Clinical/Scientific Notes

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BLINK TO VISUAL THREAT DOES NOT HERALD CONSCIOUSNESS IN THE VEGETATIVE STATE

The blink response to visual threat is a standard bedside method for testing visual processing. In response to a sudden gesture directed toward the eyes, a person with a normal blink response will promptly contract both orbicularis oculi muscles to close the eyelids momentarily. There is no consensus as to whether blinking to visual threat (BVT) is purely reflex[1] or a cognitively mediated behavior that heralds consciousness; i.e., is incompatible with the diagnosis of the vegetative state (VS).[2,3] Some authors stated that "one should be extremely cautious in making the diagnosis of the VS when there is . . . response to threatening gestures."[4] Others stated that "react(ion) to visual threat" is a "compatible but atypical feature" of VS.[5] Similarly, other guidelines stated that the "threat response is usually absent" in VS.[6] Finally, BVT was not mentioned in some workgroup criteria on the minimally conscious state.[7]

The aim of the study was to determine the incidence of BVT in patients whose clinical features are in all other respects typical of the VS, as assessed by means of validated testing.[7] We also investigated whether the presence of BVT in patients considered vegetative is predictive of recovery of consciousness.

Methods. The BVT was assessed (four trials per eye) in patients in a VS (by means of the Coma Recovery Scale Revised [CRS-R])[7] of traumatic or nontraumatic etiology in the acute (<4 weeks) or subacute setting (≥4 weeks), by quickly moving a finger 1 inch in front of the patient's eye, while avoiding contact with the eyelashes or inadvertent production of a breeze. All patients were assessed free of sedative drugs. As stated elsewhere,[7,3] BVT was defined as a blink promptly following presentation of visual threat on at least two trials with either eye. To avoid misinterpreting spontaneous blinks as BVT, we tested patients between spontaneous blinks. However, in the absence of a controlled laboratory protocol, this bias cannot be formally excluded. Outcome was studied at 1 year for traumatic and 3 months for nontraumatic cases. Patients who died or remained vegetative (unfavorable outcome) were compared to patients who recovered from VS (favorable outcome). Differences between outcomes in patients with and without blink were calculated using χ^2 testing, thresholded for significance at $p < 0.05$.

Results. Out of 91 patients with VS included (60 in New Jersey and 31 in Liège; mean age 45 ± 20 years), 19 were studied in the acute (15 ± 6 days post-insult), and 72 in the subacute setting (3 ± 5 months post-insult). Etiology was traumatic in 41 and nontraumatic in 50 patients (i.e., anoxic encephalopathy [n = 27], ischemic or hemorrhagic stroke [n = 12], metabolic encephalopathy [n = 8], and tumor [n = 3]). Forty-six out of the 91 patients (51%) showed BVT (32 subacute; 26 nontraumatic). In these 46 patients, 10 died (7 nontraumatic), 22 remained in VS (14 nontraumatic), and 14 emerged from their VS (5 nontraumatic) (table e-1 on the *Neurology*® Web site at www.neurology.org).

Forty-five patients (49%) did not show BVT (40 subacute; 24 nontraumatic). Out of these 45 patients, 8 died (5 nontraumatic), 28 remained in VS (17 nontraumatic), and 9 emerged from their VS (2 nontraumatic). Differences in outcome between patients with and without BVT were not significant. Positive predictive value of BVT on recovery from VS was only 30% while negative predictive value was 80% (table).

Discussion. Nearly half of our patients in VS showed a BVT in the absence of any other clinical sign of consciousness when assessed by means of standardized testing,[7] suggesting that BVT may be a common clinical feature of VS. In the literature, BVT is ambiguous with regard to diagnostic relevance. Some authors stated that a response to visual threat "implies awareness of threat."[2] This view seems in line with neuro-ophthalmologic studies in patients with cortical blindness, neglect, and Balint syndrome, which conclude that blinking to threat requires intact primary visual cortex as well as higher order mechanisms for visual attention thought to be mediated in the inferior parietal lobule and frontal eye fields.[3] In contrast, others suggested that "a blink response to visual threat . . . does not imply consciousness."[1] Our data support the latter view and

Table	Chi-square contingency data statistics for patients in vegetative state (VS) with and without blink to visual treat				
VS with blink to visual treat (n = 46)			VS without blink to visual treat (n = 45)		
REC	VS	Died	REC	VS	Died
14 (5 NTBI)	22 (14NTBI)	10 (7NTBI)	9 (2NTBI)	28 (17NTBI)	8 (5NTBI)

Patients in VS who died, remained in VS, or recovered from VS (REC) 1 year after traumatic and 3 months after nontraumatic etiology (NTBI = nontraumatic brain injury). Positive predictive value (patients showing a blink response to visual treat who subsequently recover) 30%; negative predictive value (patients showing no blink response to visual treat who died or remained in VS) 80%. $\chi^2 = 2.018$, $p > 0.05$.

show that the presence of BVT is not a reliable predictor of recovery from VS—in contrast, its negative predictive value was 80%. BVT is commonly observed in patients who meet other requisite criteria for VS. Its presence does not necessarily herald consciousness nor recovery of consciousness in patients with severe brain injury.

e-Pub ahead of print on August 20, 2008, at www.neurology.org.

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Funded by the Belgian Fonds National de la Recherche Scientifique (FNRS), the French Speaking Community of Belgium Concerted Research Action, the Belgian Federal Public Service for Public Health, the European Commission, and the J.S. McDonnell Foundation. S.L. is Senior Research Associate at FNRS, and M.-A.B. and O.G. are Research Fellows at FNRS.

Disclosure: The authors report no disclosures.

Received February 6, 2008. Accepted in final form May 5, 2008.

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VANISHING ANEURYSM IN PRETRUNCAL NONANEURYSMAL SUBARACHNOID HEMORRHAGE

Pretruncal, nonaneurysmal subarachnoid hemorrhage is generally a benign entity with an unknown etiology.[1] A ruptured vein, cryptic arteriovenous malformation, perforating artery, or intramural hematoma have all been implicated as potential sources for hemorrhage in these patients.[2,3] We present a patient who had the typical clinical and radiographic course associated with pretruncal, nonaneurysmal subarachnoid hemorrhage but in whom, based on serial, three-dimensional rotational angiograms (3DRA), we uncovered a transient microaneurysm near the basilar apex. We posit that such self-limited microaneurysms might be the cause of some proportion of the patient population that presents with pretruncal, "nonaneurysmal" subarachnoid hemorrhage, but that, prior to the advent of 3DRA, we simply were unable to diagnose these lesions. We present this case not only to inform other practitioners who might encounter these microaneurysms, but also to raise the possibility that these lesions represent yet another important source of pretruncal nonaneurysmal subarachnoid hemorrhage.

Case report. A 47-year-old woman developed the worst headache of her life while exerting herself during weightlifting. She reported to the emergency department where a noncontrast head CT demonstrated acute, subarachnoid hemorrhage in a perimesencephalic pattern, consistent with a pretruncal nonaneurysmal subarachnoid hemorrhage. Clinically, she was a Hunt-Hess and WFNS grade 1.

A cerebral angiogram was performed, including both standard digital subtraction angiography (DSA) as well as 3DRA (Phillips), which was negative for