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Cardiopulmonary Functions in Dogs under Propofol, Ketamine and Isoflurane Anaesthesia Premedicated with Glycopyrrolate, Dexmedetomidine and Butorphanol

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ABSTRACT

Background: The study was conducted to evaluate the cardiopulmonary functions in dog under propofol, ketamine and isoflurane anaesthesia premedicated with dexmedetomidine and butorphanol.

Methods: Four groups of dogs (A,B,C and D) comprising of six animals in each groups were premedicated with glycopyrrolate @ 0.01 mg/kg, dexmedetomidine @5µg/kg IV and Butorphanol @ 0.1mg/kg IV. Induction was done with propofol (A and B) and with ketamine (C and D). The anaesthesia was maintained with isoflurane (A and C), propofol (B) and ketamine (D). The cardiopulmonary functions were recorded at 0 minute (before premedication) and 20 minutes, 40 minutes and 60 minutes.

Result: The heart rate decreased significantly in Group B while there was a significant gradual increase in heart rate in Group D. A significant decrease in respiratory rate was observed in all the groups with a lowest value in group D. The systolic pressure decreased significantly in Group A, B and C but in Group D, the systolic pressure decreased initially at 20 minute. The diastolic pressure decreased significantly in Group A and Group B and but in group D, the diastolic pressure decreased at 20 minute. A significant decrease in mean arterial pressure was recorded in Group A, B and C. In Group D, a decrease in the mean arterial pressure was noticed at 20 minute. The SpO_2 level remained near the base line values with slight variation in Group A and C where as the values remained at lower level from the base line value in Group B and D. The $EtCO_2$ level showed non-significant changes in Group A and C. In Group B and D, the $EtCO_2$ levels increased non-significantly with the highest value recorded in Group D. The ECG parameters remained within the normal limit with slight variation according to the heart rate.

Key words: Anaesthesia, Cardiopulmonary functions, Isoflurane, Ketamine, Propofol.

INTRODUCTION

Anaesthetic protocols involving single agent have largely been abandoned in favour of protocols that incorporate multiple agents from different classes to achieve sedation, analgesia and muscle relaxation together (Bednarski, 2011). Combination of two or more drugs or techniques enables to achieve all the components of anaesthesia and at the same time the side effects of an individual agent is mitigated by the others. For this purpose, different classes of preanaesthetic agents are used along with induction or maintenance agents to achieve cardiovascular and respiratory stability, reduction of secretions together with ideal components of anaesthesia. In order to optimize the advantages afforded by premedication, it is important to select premedicants, induction and maintenance agents based on the need of the individual patient, rather than using a single 'blanket' combination for all animals (Mc Kelvey and Hollingshead, 1994). Inhalation anaesthetics are used widely for the anaesthetic management of animals. They are unique among the anaesthetic drugs because they are administered and in large part removed from the body via the lungs. Inhalation agents are preferred because their pharmacokinetic characteristics favour predictable and rapid adjustment of anaesthetic depth. However, a special

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apparatus is necessary to deliver the inhalation agents (Natalini, 2001). Total intravenous anaesthesia (TIVA) either with repeated bolus injection or continuous infusion has been emphasized in the recent years as it makes the anaesthetic management easier without any sophisticated instruments. The choice of anaesthetic agent or its combination and method of administration is mainly targeted to achieve safe and effective anaesthesia without major alteration in body homeostasis. Therefore, the present study was undertaken to the cardiopulmonary functions with isoflurane, propofol and ketamine anaesthesia in glycopyrrolate, dexmedetomidine and butorphanol premedicated dogs.

