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KINKING OF CAROTID ARTERIES IS NOT A MECHANISM OF CEREBRAL ISCHEMIA:

A functional evaluation by Doppler echography

Short title: Carotid kinking evaluation by Doppler echography

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ABSTRACT

Aim: To evaluate the hemodynamic behavior of carotid kinking, as assessed by color Doppler ultrasonography at baseline and during neck movements, and their relation to neurological symptoms.

Methods: In this cross-sectional study, 60 consecutive patients with non-atheromatous carotid kinking in whom diagnostic color Doppler ultrasonography investigation of neck vessels had been requested for clinical suspicion of atherosclerotic disease were evaluated. To evaluate if there were significant changes of blood velocities as a consequence of kinking, for each carotid artery we recorded systolic and diastolic velocities both in the segments proximal to kinking, as well as intra-kinking. And the effects of postural changes and neck movements on carotid blood flow were also studied.

Results: Flow in carotid arteries with kinking was always normal, and no differences were found between flow velocity measured at the level of kinking compared to the normal tract of the vessel. During head rotation tests, flow remained largely unaffected, a substantial reduction in the velocities in the ophthalmic artery was found in 13.5% of the cases, while an increase was recorded in 27%; and no symptoms or events were recorded during the study. None of the patients referred symptoms, nor were neurological events or signs detected during the maneuvers.

Conclusion: Our results show that carotid kinkings are not a mechanism of acute cerebral ischemia, and therefore are unlikely to be a cause of neurological events or symptoms.

Key words: kinking; Doppler; ophthalmic artery; flow velocity; ultrasonography

INTRODUCTION

Atherosclerosis is the most frequent cause of extracranial carotid artery disease and stroke[1]. However, other mechanisms may also cause carotid stenosis or occlusion, such as: fibrodysplasia, trauma (with subsequent dissection of carotid arteries), aortic arch pathology as in Takayasu disease, and aortic dissection[2]. Among non-atheromatous pathologies of the carotid arteries, interest has long been placed on specific anatomical abnormalities known as "dolichoarteriopathies".

Dolicoarteriopathies of carotid arteries are frequent, ranging between 5 to 66% according to the population investigated and to the technique employed for their diagnosis[3-5]. Carotid dolichoarteriopathies are classified into different types[6]. Particular interest is given to type 3 –*Kinking*-, i.e., the inflection of two or more segments of the artery, with an internal angle of ≤90 degrees (Figure 1). Frequency of cerebrovascular symptoms in patients with carotid dolichoarteriopathies is reported as high as 15-23%[7,8]. However, despite the high prevalence of this condition, and the frequent report of symptoms apparently associated with it, previous studies had been inconclusive regarding the actual hemodynamic and prognostic significance of carotid kinkings.

Mukherjee et al.[9] proposed that carotid kinking would induce turbulent flow, with consequent intimal ulceration, platelet deposition, and possible distal thrombus embolism. Other investigators similarly believe that a causal relationship exists between cerebral ischemia and carotid dolichoarteriopathies, and advocate their surgical correction to prevent stroke[10-25]. Conversely, other authors consider carotid kinking as a mere anatomical variety, devoid of clinical consequences[26]. According to this latter hypothesis, the relatively high prevalence of symptoms in patients with carotid kinking could be explained by a referral bias, i.e., patients with symptoms are

obviously more likely to undergo examination of neck vessels, and hence to be found as having kinking; without investigating a cohort of asymptomatic subjects for comparison, one would think that the high prevalence of carotid kinking in symptomatic patients indicates a causal relationship. Indeed, we have very recently reported that performing a systematic investigation for carotid artery abnormalities reveals that the prevalence of carotid dolichoarteriopathies in the general population is high, even among children, and it is actually very similar across various groups of normal subjects and of patients with cardiovascular risk factors[27].

