T 1760 ANALYSIS OF WAVE III OF BRAIN STEM AUDITORY EVOKED POTENTIAL DURING MICROVASCULAR DECOMPRESSION OF CRANIAL NERVE VII FOR HEMIFACIAL SPASM

Krishnaiah B., Thirumala P.D., Habeych M.E., Crammond D.J., Balzer J., (Pittsburgh, PA)

Center for Clinical Neurophysiology UPMC

Introduction

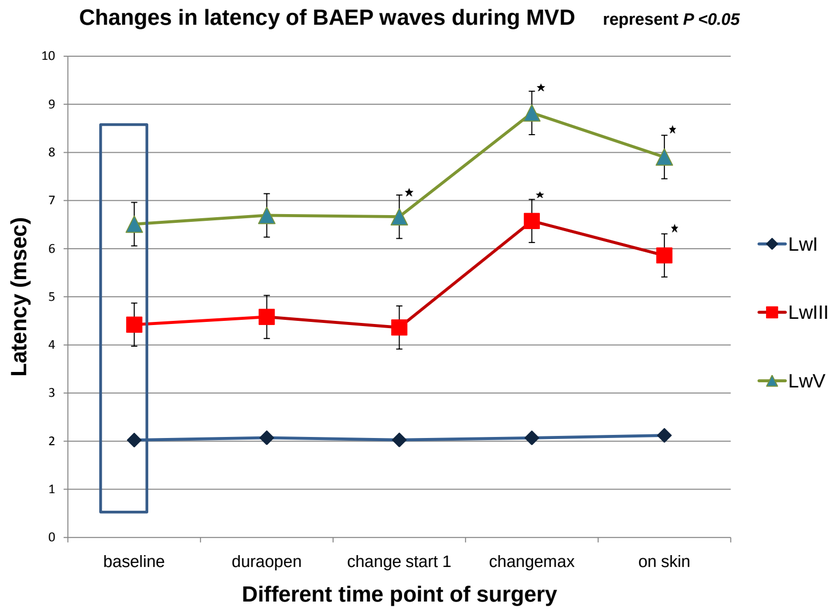
Microvascular decompression (MVD) of cranial nerve (CN) VII is an effective treatment for hemifacial spasm HFS. An infrequent but significant risk of MVD is hearing loss (HL) post operatively caused by damage to CN VIII. Intraoperative monitoring (IOM) of Brainstem Auditory evoked Potential (BAEP) during MVD has substantially decreased the incidence of HL[1].

Objective

To evaluate changes in amplitude and latency of wave III (AwIII, LwIII) of BAEP during MVD and its association with postoperative HL. Previous studies have shown changes in wave III (wIII) is an early sign of auditory nerve injury[2].

Methods

Retrospective analysis of 94 patients who underwent BAEP monitoring during MVD. We did one way ANOVA using mean, median and standard errors of wIII at different time periods and compared it with baseline to find its significance. Due to skewed distribution, the statistical significance of differences in the delay of waves was determined by Kruskal-Wallis test, and the Levene test was to determine if individual group variances were similar. HL is defined as a change in pure tone audiometry >50 dB and/or speech discrimination score of <50%[3]. The results were classified into patients who did not have HL has Group I (n=84) and who had HL as Group II (n=10).



Results of statistical analysis - No HL &HL

LwIII(ms)	Group I			Group II			P-value
	MEAN	SD	MEDIAN	MEAN	SD	MEDIAN	
Baseline	4.44	0.38	4.50	4.40	0.33	4.35	0.513
Change MAX, peak III	6.62	2.36	5.80	8.31	3.58	6.40	0.184
On-Skin, peak III	5.66	2.14	5.00	7.31	3.54	5.40	0.314
Magnitude Change MAX vs Baseline, peak III	2.17	2.34	1.40	4.01	3.65	1.70	0.084
Magnitude Change MAX vs Dura open, peak III	2.05	2.39	1.20	3.79	3.54	1.60	0.173
Magnitude ON SKIN vs Baseline, peak III	1.22	2.12	0.50	3.01	3.54	1.30	0.186
Magnitude ON SKIN vs Change max, peak III	-1.01	2.15	-0.70	-1.00	2.27	0.00	0.413
AwIII (μV)							
Baseline	0.13	0.08	0.12	0.14	0.07	0.12	0.475
Change MAX, peak III	0.08	0.07	0.07	0.05	0.06	0.05	0.225
On-Skin, peak III	0.11	0.08	0.09	0.06	0.07	0.04	0.051
% Change MAX vs. baseline, peak III	83.83	113.92	56.25	41.92	49.46	36.00	0.152
% Change MAX vs. Dura open, peak III	72.19	93.65	45.45	37.96	43.25	41.67	0.193
% On skin vs. baseline, peak III	105.09	120.27	72.73	36.83	37.14	45.45	0.015
% On skin vs. Change max , peak III	186.21	204.07	114.58	78.44	59.15	80.00	0.155

Results

Amplitude or latency of wave III were significant between groups ($p<0.05$) at on skin but regression analysis did not find wave III changes to increase the odds of HL. Correlation analysis did not find any positive relation between wIII and wV. Wave III seems to be changed early (Dura open and change start I) in both amplitude and latency but none of them is statistically significant with one way ANOVA analysis.

Conclusion

wIII is not the earliest change and it does not precede wV loss. Changes in wave III did not increase the odds of HL in patients who underwent BAEPs during MVD and it is highly unpredictable. This information might be valuable to evaluate the value of wIII as alarm criteria during MVD to prevent HL.

References

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2. Matthies C, Samii M. Management of vestibular schwannomas (acoustic neuromas): the value of neurophysiology for intraoperative monitoring of auditory function in 200 cases. Neurosurgery 1997;40:459-466; discussion 466-458

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