# Haemodynamic stability of pre-hospital Ketamine anaesthesia following a return of spontaneous circulation after a cardiac arrest due to a presumed medical aetiology

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## Background

Post-ROSC patients can present a challenge in a pre-hospital environment due to cerebral agitation, ventilatory failure and airway compromise. Patients often require pre-hospital emergency anaesthesia (PHEA) to facilitate onward transfer to definitive care, allowing local Emergency Department bypass in favour of regional centres capable of primary percutaneous coronary intervention. Whilst the efficacy and haemodynamic stability of various agents for PHEA in trauma patients have been well described [(1)], the evidence for the optimal regime for post-ROSC PHEA is minimal.

MAGPAS Air Ambulance utilise a Ketamine-Rocuronium regime for post-ROSC PHEA. All patients receive 1mg.kg$^{-1}$ Rocuronium and a 0.1mL.kg$^{-1}$ Ketamine infusion after a variable induction dose of Ketamine relative to their presentation:

***MAP >70mmHg & agitated/lively → 1mg.kg$^{-1}$***

***MAP >70mmHg & non-lively → 0.5mg.kg$^{-1}$***

***Peri arrest/MAP <70mmHg → Nil***

## Study Aims

1) We sought to investigate the haemodynamic stability of a Ketamine-Rocuronium regime in post-ROSC PHEA.

2) We hoped to compare this to the data presented by Miller et al[(2)], who described a post-ROSC Fentanyl-Midazolam-Rocuronium regime in post-ROSC patients.

## Methods

A retrospective case review was conducted for all patients who achieved a ROSC and received PHEA between 1st October 2015 and 31st May 2019.

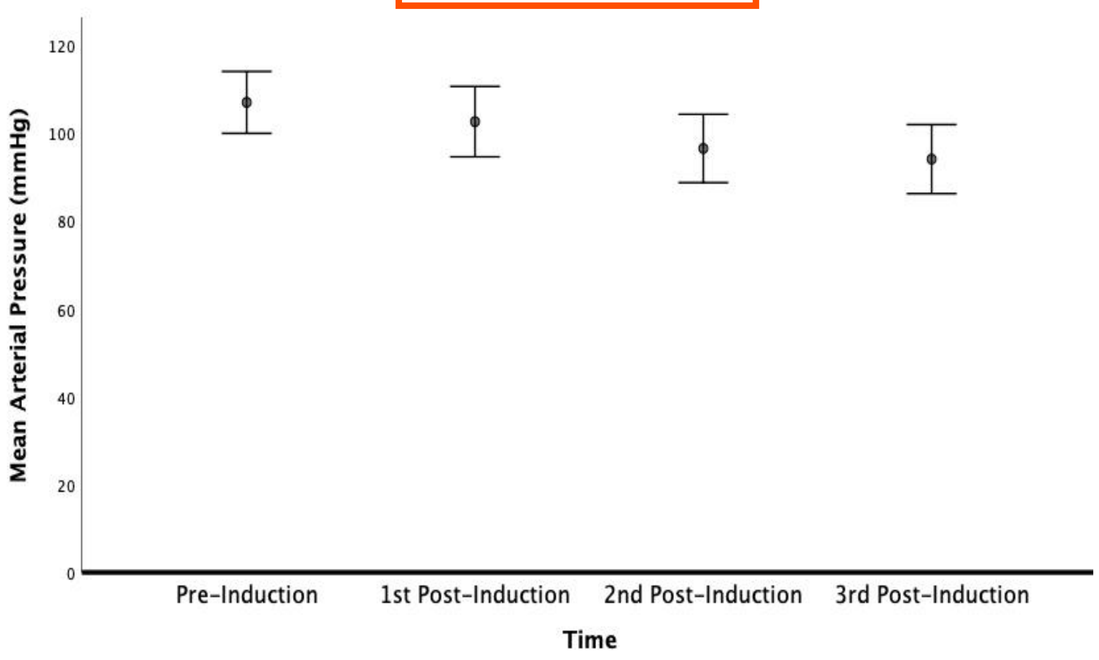
Patients were included if they were >18, had suffered a non-traumatic cardiac arrest and subsequent ROSC, received a Ketamine induction of PHEA and had sufficient haemodynamic observations available for analysis.

Sufficient observations include heart rate (HR) and mean arterial pressure (MAP) within 3 minutes prior to induction and subsequently at 3, 6 and 9 minutes.

