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AUTOR DO ARTIGO: STUBBING JF, SWEENWY BP

TÍTULO DO ARTIGO: DIFFUSION HYPOXIA, DOES IT EXIST? A STUDY IN ASA I PATIENTS

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Recebi o pedido Nº Data _____/_____/_____ Assinatura_____

Voltar

TABLE

	Pre-reaming baseline	*Post-reaming baseline*
MAP mmHg	159 ± 10	151 ± 7
PAP mmHg	16.6 ± 7.3	13.3 ± 3.2
CO l/min	3.7 ± 0.9	3.4 ± 1
PVR cm·dyne^{-1} sec^{-5}	197 ± 52	243 ± 72
PaO_2 mmHg	104 ± 7	105 ± 6
QS/QT %	25.1 ± 2.5	20.3 ± 1.8
6PGF (a) ng·ml^{-1}	0.24 ± 0.06	0.29 ± 0.13
6PGF (v) ng·ml^{-1}	0.25 ± 0.09	0.33 ± 0.14
TxB_2 (a) ng·ml^{-1}	0.73 ± 0.24	0.87 ± 0.55
TxB_2 (v) ng·ml^{-1}	0.73 ± 0.25	0.86 ± 0.37

40

	Post-cemented arthroplasty		
	1 min	*5 min*	*15 min*
MAP mmHg	133 ± 25	143 ± 19	145 ± 14
PAP mmHg	31.5 ± 13*	30 ± 10.5*	25.9 ± 5.1*
CO l/min	—	3.0 ± 0.7	3.1 ± 3.1
PVR cm·dyne^{-1} sec^{-5}	—	738 ± 305*	628 ± 221*
PaO_2 mmHg	96 ± 4*	88 ± 5*	88 ± 7*
QS/QT %	—	26.3 ± 4.2*	26.3 ± 4.5*
6PGF (a) ng·ml^{-1}	1.14 ± 0.42*	0.72 ± 0.33*	0.36 ± 0.11
6PGF (v) ng·ml^{-1}	0.80 ± 0.19*	0.64 ± 0.21*	0.46 ± 0.19
TxB_2 (a) ng·ml^{-1}	1.22 ± 0.65*	1.04 ± 0.41	0.98 ± 0.40
TxB_2 (v) ng·ml^{-1}	1.16 ± 0.62*	0.89 ± 0.32	1.01 ± 0.40

Data presented as mean ± 1 SD.
*Denotes a significant change from baseline ($P < 0.05$) using two-way analysis of variance and Dunnett's multiple range test.

Discussion

The increases in PAP, PVR and $\dot{Q}_S/\dot{Q}_T$, immediately after CA were associated with an increase in 6PGF and TxB_2 levels. However, the reaming process is not responsible for these changes. Although pulmonary hypertension persisted for the 60-minute period after CA the TxB_2 response was transient. Our findings suggest that fat microembolism after CA is associated with the production of arachidonic acid metabolites. Whether this prostanoid response is the cause of the pulmonary haemodynamic changes after fat microembolism or only a marker for endothelial and cellular injury is not known. However, the significantly higher 6PGF level in the arterial blood at one minute compared with the mixed venous sample verifies that the lung is the source of prostaglandin production.

Diffusion hypoxia, does it exist? A study in ASA I patients

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Hypoxia is a frequent and major complication of general anaesthesia. The contribution of post nitrous oxide hypoxia (diffusion hypoxia) to the overall hypoxia has yet to be determined. This study was set up to try to determine that contribution.

