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*British Journal of Anaesthesia*, 120 (5): 890-891 (2018)

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## Can nitric oxide tame the ketamine tiger?

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Ketamine is notorious for causing diverse central nervous system effects, and for its promiscuous binding to many different neuronal receptors. Indeed, ketamine is an established pharmacological model for schizophrenic psychosis.<sup>1</sup> In this issue of the *British Journal of Anaesthesia*, Jonkman<sup>2</sup> shows that it was possible to attenuate the psychedelic or psychotomimetic effects of racemic ketamine—but not those of its S(+)-enantiomer—by infusing a low dose of sodium nitroprusside. The underlying rationale is that the ketamine blockade of N-methyl-D-aspartate (NMDA) type glutamate receptors induces multiple downstream intracellular effects, primarily via blockade of Ca<sup>2+</sup> entry. The resulting net decrease in the evanescent inter- and intra-cellular messenger nitric oxide causes a decrease in cyclic guanosine monophosphate (cGMP).

As with all good research, this paper generates more important and wide ranging questions, which in turn expand our understanding in new directions. The psychedelic effects of ketamine occur at concentrations that overlap with those required for effective analgesia. Could sodium nitroprusside be used clinically to allow ketamine to be a more effective analgesic drug? Given the potential for harm caused by inadvertent errors in sodium nitroprusside delivery and dosing, and the concomitant necessity for close monitoring, this would seem unlikely. However, use of other more easily administered nitric oxide donor drugs, such as transdermal nitroglycerin, deserves further evaluation. The authors are correctly cautious as to the potential for clinical use—but mainly because of another question.

Tam metin



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