amaurosis fugax and transient monocular blindness and are due to vascular insufficiency in the optic nerve and retina, whereas transient binocular blindness indicates involvement of the posterior visual pathways. Secondly, one should ascertain the duration of the attacks. Accompanying neurologic signs or symptoms provide additional evidence as to the localization of the ischemic process in the visual system.

Amaurosis fugax is a monocular fleeting attack of partial to total (rare) blindness lasting from seconds to a few minutes. It is usually considered to be due to emboli but may be due to a perfusion deficit at the nerve head as in papilledema or incipient anterior ischemic neuropathy. The word obscuration means very fleeting loss of vision (few seconds). Transient visual obscurations may be most aptly applied to this phenomenon seen with papilledema; thus, as in pseudotumor cerebri especially and rarely in brain tumor, these attacks can be characteristic and associated with headache and other neurologic signs or symptoms.

Transient Monocular Blindness is an episode of transient visual obscurations lasting longer in duration than amaurosis fugax which may be partial or total. Partial blindness may take the form of a shadow coming from below up, from above down, or from the side (usually temporal). It may progress towards fixation and then regress. A "shutter" effect may constrict the field and then disappear. These attacks may last many minutes, hours and rarely several days. These episodes are probably related to hemodynamic changes such as in hypertension or some hematological cause. Migraine is an uncommon cause of monocular visual loss. Patients with bilateral carotid disease causing orbital hypoxia either due to retinal or optic nerve hypoperfusion can get transient visual loss but this is not simultaneous or binocular.

Transient Binocular Blindness is usually due to cerebrovascular disease affecting the calcarine cortex. It is bilateral and involves either the homonymous field, the inferior or superior altitudinal fields or the central fields. These episodes usually last 5 to 30 minutes and occasionally as long as several hours. They may exact-
ly reproduce either the fortification (teichopsic) or the scintillating scotoma seen in classic migraine which characteristically lasts 15–30 minutes and really cannot be differentiated from vertebro-basilar insufficiency. It was this association that led the author to name these attacks ‘Isolated Ophthalmic Migraine’. Others have referred to these visual episodes as Acephalgic Migraine since they do not have headache associated with them.

Fischer recently reported his experience at length of ‘migrainous accompaniments’ including transient attacks of blindness, homonymous hemianopsia and blurring in patients after the age of 40 without headache and with normal cerebral angiograms. He defended the use of the term ‘migraine’ as used by this author. However, to save confusion, these visual attacks would probably be best referred to as Transient Binocular Blindness. Their pathogenesis may be different from the vasoconstriction as defined by Wolff or the shunting mechanism as described by Heyck. Fisher feels they are rarely caused by basilar artery disease.

The following terminology is suggested: 1) transient blurred vision as an overall designation, 2) transient monocular blindness to be divided into — a) amaurosis fugax (seconds to minutes), b) transient monocular blurring, more prolonged unilateral attacks, 3) transient binocular blindness — homonymous attacks of 5–30 minutes duration. The mechanism and the pathogenesis of these symptoms are complex and, in particular, the attacks of transient binocular blindness are poorly understood but if we all refer to them in similar terminology we may have taken a step forward.

References

Cerebral Infarction Secondary to Unsuspected Intracranial Fibromuscular Dysplasia Following Bypass of Aortic Coarctation

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SUMMARY Fibromuscular dysplasia (FMD) is an uncommon finding in the cerebral circulation. We present a case of unsuspected intracranial FMD in a patient dying from a large cerebral infarct following a bypass operation for coarctation of the aorta. The need for recognizing the possible co-existence of these two lesions is emphasized.

Fibromuscular dysplasia (FMD) is a nonatheromatous, segmental stenosing angiopathy. There are reports of involvement of nearly all systemic arteries. FMD, however, is uncommon in the intracranial circulation. Many patients with FMD harbor other vascular or developmental lesions but it has not previously been reported in association with tubular segmental aortic stenosis or coarctation. This report concerns fatal cerebral infarction in a patient with unsuspected intracranial FMD following bypass of an aortic coarctation. It illustrates the importance of recognizing the possible co-existence of FMD and other vascular anomalies.

Case Report
A 41 year old right-handed man was admitted to the Victoria General Hospital on September 28, 1983, for elective repair of an aortic coarctation. During the year prior to admission he complained of exertional dyspnea, intermittent claudication and chest discomfort. For a number of years he had been taking propranolol for hypertension.

He was described as a ‘‘small thin male.’’ He was mildly retarded. Blood pressure was 170/60 in both arms. Femoral pulses were delayed and diminished. A
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