MATERIALS AND METHODS

Twenty four female dogs between 1-6 years of age and irrespective of breeds presented for elective ovariohysterectomy were randomly divided into four groups *viz.* Group A, B, C and D comprising six animals in each. The animals were fasted for 12 hours and water was withheld for 8 hours prior to the procedure. The surgical site was prepared in all the animals and intravenous PTFE catheter with injection valve and luer lock plug was fixed and slow intravenous fluid (NSS) was administered throughout the procedure. All the animals were premedicated with glycopyrrolate @ 0.01 mg/kg IM. Fifteen minutes later the animals were given dexmedetomidine @5 µg/kg IV and Butorphanol @ 0.1 mg/kg IV. Induction of anaesthesia was done with propofol IV till effect in Group A and B and with ketamine IV till effect in Group C and D. Anaesthesia was maintained with isoflurane with fresh gas flow rate 10 ml/kg/minute at a higher value of 3-5% for initial 5 minutes for stabilization and then the vaporizer settings were changed accordingly to maintain surgical plane of anaesthesia in Group A and C. In Group B and D anaesthesia was maintained with propofol continuous rate infusion @ 0.2-0.5 mg/kg/min and ketamine continuous rate of infusion @ 0.002-0.02 mg/kg/min.

Heart rate was recorded using a stethoscope before induction and by multi-parameter monitor during maintenance of anaesthesia and was expressed in beats per minute. Respiratory rate was recorded by observing the thoracic movements before induction and by multi-parameter monitor during maintenance of anaesthesia and was expressed in breaths per minute. The systolic pressure, diastolic pressure, mean arterial pressure (mmHg), SpO_2 (%) and $EtCO_2$ (mmHg) were measured by using multi-parameter monitor. Electrocardiography (ECG) was recorded by lead II at paper speed of 50 mm/second with alligator clips connected to animal's limbs. All the parameters were recorded at 0 minute (before premedication) and 20 minutes, 40 minutes and 60 minutes (during maintenance).

The data obtained were analyzed using statistical package SPSS version 16. One way ANOVA based on Fisher's least significant difference method was used to determine the significant difference among the different treatments and time intervals. The significant values in the ANOVA were further tested through Duncan multiple range test. Results are presented as mean±SE and differences were considered significant when $P<0.05$.

RESULTS AND DISCUSSION

The mean heart rate at 0 min did not show any significant difference among the groups. However, there were significant changes in mean heart rate among the groups at 20 min, 40 min and 60 min of observation period. The values of heart rate were found to be lower in Group B and higher in Group D in the subsequent observation periods. The heart rate at different time intervals decreased non-significantly in Group A with highest fall at 20 min. In Group B, the heart rate decreased significantly ($P<0.05$) with the lowest value at 60 min. In Group C, the heart rate increased initially at 20 min and thereafter decreased gradually; however, the changes were statistically non-significant. The heart rate showed a significant ($P<0.05$) gradual increase from 0 min till the end of the observation period in Group D. The values in heart rate recorded in different treatment groups and at different time interval were within the physiological limit. The initial non-significant decrease in heart rate observed in Group A and B might be due to the effect of butorphanol and dexmedetomidine as both the drugs caused decrease heart rate in animals (Greene *et al.,* 1990; Pypendop and Verstegen, 1998). The dexmedetomidine causes a vasoconstriction in both the pulmonary and systemic circulations initially and then elicits a decrease in heart rate and cardiac output with a slight depressive effect on ventilation (Pascoe, 2015). In Group C and D, the rise in heart rate might be due to cardiac stimulatory effects of ketamine (Kumar *et al.,* 2014). Ketamine stimulates the cardiovascular system resulting in an increase in heart rate principally through the sympathetic nervous system (Kolawole, 2001). Ketamine has an antagonistic interaction with mono-aminergic, muscarinic and nicotinic receptors and produces anticholinergic symptoms (Pai and Heining, 2007).

A significant ($P<0.01$) difference in respiratory rate among the groups was observed at 60 min of observation period with a lowest value in Group D. A decreasing trend in respiratory rate from pre-induction values was observed in all the groups till the end of the observation period. The changes were found to be significant in Group A ($P<0.05$) and Group B, C and D ($P<0.01$). The decreased respiratory rate observed in all the groups might be due to depression of respiratory centre caused by isoflurane (Galloway *et al*., 2004), ketamine (Narayanan *et al.,* 2011) and propofol (Suarez *et al*., 2012). The respiratory depression with ketamine anaesthesia might also be due to airway relaxation by acting on various receptors, inflammatory cascades and bronchial smooth muscles as reported by Goel and Agrawal (2013).