## Results

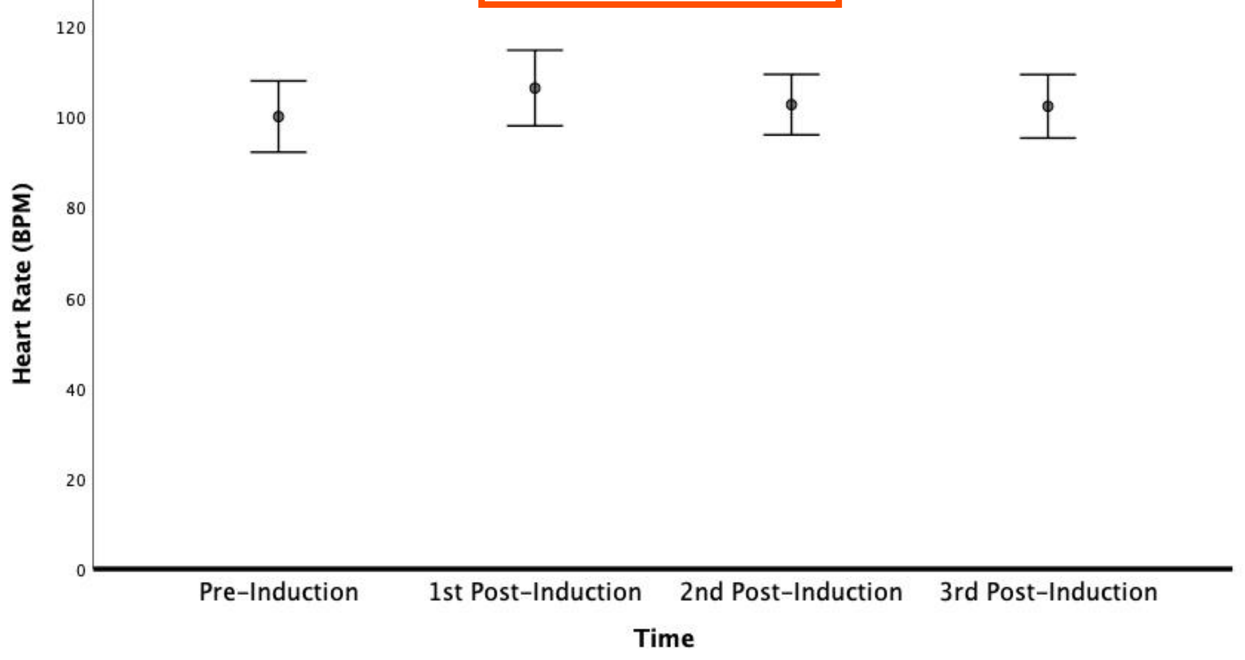
55 patients were included for analysis.

### MAP




- Following induction, the MAP differed significantly over time *(repeated measures ANOVA with Huynh-Feldt correction).*
- Only the 9 minute MAP was statistically significantly different (p=0.036) compared to the pre-induction value *(post-hoc tests using Bonferroni correction).*
- There was no statistically significant difference between the pre-induction MAP and values at 3 (p=1.0) or 6 minutes (p=0.052) or between the MAP at 3 and 6 minutes (p=0.2), 3 and 9 minutes (p=0.21) and 6 and 9 minutes (p=0.1).

### HR




- Following induction, there was no statistically significant change in HR over time (p=0.31) *(repeated measures ANOVA with Huynh-Feldt correction).*
- There was no statistically significant difference between the pre-induction HR and the values recorded at 3 (p=0.351), 6 (p=1) and 9 (p=1) minutes *(post-hoc tests using Bonferroni correction).*
- There were no changes between the HR at 3 and 6 minutes, 3 and 9 minutes and also 6 and 9 minutes (p=1).

## Discussion

- Ketamine is often described as a haemodynamically stable agent due to sympathetic and adrenocortical stimulation increasing endogenous catecholamine and norepinephrine levels. However it is thought that this effect is seldom seen in patients with limited myocardial reserve and increased myocardial oxygen demand[(3)(4)].
- Waxman et al found that Ketamine reduces left ventricular output by 50%[(3)] and other studies have failed to demonstrate haemodynamic stability following Ketamine induction in critically ill patients. This may be as Ketamine relies on secondary sympathomimetic mechanisms to maintain haemodynamic stability, which are likely to be absent or reduced in catecholamine deplete patients[(4)].
- However, it is possible that the haemodynamic changes described are due to the pre-existing haemodynamic instability from myocardial dysfunction and vasoplegia caused by systemic hypoperfusion and reperfusion in addition to the underlying aetiology. Ketamine has an onset of action within seconds, so any haemodynamic effects would be expected quickly whereas we found changes only at 9 minutes.
- Whilst we saw an undesirable drop in MAP 9 minutes after induction, we were able to observe stable MAPs prior to this point, minimal changes in HR and relatively few episodes of hypertension and tachycardia which are valuable features of an induction agent for post-ROSC PHEA.
- Given that Ketamine is the agent of choice in trauma PHEA, there is a significant benefit in using a common agent for both allowing clinicians to become familiar and well-rehearsed in its use in a pre-hospital environment therefore increasing cognitive bandwidth and decreasing the opportunity for procedural error.

1) Lyon RM, Perkins ZB, Chatterjee D, Lockey DJ, Russell MQ. Significant modification of traditional rapid sequence induction improves safety and effectiveness of pre-hospital trauma anaesthesia. Critical Care. 2015 Dec;19(1):134.
2) Miller M, Groombridge CJ, Lyon R. Haemodynamic changes to a midazolam–fentanyl–rocuronium protocol for pre-hospital anaesthesia following return of spontaneous circulation after cardiac arrest. Anaesthesia. 2017 May;72(5):585-91.
3) Waxman K, Shoemaker WC, Lippmann M. Cardiovascular effects of anesthetic induction with ketamine. Anesthesia and analgesia. 1980 May;59(5):355-8.
4) Morris C, Perris A, Klein J, Mahoney P. Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent?. Anaesthesia. 2009 May;64(5):532-9.

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