Vagal nerve stimulation decreases blood-brain barrier disruption after traumatic brain injury : Journal of Trauma and Acute Care Surgery

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ORIGINAL ARTICLES

Vagal nerve stimulation decreases blood-brain barrier disruption after traumatic brain injury

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This study was presented as poster at American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery 2011.

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Journal of Trauma and Acute Care Surgery 72(6):p 1562-1566, June 2012. | DOI: 10.1097/TA.0b013e3182569875

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Abstract

BACKGROUND

Traumatic brain injury (TBI) may alter sympathetic tone causing autonomic abnormalities and organ dysfunction. Vagal nerve stimulation (VNS) has been shown to decrease inflammation and distant organ injury after TBI. It is unknown whether VNS may reduce blood-brain barrier (BBB) dysfunction after TBI. We hypothesize that VNS prevents TBI-induced breakdown of the BBB, subsequent brain edema, and neuronal injury.

METHODS

A weight-drop model was used to create severe TBI in balb/c mice. Animals were divided into three groups: TBI—TBI only; TBI or VNS—animals that were treated with 10 minutes of VNS immediately before TBI; and sham—animals with opening of the skull but no TBI and VNS treatment. Brain vascular permeability to injected (M_r 70,000) FITC-dextran was measured by radiated fluorescence 6 hours after injury. Injured tissue sections were stained for perivascular aquaporin 4 (AQP-4), an important protein causing BBB-mediated brain edema. Fluorescence was quantified under laser scanning by confocal microscopy.

RESULTS

Six hours after TBI, cerebral vascular permeability was increased fourfold compared with sham (mean [SD], 6.6^{E+08} [5.5^{E+07}] arbitrary fluorescence units [afu] vs. 1.5^{E+08} [2.9^{E+07}] afu; $p < 0.001$). VNS prevented the increase in permeability when compared with TBI alone (mean [SD], 3.5^{E+08} [8.3^{E+07}] afu vs. 6.6^{E+08} [5.5^{E+07}] afu; $p < 0.05$). Perivascular expression of AQP-4 was increased twofold in TBI animals compared with sham (mean [SD], 0.96 [0.12] afu vs. 1.79 [0.37] afu; $p < 0.05$). Similarly, VNS decreased post-TBI expression of AQP-4 to levels similar to sham (mean [SD], 1.15 [0.12] afu; $p < 0.05$).

CONCLUSION

VNS attenuates cerebral vascular permeability and decreases the up-regulation of AQP-4 after TBI. Future studies are needed to assess the mechanisms by which VNS maintains the BBB.

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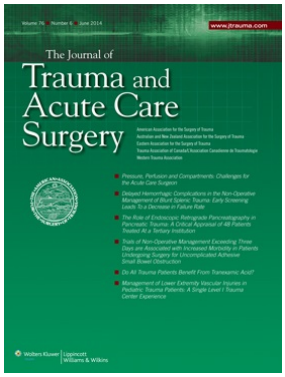

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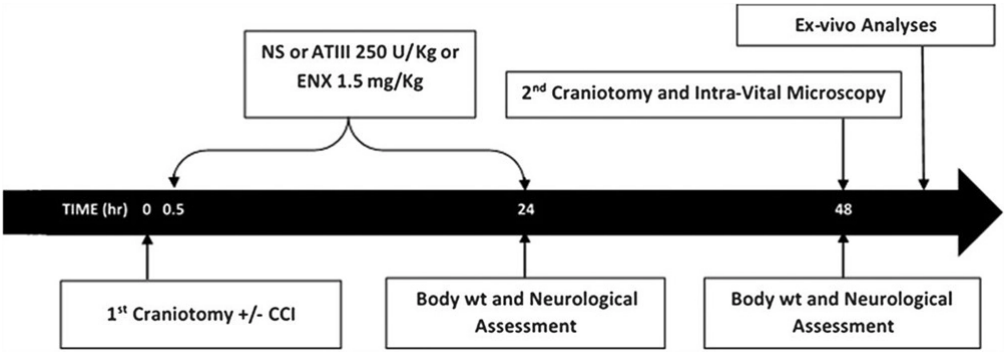