The systolic pressure differed significantly among the groups at 20 min ($P<0.05$), 40 min and 60 min ($P<0.01$). The systolic pressure gradually decreased with significance ($P<0.01$) throughout the period of observation in Group A, B and C. In Group D, the systolic pressure decreased initially at 20 min and thereafter it gradually returned towards the

pre-induction values at 60 min. The changes in systolic pressure in Group D were significant (P<0.05). The reduction in systolic pressure in all the groups might be due to peri-operative sympatholysis and stimulation of central α2 and imidazoline receptors (Naaz and Ozair, 2014). The decreased systolic pressure might also be due to vasodilatation and decreased contractility of the heart due to effect of propofol and isoflurane (Ilkiw *et al.,* 1992). Propofol decreases blood pressure due to a decrease in peripheral vascular resistance, decreased sympathetic outflow and myocardial depression (Cullen and Reynoldson, 1993), peripheral vasodilatation and direct negative inotropic action (Jena *et al*., 2014). In Group C, the cardiovascular stimulating effects induced by ketamine might have been prevented by dexmedetomidine, butorphanol and isoflurane and in Group D the rise in systolic pressure in later period might be attributed to the cardiac stimulatory effects of ketamine as a result of its sympathomimetic effects mediated from within the central nervous system (Lin, 2007).

There was a significant variation in diastolic pressure at 20 min (P<0.05) and during the subsequent observation periods (P<0.01) among the groups. The diastolic pressure decreased significantly in Group A (P<0.05) and Group B (P<0.01) after administration of anaesthesia till the end of the observation period. A gradual decrease in diastolic pressure was also observed in Group C; however, the changes were statistically not significant. In group D, the diastolic pressure decreased initially from the base line value at 20 min and gradually increased towards the end of the observation period. The changes in the diastolic pressure in Group D at different observation periods were not significant. The decreased diastolic pressure recorded in the present study might be due to arterial and venous vasodilatation and decreased contractility of the heart due to effect of propofol and isoflurane (Ilkiw *et al.,* 1992) or due to peri-operative sympatholysis and stimulation of central alpha 2 adrenargic receptors (Naaz and Ozair, 2014). Propofol decreases blood pressure due to a decrease in peripheral vascular resistance, decreased sympathetic outflow and myocardial depression (Cullen and Reynoldson, 1993), peripheral vasodilation and direct negative inotropic action (Jena *et al*., 2014). In Group C, it might be due to the effect of dexmedetomidine, butorphanol and isoflurane. In Group D, the rise in diastolic pressure in later period might be attributed to the cardiac stimulatory effects of ketamine due to its sympathomimetic effects mediated from within the central nervous system (Lin, 2007).

The mean arterial pressure varied significantly among the groups at 20 min (P<0.05) and also at 40 min and 60 min of observation (P<0.01). A significant (P<0.01) decrease in mean arterial pressure was recorded at 20 min, 40 min and 60 min in Group A, B and C as compared to the base line values. In Group D, a decrease in the mean arterial pressure was noticed at 20 min and thereafter, it reached near the pre-induction value at the end of the observation period. However, the changes in mean arterial pressure in Group D were not significant. Barletta *et al*. (2011) also reported similar observations under ketamine anaesthesia with dexmedetomidine and buprenorphine. Decreased mean arterial pressure observed in Groups A, B and C might be due to arterial and venous vasodilatation and decreased contractility of the heart due to effect of propofol and isoflurane or due to peri-operative sympatholysis and stimulation of central alpha 2 adrenargic receptors. In Group D, the rise in mean arterial pressure might be attributed to the cardiac stimulatory effects of ketamine owing to its sympathomimetic effects mediated from within the central nervous system.