Admittedly, the hypothesis that carotid kinkings can be a causative mechanism of cerebral ischemia does have some attractiveness, as it rests on seemingly obvious anatomical considerations suggesting that major bends of the carotid arteries ought to affect cerebral blood flow, particularly when neck vessels are stretched out or further bent because of neck movements. By this same reasoning, however, it derives that if carotid kinkings were responsible for ischemic events, it should be possible to induce or to reproduce cerebral ischemia by functional hemodynamic tests, therefore being able to put to direct test this hypothesis, or to disprove it.

Therefore, the objective of this study was to evaluate the hemodynamic behavior of carotid kinking, as assessed by color Doppler ultrasonography at baseline and during neck movements, and their relation to neurological symptoms.

METHODS

This was a cross-sectional study designed to investigate the hemodynamic consequences of non-atheromatous kinkings in the occurrence cerebrovascular symptoms in adult patients, recruited among individuals referred to our outpatient clinics for evaluation. The population in study consisted in 60 consecutive patients with non-atheromatous carotid kinking in whom diagnostic color Doppler ultrasonography investigation of neck vessels had been requested for clinical suspicion of atherosclerotic disease.

Patients were studied in dorsal decubitus position, with the head slightly turned away from the side of the carotid under examination. An ATL Apogee CX echographic machine equipped with 2 mechanical sectorial transducers unit was used: a 7.5 MHz one was utilized for imaging neck carotid arteries and 2.75 MHz transducer was used for ophthalmic arteries. Regarding the last one it was also necessary in a few cases of high carotid bifurcations, to be able to examine the full stretch of the internal carotid artery.

The study began by placing the transducer at the base of the neck and moving it up to the jaw. Conventional images were acquired on the longitudinal axis and then on the transverse axis; then, color Doppler mapping was performed; the study of each segment ended with pulsed-wave Doppler. Images were stored on videotape and printed on black and white and color photographic paper. An experienced investigator unaware of patients' clinical characteristics performed the analysis. To evaluate if there were significant changes of blood velocities as a consequence of kinking, for each carotid artery we recorded systolic and diastolic velocities both in the segments proximal to kinking, as well as intra-kinking.

To evaluate the effects of postural changes and neck movements on carotid blood flow, the study continued with the patient in dorsal decubitus position, while asking to keep his/her eyes closed during the entire test, which was done on both sides, as previously reported[28,29]. For imaging of the ophthalmic arteries the transducer was placed in the middle of the upper eyelid, with a slight tilting towards the inside, and the two-dimensional echographic image of the eyeball was obtained. The ophthalmic artery was first visualized by color Doppler, and then the characteristic spectrum on pulsed-wave Doppler signal was recorded. Maximum systolic and end-diastolic velocities were measured in the ophthalmic arteries ipsilateral to the kinking. Pulsatility and resistance indexes were also measured. Then, the patient was asked to slowly rotate his/her head internally and then externally, while the operator tried to maintain the same image and the same angle during this movement. The above parameters were registered during each maneuver, and compared to the basal ones. Occurrence of symptoms in asymptomatic patients was evaluated, as well as the reproduction of the same symptoms, or the occurrence of new neurological symptoms in previously symptomatic patients.

Statistical Analysis

Values are expressed as mean ± standard deviation. Statistical analyses were performed using absolute values and processed through GraphPad Prism version 4.0 for Windows (GraphPad Software, San Diego, CA). Comparisons among groups were carried out using Chi2 test (univariate and multivariate analysis), paired t test or one way analysis of variance (Student-Newman-Keuls), as appropriate. A value of $p<0.05$ was considered significant.

RESULTS

Patient Population: We studied 60 consecutive patients with carotid kinking (41 women, 19 men), with a median age of 71 years (interquartile range 6.5), in whom ultrasonography investigation of neck vessels had been requested for clinical suspicion of atherosclerotic disease. Thirty-three patients (55%) had previously complained of neurological symptoms, while 27 patients (45%) were asymptomatic or presented non-specific symptoms. The symptomatic patients suffered from vertebro-basilar syndrome (15; 25%), dizziness (5; 8%), transient ischemic attack (3; 5%), stroke (3; 5%), syncope (3; 5%) and miscelllaneous entities (aphasia, paresthesia, congestive heart failure and a candidate for endarterectomy; 7%).