Methods

Twelve ASA physical status I patients aged 16–65 years agreed to take part in the study. All were undergoing minor procedures involving general anaesthesia. All patients were seen and informed consent obtained, premedication of papaveretum and hyoscine was prescribed, dosage adjusted for weight. In the anaesthetic room, the patient was connected to a Kontron pulse oximeter, pulse and BP recorded, and an intravenous cannula inserted into one hand. Anaesthesia was induced with thiopentone, then maintained with oxygen 33 per cent in air and halothane. A vaporiser setting of three per cent, approximately four times the minimal alveolar concentration (MAC) in air was used. Spontaneous respiration via a mask and a Mapleson A circuit was maintained for ten minutes. Regular pulse, BP, end–tidal carbon dioxide ($ETCO_2$), and oxygen saturation were recorded. The patient was then disconnected from the anaesthetic circuit and allowed to breathe air for up to ten minutes or until the first sign of lightening of anaesthesia occurred. Anaesthesia was then re-established with oxygen 33 per cent in nitrous oxide (N_2O) 66 per cent, and halothane with a vaporiser setting of two per cent, approximately four MAC in N_2O. The patient was then taken into the operating theatre and the procedure performed on this anaesthetic mixture. At the end of the procedure, the circuit was disconnected and the patient allowed to breathe air. Throughout the entire procedure, regular recordings of pulse, BP, $ETCO_2$ and oxygen saturation levels were recorded. The trial was abandoned and oxygen administered if saturation dropped below 85 per cent oxygen at any stage.

Results

Statistical analysis of data employing Students t test was performed. Oxygen saturation changes at two, five and ten minutes and maximum saturation changes were analysed, and revealed no significant difference between the two groups. (Significance assessed as a "β" value of 0.05). There was no difference to the mean time of maximum saturation change, in both groups this was four minutes. $ETCO_2$ at the completion of ten minutes air/oxygen/halothane and at the end of the period of

TABLE Oxygen saturation change with time % change

Patient no.	*At 2 min*		*At 5 min*		*At 10 min*		*Maximum change*	
	Air	*N_2O†*	*Air*	*N_2O*	*Air*	*N_2O*	*Air*	*N_2O*
1	5	7	2	7	–	–	5	7
2	2	5	2	3	–	–	2	5
3	2	2	3	2	–	–	3	2
4	2	2	3	6	–	–	3	6
5	3	4	2	4	5	3	5	4
6	2	4	4	2	2	3	4	6
7	2	4	3	3	4	3	5	4
8	3	2	2	2	2	4	3	4
9	3	4	5	2	5	4	6	5
10	3	3	2	5	3	4	4	5
11	7	5	2	2	2	1	7	5
12	2	4	2	3	2	1	8	6

*Air = air/O_2/halothane technique
†N_2O = N_2O/O_2/halothane technique

N_2O/oxygen/halothane was performed using Student's t test and no significant difference was found between the two groups. (Table).

TABLE

			PGE1		
	Baseline	*PGF2a*	*Low*	*Inter*	*High*
CI	4.5±1.2	3.3±0.5*	3.1±0.3	3.2±0.5	3.5±0.5
SVR	24±7	32±6*	32±8	31±10	27±8a
PVR	2.3±0.5	5.8±1.2*	5.6±1.5	5±1.2	4.4±1.3a
CCP	9±3	15±3*	14±3	13±3	12±3a
Slope	1.5±1.2	2.8±0.5*	2.8±0.6	2.5±0.6	2.5±0.6

Values are mean ± SD. $P < 0.05$ by paired t test * vs baseline; a vs PGF2a.

Discussion

The Fink principle to describe diffusion hypoxia was first described in 1955, but with the introduction of the pulse oximeter the assessment of second to second changes in oxygen saturation has become possible, using simple, accurate and non-invasive techniques. Fink originated the theory after an *in vitro* experiment, and followed that up with a study on eight patients who were allowed to breathe room air immediately after completion of anaesthesia including a mixture of N_2O and oxygen. His results showed reductions in oxygen saturation of varying degrees and duration in the presence of supposedly adequate spontaneous ventilation, although the extent to which respiratory depression was responsible for any hypoxia was not accurately determined. By measuring $ETCO_2$ and obtaining no significant difference at the end of the anaesthetic periods, respiratory depression should have been excluded as a cause of hypoxia in this study.