The SpO_2 level remained near the base line values with slight variation in Group A and C. The values of SpO_2 remained at lower level from the base line value at the subsequent observation period in Group B and D. In Group A and C the changes were not significant at different time intervals. The changes were significant (P< 0.01) in Group B and D and the value at 60 min in Group D was near to the critical level. The decreased SpO_2 in Group B and D found in the present study might be due to respiratory depression caused by propofol (Suarez *et al*. 2012) and ketamine along with dexmedetomidine and butorphanol (Santos *et al.,* 2007). The SpO_2 level maintained in Group A and C near the pre-induction value might be due to the administration of oxygen along with isoflurane. Leppanen *et al*. (2006) stated that use of pulse oximetry monitoring (sensor placed in the tongue) in animals were not reliable as haemoglobin saturation measured by arterial blood gas analysis displayed substantially higher values. Pooling of deoxygenated blood in the tongue due to peripheral vasoconstriction may explain the erroneously low hemoglobin saturation values displayed by pulse oximetry in animals.

The $EtCO_2$ level showed slight variation (non-significant) in Group A and C. In Group B and D, the $EtCO_2$ levels increased non-significantly at different observation periods with a highest value recorded in Group D at 60 min. Hypercapnia during anaesthesia were observed by Aguiar *et al.* (2001) with propofol and opioids, Kazuto (2001) with ketamine, Santos *et al.* (2007) with butorphanol and desflurane and Fayyaz *et al*. (2009) with isoflurane, ketamine-diazepam or propofol-diazepam in dogs. The non-significantly increased $EtCO_2$ and respiratory depression in the present study without hypercapnia might be due to the respiratory depression caused by propofol and ketamine. The maintenance of normal $EtCO_2$ in Group A and C observed in the present study might be due to adequate oxygenation throughout the anaesthetic period.

The P-wave duration was recorded with slight variations (non-significant) in all the groups during the study period. No significant increase was recorded for duration of P-wave at different time intervals as compared to the respective pre-induction values. Insignificantly increased P-wave duration observed in the present study in all the groups was also reported by Khurana *et al.* (2014) with butorphanol-diazeapam-propofol and butorphanol-acepromazine-propofol and Rafee *et al.* (2016) with dexmedetomidine-ketamine with

Table: Effects of anesthetic treatments on cardiopulmonary functions at different time intervals in dogs.