All patients showed at least one major cardiovascular risk factor; many patients presented more than one risk factor, and eleven patients (18%) had 3 or more risk factors. Patients presented hypertension (27; 45%), cigarette smoking (11; 18%), dyslipidemia (10; 17%), diabetes mellitus (8; 13%), and coronary heart disease (7; 12%).

With respect to the side affected, the left side predominated, being present in 34 cases (57%) vs 12 (20%) on the right side and 14 cases of (23%) bilateral kinking ($p<0.001$). Preferential involvement of the left carotid artery is consistent with previous reports[28].

Basal Assessment: As expected, color and pulsed-wave Doppler showed turbulences at the site of the kinking. However, both maximum systolic velocity and end-diastolic velocity of internal carotid arteries were substantially unaffected by kinking (Table 1, Fig. 2). Moreover, no differences were found with respect to carotid

flow velocities when data from symptomatic patients were analyzed separately (Table 2).

Functional assessment: Images obtained through the ultrasonographic study of the ophthalmic arteries allowed us to record and measure basal peak systolic and end-diastolic velocities, and their changes occurring during internal and external rotation of the head (functional test). Table 3 reports various indices of flow velocity in the ophthalmic artery at baseline, and during neck movements: on the average, small, non-significant differences between groups were seen. At the individual level, 44 patients (59.46%) maintained similar basal and post-test velocities. In 10 (13.51%) a decrease >20% of the basal values was observed, while in 20 (27.03%) an increase in velocity values >20% was recorded (Figure 3). None of the patients referred symptoms, nor were neurological events or signs detected during the maneuvers. After a flow obstruction, hyperfusion can be observed. However, none of the patients experienced these complaints.

DISCUSSION

The present study shows that blood flow through carotid arteries with major alterations of their course -such as >90 degree kinking- is largely unimpeded, even during torsion movement of the neck and head. Importantly, most of patients in our series showed cardiovascular risk factors, and many of them had a history of cerebrovascular symptoms. Taken together, these findings indicate that presence of anatomical alterations of the course of carotid artery is unlikely to be an important pathogenic mechanism of cerebral ischemia.

Over the past 30 years a number of papers have reported an association between carotid dolichoareriopathies and symptoms of cerebral ischemia, or even frank cerebrovascular events; importantly, these studies almost invariably concluded that this was a mechanism of disease, and that surgery was the therapy of choice[10-25]. However, in reviewing those earlier reports the issue of a possible referral bias should be seriously considered, as the vast majority of those papers were based on the findings of surgical series, and therefore it is not surprising that their condition was deemed to require surgical correction. Furthermore, gauging severity of stenosis in bended segments is difficult, and prone to errors which may lead to overestimation, particular when images taken on a single plane are used. Finally, it has to be emphasized that many patients in those series also had a high-risk profile in terms of cardiovascular risk factors, which in and of itself could have been responsible for their neurological symptoms.

In a previous study in a large cohort[27], we demonstrated that carotid kinking and coiling are related to embryological disorders, and not to atherosclerotic remodeling or vascular degeneration, because of their similar prevalence in children compared to adults, and because there was no association with cardiovascular risk

factors. The results of the present study extend those observations, by directly documenting that carotid dysembryoplasias would not cause neurological events or symptoms. Of note, we did not find any case of narrowing kinking in our patients. It is our point of view that criterium used in the literature to define carotid artery stenosis by kinking are confusing and not validated.

To our knowledge, these findings are entirely novel. However, previous observations indirectly lend further support to our conclusion that carotid kinkings are benign embryological alterations. In 1997, Oliviero et al.[30] demonstrated, in hypertensive patients with carotid kinks, that the incidence of neurological events was similar to matched controls with hypertension but no kinks. More recently, the same group published the results of subsequent follow-up of these patients[31], confirming that the group of hypertensive patients with carotid kinking showed a similar, or even better, outcome than the control group of hypertensive patients without kinking. Moreover, they also demonstrated that intima-media thickness at the site of kinking was not increased compared to that measured at the carotid bifurcation.