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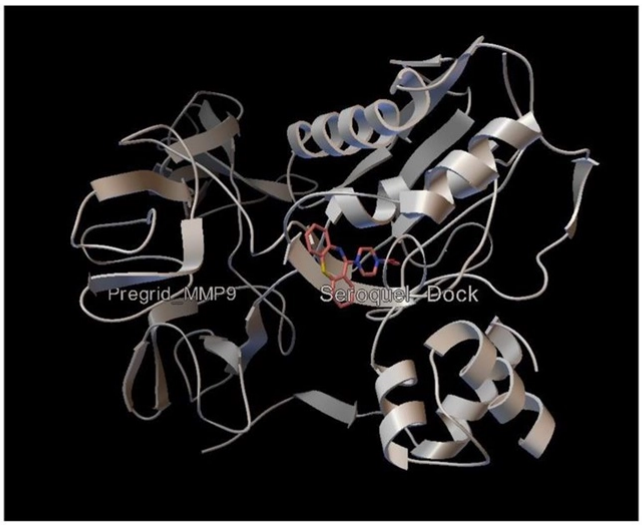
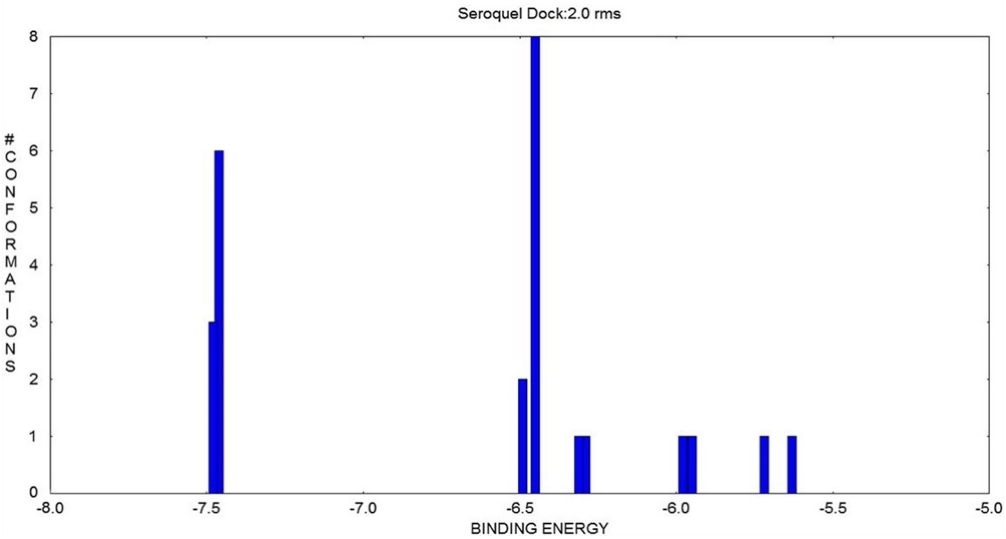




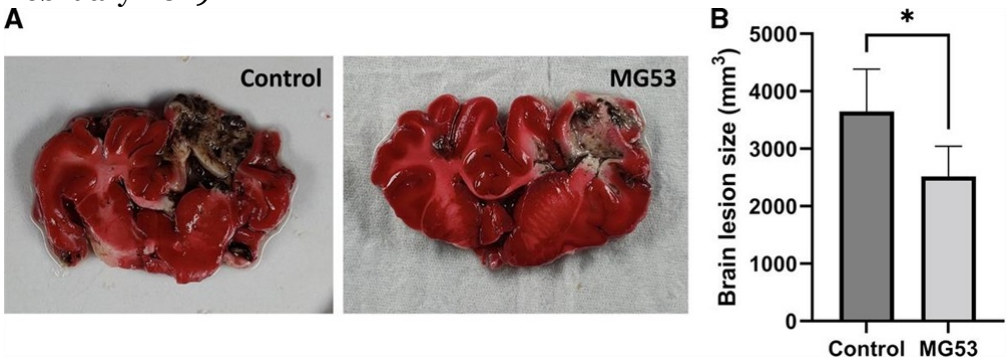
[Quetiapine protects the blood-brain barrier in traumatic brain injury]

