Evolution of vital parameters during endoscopist-controlled sedation in patients breathing room air.

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Introduction:

Sedation using a combination of opiates and benzodiazepines during colonoscopy improves patient comfort and facilitates the endoscopy and is still commonly used. However, the risk for hypoxemia, hypoventilation and pulmonary aspiration mandates dedicated monitoring. In this study we investigate the evolution of several vital signs over the course of the procedure.

Material:

After obtaining institutional approval and informed consent, 230 consecutive patients were included in this single-center prospective observational study. The patients were breathing room air. Midazolam and pethidine was administered at the discretion of the endoscopist. Respiration Rate, transcutaneous CO_2 tension ($P_{tc}CO_2$), end-tidal CO_2 tension ($P_{et}CO_2$), Peripheral arterial oxygen saturation (S_pO_2), Bispectral index (BIS), mean arterial pressure (MAP) and heart rate were recorded.

Results:

Sedation caused significant changes in vital signs.

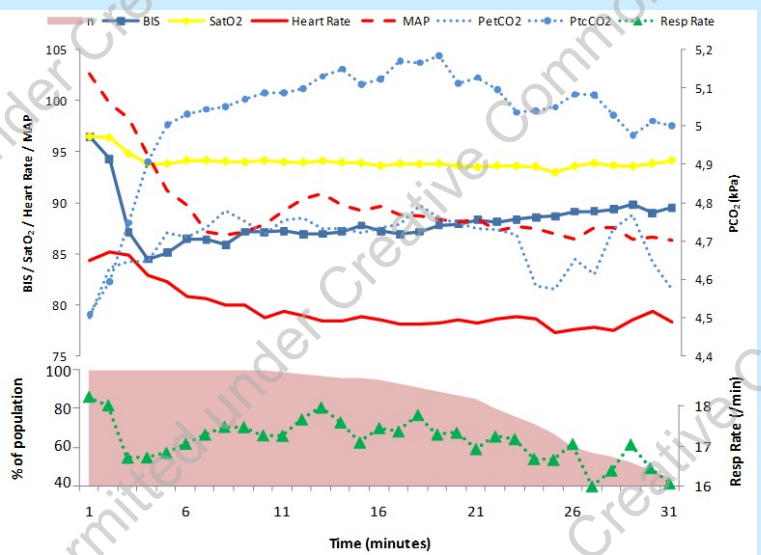

Figure 1 : The evolution of the average value of all parameters together with the percentage of the population still under sedation at any moment after induction (n).

	Ultimate value	Incidence(%)	>Onset time (sec)	Total time (sec)
Sp < 90%	90(5)^L	36	655(498)	226(325)
BIS < 75	77(8)^L	29	389(419)	224(299)
BIS < 70		17	321(340)	126(188)
↑$P_{et}CO_2$ > 1kPa	5.4(0.6)^H	27	599(537)	270(509)
↑$P_{et}CO_2$ > 2kPa		2	255(364)	939(951)
↑$P_{et}CO_2$ > 3kPa		1	10(7)	1150(1110)
↑$P_{tc}CO_2$ > 1kPa	5.5(0.7)^H	31	773(578)	581(529)
↑$P_{tc}CO_2$ > 2kPa		4	1102(754)	474(398)
↑$P_{tc}CO_2$ > 3kPa		0	-	-
Resp rate <8/min	10(4)^L	15	643(574)	226(365)
Heart rate <50 BPM	67(18)^L	9	424(267)	224(342)
MAP < 80 mmHg	76(18)^L	59	431(520)	765(573)
MAP < 70 mmHg		36	525(533)	564(452)

Discussion:

Benzopiate sedation resulted in significant intra- and inter-patient variability in respiratory rate, increased dead space ventilation, hypercapnia and hypoxia.

The high incidence of hypoxia suggests additional O_2 administration could be considered. However, although this could prevent hypoxia, it may further delay detection of hypoventilation and oversedation by S_pO_2. Together with the absence of hypoxic drive, this would increase the risk and degree of hypercapnia. We observed a superior reliability of the $P_{tc}CO_2$. However, we may speculate that additional O_2 administration will result in a preserved S_pO_2, and subsequent CO_2 accumulation. The consequential increased hypercapnic drive could restore an adequate tidal volume and therefore the reliability of $P_{et}CO_2$.

Conclusion:

Sedation with midazolam/pethidine caused significant hypoxia, hypercapnia and hypoventilation caused by dead space ventilation. Combined monitoring of CO_2 and S_pO_2 is required during sedation.