Of note, Toole and Tucker[32] performed a post-mortem study in which perfusion fluids were injected in order to evaluate the hemodynamic behavior of the neck vessels during rotation head maneuvers. They concluded that “variation in flow through these vessels was frequently observed when the head was turned to various positions”, and emphasizes “the need for attempts to demonstrate these phenomena in the living”. After almost 50 years, the present study might finally address this interesting issue.

CONCLUSIONS

Carotid kinkings do not cause cerebral ischemia, and therefore are not a likely mechanism of neurological events or symptoms. Surgical correction of these embryological alterations should require solid documentation of their inducing cerebral ischemia, or clear evidence of a significant stenosis of the affected segment.

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TITLE OF TABLES

Table 1. Pre-kinking and kinking systolic and diastolic velocities in all patients

Table 2. Pre-kinking and kinking systolic and diastolic velocities in symptomatic and asymptomatic patients

Table 3. Ultrasonographic values obtained during the functional test (internal and external rotation of the head)

LEGEND OF FIGURES

Figure 1.

Type 3 of dolichoarteriopathies (kinking), according to the definition of Weibel and Fields[6].

Figure 2.

Right (R) and left (L) internal carotid arteries maximum systolic velocities (MSV) and end diastolic velocities (EDV) , prior to kinking (PK) and intra-kinking (K)

Figure 3.

Percentage change of maximum systolic velocities (MSV) in ophthalmic arteries in relation to basal velocities, through the functional test

Table 1. Pre-kinking and kinking systolic and diastolic velocities in all patients

	Pre-Kinking (m/sec)	Kinking (m/sec)	p
maximal systolic velocity (right)	0.603±0.18	0.647±0.16	NS
maximal end diastolic velocity (right)	0.196±0.06	0.223±0.06	NS
maximal systolic velocity (left)	0.628±0.26	0.732±0.35	NS
maximal end-diastolic velocity (left)	0.211±0.11	0.258±0.20	NS

NS=not significant (paired t test)

Table 2. Pre-kinking and kinking systolic and diastolic velocities in symptomatic and asymptomatic patients

	Pre-Kinking (m/sec)		Kinking (m/sec)	
	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic
maximal systolic velocity (right)	0.62±0.19	0.54±0.11	0.65±0.17	0.64±0.13
maximal end-diastolic velocity (right)	0.19±0.06	0.21±0.05	0.22±0.06	0.22±0.05
maximal systolic velocity (left)	0.62±0.27	0.65±0.22	0.74±0.38	0.71±0.23
maximal end-diastolic velocity (left)	0.22±0.13	0.19±0.06	0.27±0.23	0.22±0.07

Table 3. Ultrasonographic values obtained during the functional test (internal and external rotation of the head)

m/sec	ROA basal	ROA rotation to left	ROA rotation to right	p	LOA basal	LOA rotation to left	LOA rotation to right	p
IP	1.311±0.32	1.336±0.36	1.245±0.31	NS	1.398±0.57	1.441±0.51	1.362±0.48	NS
IR	0.697±0.07	0.711±0.09	0.695±0.09	NS	0.785±0.33	0.817±0.44	0.857±0.56	NS
A/B	3.586±1.12	3.880±1.42	3.582±1.20	NS	3.585±1.30	3.770±1.34	3.808±1.49	NS
V1	0.373±0.14	0.443±0.18	0.382±0.13	NS	0.429±0.15	0.424±0.18	0.514±0.57	NS
V2	0.111±0.05	0.114±0.05	0.116±0.04	NS	0.151±0.16	0.125±0.06	0.133±0.07	NS
MV	0.217±0.09	0.225±0.09	0.221±0.07	NS	0.266±0.17	0.245±0.19	0.238±0.10	NS

