Analysis of the effect of dexmedetomidine on platelet function with TEG®6s PlateletMapping® assay in whole blood

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Introduction

- ✓ Dexmedetomidine (DEX) is a highly selective alpha2-adrenoceptor (alpha2-AR) agonist.
- ✓ A recent study has shown that high doses of DEX induced adenosine diphosphate (ADP)-triggered platelet aggregation via alpha2-ARs in platelet-rich plasma obtained from healthy volunteers.¹⁾
- ✓ However, the effects of DEX on platelet function and clot formation in whole blood remain unclear.
- ✓ The aim of the present study was to evaluate the effects of DEX on platelet function and clot formation in whole blood using TEG®6s and Platelet Mapping® ADP assay.

Materials and Methods

- Healthy, male, non-smoker volunteers (25–35 years old) were enrolled in the study (n = 6). Whole blood was obtained and the blood was immediately dispensed into three heparinized blood collection tubes with a volume of 4 ml.
- DEX diluted with normal saline (NS) was added to whole blood in each tube to make final concentrations of 10 nM (DEX10) and 100 nM (DEX100). An equal volume of normal saline was added to blood in the control group.
- The whole blood samples were incubated at room temperature for 3 min and approximately 0.4 ml of the sample was pipetted into the entry point of the Platelet Mapping® ADP Cartridge.

Figure 1. Experimental protocol.

Figure 2. TEG®6s Platelet Mapping®.

Statistical analysis

- Variables are expressed as medians [interquartile range], and they were compared between the three groups by the Wilcoxon matched-pairs signed rank test. Applying Bonferroni correction for multiple comparisons, $p < 0.017$ was considered statistically significant.
- A sample size of six subjects was estimated to provide more than 90% power at the 0.017 level of statistical significance to detect a 15% change²⁾ in MA_{ADP} .

Results

Figure 3. Box-and-whisker plots of platelet function evaluated by using TEG6s® and PlateletMapping® ADP assay (n = 6). P values are expressed in the figure.

Table 1. Other parameters measured by kaolin with the HKH assay.

	Control	DEX10	DEX100	P value (95% confidence interval of the difference)		
				Control vs. DEX10	Control vs. DEX100	DEX10 vs. DEX100
R-time min	8.2 [7.7–8.4]	8.8 [8.4–9.4]	8.2 [7.6–9.1]	0.563 (-1.7 to 1.2)	<0.999 (-0.6 to 0.8)	0.469 (-1.8 to 1.2)
Alpha degree	65.1 [63.2–67.2]	65.7 [63.0–66.5]	66.2 [64.0–68.5]	0.938 (-3.6 to 3.7)	0.094 (-0.3 to 2.0)	0.313 (-2.9 to 3.3)
K-time min	2.1 [1.9–2.3]	2.0 [1.9–2.3]	2.0 [1.8–2.3]	0.813 (-0.5 to 0.3)	0.375 (-0.2 to 0.1)	0.656 (-0.2 to 0.4)

Conclusions

- ❑ DEX did not alter clot strength induced by platelet activation, as shown by using TEG®6s and Platelet Mapping® ADP assay in whole blood.
- ❑ Further clinical studies are needed to elucidate the effect of DEX on platelet function *in vivo*.

References

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2. Wesley MC, McGowan FX, Castro RA, Dissanayake S, Zurakowski D, Dinardo JA. The effect of milrinone on platelet activation as determined by TEG platelet mapping. Anesth Analg. 2009;108:1425-1429.