Parameters	Group	0 min	20 min	40 min	60 min
	A	119.33±6.63	103.50±5.42^{A}	107.83±4.67^{A}	109.67±3.45^{B}
Heart rate	B	122.83±5.98^{b}	101.67±5.31^{Aa}	99.50±3.42^{Aa}	98.83±3.13^{Aa}
(beats/min)	C	118.83±5.04	127.83±4.61^{B}	122.83±3.30^{B}	120.17±2.80^{C}
	D	120.67±6.00^{a}	133.83±3.48^{Bab}	136.83±5.67^{Cb}	138.33±3.35^{Db}
	A	27.33±2.60^{b}	19.83±2.60^{a}	17.33±2.17^{a}	16.67±1.73^{Ca}
Respiratory rate	B	28.50±3.22^{b}	17.17±1.54^{a}	15.50±1.18^{a}	14.80±1.19^{BCa}
(breaths/min)	C	26.83±3.00^{c}	17.17±1.51^{b}	14.50±0.67^{ab}	13.67±0.49^{Aa}
	D	29.83±3.46^{b}	18.50±2.08^{a}	15.67±2.09^{a}	12.17±0.48^{ABa}
	A	124.67±2.09^{b}	110.50±3.31^{ABa}	104.83±3.35^{Aa}	102.83±1.82^{Aa}
Systolic pressure	B	126.17±2.63^{c}	108.83±3.63^{Ab}	100.67±3.55^{Aab}	98.83±2.97^{Aa}
(mmHg)	C	128.17±2.86^{c}	117.83±1.89^{BCb}	108.33±2.68^{Aa}	105.33±2.89^{Aa}
	D	129.17±1.64^{b}	121.17±2.20^{Ca}	123.83±2.46^{Bab}	127.67±3.33^{Bab}
	A	91.67±3.01^{b}	85.67±2.65A^{Bab}	83.17±2.32^{Bab}	80.17±2.67^{Ba}
Diastolic pressure	B	87.17±3.29^{c}	80.83±1.90^{Abc}	76.17±2.21^{Aab}	73.17±1.35^{Aa}
(mmHg)	C	85.33±3.01	80.83±2.65^{A}	78.17±2.32^{AB}	77.67±2.67^{AB}
	D	93.33±2.58	89.83±1.85^{B}	94.33±1.86^{C}	96.50±2.11^{C}
	A	102.67±2.44^{b}	94.17±2.91^{ABa}	90.50±2.33^{Ba}	87.83±1.82^{Ba}
Mean arterial pressure	B	100.00±2.62^{c}	90.17±1.96^{Ab}	84.00±2.21^{Aa}	81.67±1.31^{Aa}
(mmHg)	C	99.67±2.50^{b}	93.00±1.84^{Aa}	88.17±1.56^{ABa}	87.00±2.05^{ABa}
	D	105.33±2.14	100.50±1.73^{B}	104.00±1.95^{B}	106.83±2.24^{C}
	A	96.17±0.60	95.83±0.91	95.17±1.14^{B}	96.33±0.92^{B}
SpO_2	B	97.17±0.91^{b}	92.17±1.90^{a}	89.67±1.94^{Aa}	88.83±1.54^{Aa}
(%)	C	96.33±0.67	94.67±0.99	93.83±0.98^{B}	94.67±1.63^{B}
	D	97.33±0.33^{c}	91.83±1.17^{b}	87.83±0.91^{Aa}	85.67±1.71^{Aa}
	A	34.33±2.11	35.83±1.17	36.67±1.15	35.67±1.31
$ETCO_2$	B	35.83±1.08	37.17±1.05	38.17±2.12	37.17±1.64
(mmHg)	C	34.83±2.48	36.33±2.42	37.17±2.39	36.83±2.27
	D	35.17±2.63	38.17±2.63	39.33±2.30	39.83±2.14
	A	0.035±0.002	0.036±0.002	0.036±0.002	0.035±0.003
P-Wave duration	B	0.037±0.003	0.039±0.002	0.038±0.002	0.038±0.001
(Sec)	C	0.036±0.003	0.038±0.002	0.037±0.001	0.036±0.001
	D	0.039±0.003	0.040±0.002	0.041±0.002	0.040±0.001
	A	0.18±0.02	0.16±0.01	0.17±0.02	0.17±0.02
P-Wave amplitude	B	0.19±0.01	0.17±0.01	0.16±0.01	0.16±0.01
(mV)	C	0.16±0.02	0.17±0.01	0.17±0.01	0.16±0.01
	D	0.17±0.02	0.18±0.01	0.19±0.01	0.19±0.01
	A	0.090±0.002	0.095±0.004	0.093±0.006	0.092±0.007
P-R Intervals	B	0.090±0.003	0.093±0.006	0.093±0.002	0.095±0.001
(Sec)	C	0.093±0.003	0.089±0.003	0.090±0.005	0.092±0.002
	D	0.096±0.006	0.092±0.003	0.090±0.003	0.089±0.002
	A	0.051±0.003	0.053±0.002	0.052±0.003	0.051±0.003
QRS duration	B	0.055±0.003	0.057±0.001	0.056±0.004	0.057±0.001
(Sec)	C	0.056±0.001	0.055±0.002	0.054±0.002	0.055±0.002
	D	0.054±0.002	0.052±0.002	0.050±0.001	0.051±0.002
	A	1.58±0.22	1.62±0.12	1.57±0.18	1.57±0.08
QRS amplitude	B	1.45±0.14	1.63±0.11	1.60±0.14	1.58±0.12
(mV)	C	1.68±0.21	1.53±0.11	1.58±0.14	1.57±0.10
	D	1.67±0.19	1.58±0.11	1.57±0.13	1.52±0.16