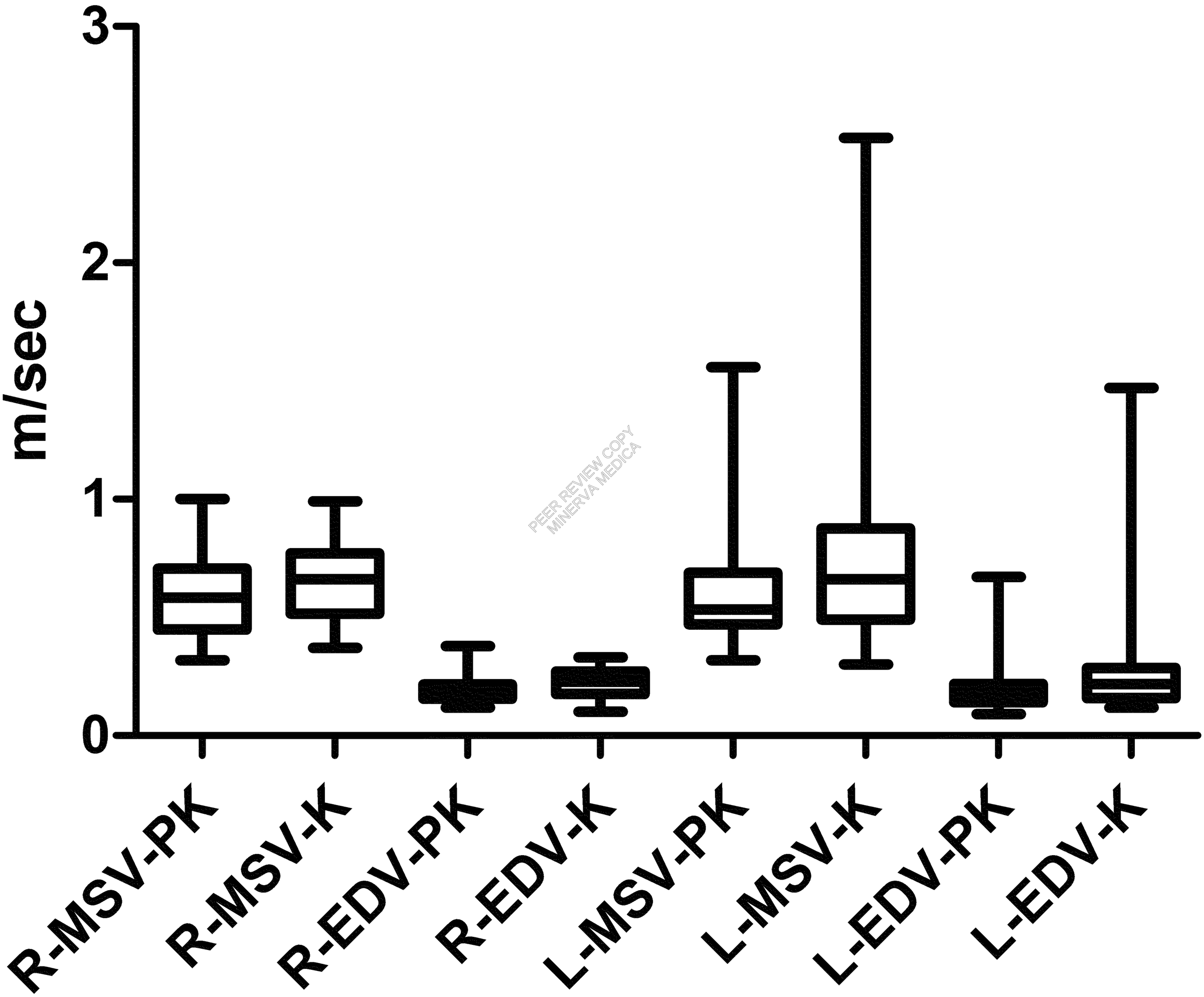
ROA=right ophthalmic artery; LOA=left ophthalmic artery; IP= pulsatlity index; IR= resistant index