November 2018

Gene		Sequence
NKCC1	Forward	3′-CATGGTGTCAGGATTTGCAC-5′
	Reverse	3′-AACCTTTCGCAAACATCTGG-5′
VEGF	Forward	3′-GCCCATGAAGTGGTGAAGTT-5′
	Reverse	3′-TATGTGCTGGCTTTGGTGAG-5′
Occludin	Forward	3′-TCTCAGCCGGCATACTCTTT-5′
	Reverse	3′-ATAGGCTCTGTCCCAAGCAA-5′
Claudin-5	Forward	3′-CGCTTGTGGCACTCTTTGT-5′
	Reverse	3′-ACTCCCGGACTACGATGTTG-5′
GAPDH	Forward	3′-ACAGCAACAGGGTGGTGGAC-5′
	Reverse	3′-TTGAGGGTACAGCGAACTT-5′

- ## NF-κB mediates early blood-brain barrier disruption in a rat model of traumatic shock

  February 2019



- ## Recombinant human MG53 protein attenuates brain lesion size in a large animal model of traumatic brain injury

  November 2022

TABLE 1 - Baseline Characteristics of Study Sample

	No TBI (n = 732)	TBI (n = 291)	*p*
Age, y	34.1 (26.2–47.7)	35.9 (27.0–51.8)	0.0531
Female sex	136 (18.6)	75 (25.8)	0.0103*
ISS	10.0 (4.0–20.5)	29.0 (22.0–38.0)	<0.0001*
AIS head score	0.0 (0.0–0.0)	4.0 (3.0–5.0)	<0.0001*
AIS chest score	0.0 (0.0–3.0)	2.0 (0.0–3.0)	0.0004*
AIS abdomen/pelvis score	0.0 (0.0–2.0)	0.0 (0.0–2.0)	0.0150*
AIS extremities score	0.0 (0.0–2.0)	0.0 (0.0–2.0)	0.1342
Blunt trauma	316 (43.2)	242 (83.2)	<0.0001*
Prehospital SBP, mm Hg	108.0 (88.0–130.0)	110.0 (86.0–140.0)	0.2418
ED SBP, mm Hg	120.0 (93.5–140.0)	122.0 (92.0–142.0)	0.2138
Prehospital GCS score	15.0 (12.0–15.0)	5.0 (3.0–12.0)	<0.0001*
ED GCS score	15.0 (13.0–15.0)	6.0 (3.0–13.0)	<0.0001*
ED temperature, °C	36.7 (36.4–36.9)	36.5 (36.0–36.9)	0.0027*
Shock	185 (25.3)	97 (33.3)	0.0092*
ED BD, mEq/L	5.4 (3.0–9.2)	7.4 (5.0–10.5)	0.0007*
ED platelet count	251.0 (205.0–304.0)	252.5 (196.0–304.0)	0.6560
ED INR	1.1 (1.0–1.2)	1.2 (1.1–1.3)	<0.0001*
ED rTEG ACT	113.0 (105.0–128.0)	121.0 (105.0–128.0)	0.3480
ED rTEG angle, degrees	73.4 (67.9–77.0)	72.0 (66.0–75.9)	0.0026*
ED rTEG MA, mm	63.3 (58.5–67.5)	62.4 (56.5–66.3)	0.0246*
ED rTEG LY30, %	1.7 (0.8–2.9)	1.3 (0.6–2.6)	0.0353*
Fibrinolysis phenotype			
Shutdown	122 (17.8)	71 (25.0)	
Physiologic	522 (76.2)	177 (62.3)	<0.0001*
Hyper	41 (6.0)	36 (12.7)	
Massive transfusion in first 6 h	70 (9.6)	47 (16.2)	0.0028*
TXA	22 (3.0)	16 (5.5)	0.0572
ICU LOS, d	2.0 (0.0–4.0)	5.0 (2.0–13.0)	<0.0001*
Ventilator-free days	28.0 (26.0–28.0)	18.0 (0.0–26.0)	<0.0001*
Hospital LOS, d	5.0 (2.0–11.0)	10.0 (3.0–23.0)	<0.0001*
Mortality	58 (7.9)	95 (32.6)	<0.0001*

Continuous variables are displayed as median (interquartile range) and categorical variables are displayed as n (%).
*Statistically significant.
BD, base deficit; INR, international normalized ratio; ICU, intensive care unit; LOS, length of stay.

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[Traumatic brain injury provokes low fibrinolytic activity in severely injured patients]

July 2022

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