Conclusion

Diffusion hypoxia does not appear to be a significant problem following minor surgery in healthy adults who received anaesthesia involving spontaneous respiration and little cardiovascular and respiratory disturbance. Routine administration of oxygen in the absence of other problems may not be necessary in these patients. It does not follow that the same is true of other groups of patients having more major procedures.

Prostaglandin E1 efficacy in canine model of pulmonary hypertension

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An acute increase in pulmonary vascular resistance (PVR) following repair of congenital heart lesions may seriously compromise a successful surgical outcome. Active vasoconstriction is partly responsible for these episodes of pulmonary hypertension and results from the highly reactive nature of these patients' pulmonary vasculature. In this critical situation inotropic agents may fail to increase cardiac output and vasodilators such as tolazoline and phentolamine have been used.[1] However, the fear that they may induce systemic hypotension limits their use. Prostaglandin E1 (PGE1) has been tried in various types of pulmonary hypertension but there exist great discrepancies in doses used and effects on PVR. We therefore evaluated incremental dose-response of PGE1 on pulmonary vascular dynamics in a model of vasoconstrictive pulmonary hypertension.

Methods

In eight dogs the pulmonary artery pressure (PAP)/flow relationship was assessed. Using an adjustable arteriovenous fistula, PAP/CI lines were generated by altering CI. The slope of these lines represents vascular conductance in the pulmonary vessels set up as parallel units, leading to capillaries submitted to a critical closing pressure (CCP), the mean of which is the intercept of the PAP/CI line at zero flow. The intercept thus represents the effective vascular outflow pressure which is usually higher than the assumed value (left atrial pressure). An aortic electromagnetic flow-probe was used to derive CI values.

Results

By infusing prostaglandin F2 alpha (PGF2a) into the main pulmonary artery, we caused vasoconstriction (see table): PVR increased almost threefold due to a 90 per cent increase in PAP/CI line slope and 60 per cent increase in CCP. PAP increased from 16 ± 5 to 24 ± 4 mmHg. CI decreased by 25 per cent. Mean arterial pressure (MAP) was unchanged and systemic vascular resistance (SVR) increased by 33 per cent. After stabilization three dose ranges of PGEl were tested by 15-minute infusion in a peripheral vein: low (0.04 to 0.08 $\mu g \cdot kg^{-1} \cdot min^{-1}$, intermediate (0.12 to 0.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and high (0.24 to 0.32 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Results are presented in the Table. PVR was substantially decreased only by high-dose PGE1 (−24 per cent). This was mainly due to a decrease in CCP (−23 per cent). PAP decreased from 24 ± 4 to 20 ± 5 mmHg. Mean CI remained constant throughout PGEl administration. At the highest dose CI was: increased in three dogs, unchanged in three and decreased in two. At high-dose PGE1, MAP decreased from 109 ± 21 to 96 ± 18 mmHg. SVR was decreased by 14 per cent. Mean left and right atrial pressures remained unchanged. Arterial gases measured in five dogs were unchanged by PGF2a hypertension but PO_2 decreased from 162 ± 74 to 94 ± 45 mmHg and PCO_2 increased from 38 ± 4 to 51 ± 8 mmHg following PGE1 administration.

Prostaglandin E1 decreased PVR substantially only at the highest dose studied by decreasing pulmonary vascular outflow pressure. Although encouraging because it decreased PVR relatively more than SVR (24 vs 14 per cent) it failed to increase CI in five out of eight dogs and had unfavourable effects on arterial blood gases. Since hypoxaemia is a great stimulus of pulmonary vasoconstriction, this effect should be further studied in models of vasoconstrictive pulmonary hypertension before PGE1 use is advocated following repair of congenital heart disease.

Reference

1 *Wheller G, George BL, Mulder DJ, Jarmakani JM.* Diagnosis and management of postoperative pulmonary hypertensive crisis. Circulation 1979; 60: 1640–4.