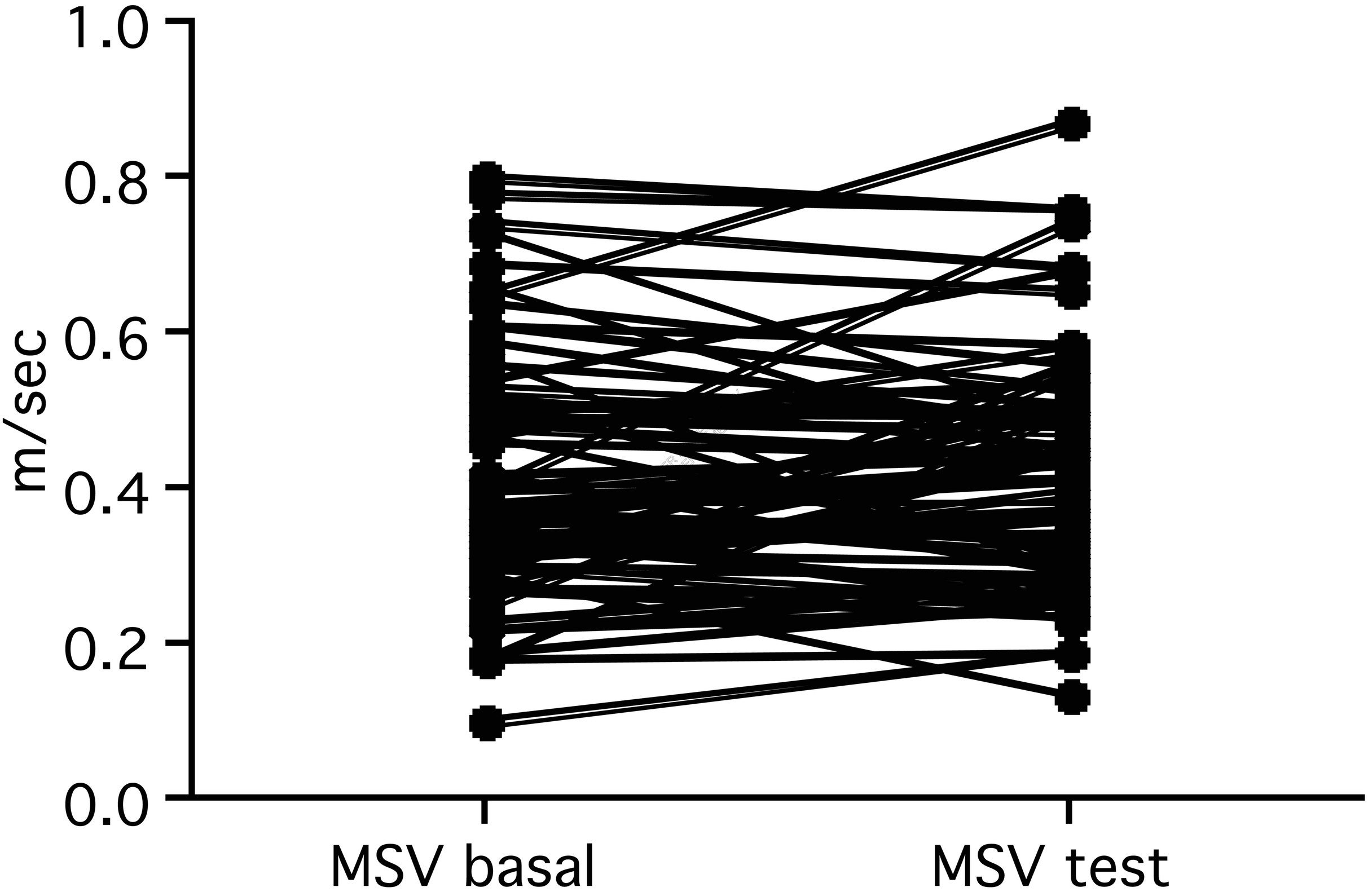
V1=Maximal systolic velocity; V2=End diastolic velocity; MV= mean velocity

A/B= Maximal systolic velocity/ End diastolic velocity

NS=not significant (one way analysis of variance (ANOVA) Student-Newman-Keuls)

Type 3
Kinking

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m/sec
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