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Authors' reply to the comment by Kox et al.

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Sir: Recent reports provide evidence of a link between parasympathetic and sympathetic nervous activity and inflammation. Inflammatory cytokines produced in damaged tissues may activate afferent signals that input to the nucleus tractus solitarius resulting in activation of vagus efferent (i.e. parasympathetic) activity, which inhibits cytokine synthesis through the cholinergic

anti-inflammatory pathway [1]. Information can also be relayed to the hypothalamus and the dorsal vagal complex to stimulate the release of ACTH, thereby activating the humoral anti-inflammatory pathway. Activation of the sympathetic nervous system, resulting in increased circulating catecholamines can also impact on inflammation. Using the spectral analysis of heart rate Kawahara et al. [2] recently documented an apparently enhanced parasympathetic activity during the acute phase in patients following subarachnoid haemorrhage. Dr. Kox and colleagues raise important issues, the ex vivo stimulation of immune cells obtained from patients with subarachnoid haemorrhage has merit, and we agree that it is important to study the parasympathetic nervous system and the "cholinergic anti-inflammatory pathway" in the setting of subarachnoid haemorrhage. The latter was beyond the scope of our recent investigation given that our primary focus, and expertise, rests with inves-

tigations of the sympathetic nervous system and its impact in health and disease.

References

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