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	A	0.19±0.01	0.21±0.02	0.21±0.01^{AB}	0.21±0.02^{AB}
QT Interval	B	0.20±0.02	0.23±0.02	0.25±0.01^{B}	0.25±0.01^{B}
(Sec)	C	0.23±0.02	0.21±0.01	0.22±0.01^{AB}	0.22±0.01^{AB}
	D	0.21±0.02	0.19±0.02	0.19±0.02^{A}	0.19±0.02^{A}
	A	0.18±0.03	0.19±0.03	0.18±0.03	0.18±0.02
T-Wave amplitude	B	0.15±0.01	0.16±0.01	0.15±0.02	0.16±0.02
(mV)	C	0.19±0.03	0.20±0.02	0.19±0.02	0.18±0.01
	D	0.17±0.02	0.19±0.02	0.18±0.02	0.18±0.01

Values in the same row with different superscripts (small font) differ significantly and values in the same column with different superscripts (upper font) differ significantly.

butorphanol or pentazocine. The slightly increased P-wave duration observed in the present study might be due to butorphanol lengthening the time for action potential and increasing the cardiac conduction time (Cardoso *et al.,* 2016).

P-wave amplitude values were recorded with slight variations at 0 min and the variations after the administration of the drugs in all groups were statistically not significant. No significant variation in P-wave amplitude value was recorded at different time intervals as compared to the respective pre-induction values in all the groups. The P-wave amplitude values showed decrease initially in Group A and B whereas increased in Group C and D. The P wave amplitude value in dogs is related to heart rate and lower the heart rate is smaller is the amplitude of P wave (Avdosko *et al*., 2010).

There was slight increase (non-significant) in P-R intervals in Group A and B initially and slight decrease (non-significant) in Group C and D. Increased P-R interval observed in the present study might be due to slower heart rate as P-R interval is closely related to heart rate (Ahmad *et al*., 2012) which might have caused by dexmedetomidine and propofol. The non-significant reduction in P-R interval in Group C and D might be due to an increase in heart rate caused by ketamine (Stepien, 1995). The reduction in P-R intervals reflected an increase in the conductivity of the cardiac stimuli between the SA node and AV node (Tilley, 1992).

A slight non-significant increase in QRS-duration was observed in Group A and B and similarly slightly non-significant decreased values were noticed in group C and D following administration of anaesthetics. These changes might be related with the change in heart rate as the intraventricular conduction time is reflected by QRS duration. The duration gets shorter as the heart rate increases and longer as the heart rate decreases (Mason *et al.,* 2016).

The QRS amplitude values showed non-significant increase at 20 min in Group A and B which remained near the base line values at the subsequent time intervals. The values of QRS amplitude decreased non-significantly in Group C and D at 20 min and values remained below the base line values at the subsequent periods of observation. The changes observed in the QRS amplitude might be due to the changes in the heart rate as QRS complex was indirectly related to the heart rate (Gunay and Balikci, 2001).

The Q-T interval varied significantly ($P<0.05$) at 40 and 60 min. The values of Q-T interval increased (non-significant) at 20 min in Group A and remained unchanged till the end of the observation period. In Group B, the Q-T interval showed a non-significant rise. In Group C and D, the Q-T interval decreased non-significantly and remained below the pre-induction level at the subsequent observation periods. Increased Q-T interval observed in the present study might be due to the changes in the heart rate during the anaesthetic period as heart rate and Q-T intervals were inversely proportional (Singh *et al*., 2013).

The values of T-wave duration did not show any significant variation either among or within the groups throughout the observation period. T-wave amplitude is indicative of myocardial hypoxia; however, the T-wave values in the present study were in normal range. The diagnostic value of T wave changes in dog is very limited in comparison to human as T wave morphology is highly variable in small animals (Martin, 2007).

CONCLUSION

The changes in the cardiac parameters remained within the physiological limits in all the anaesthetic combinations. The respiratory parameters were well preserved with isoflurane maintenance but the oxygen saturation values were near the critical level in ketamine-CRI.

Conflict of interest

The authors declare that there are no conflicts of interest.

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