Clinical Reports

Arterial oxygen saturation following premedication in children with cyanotic congenital heart disease

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To determine the effects of premedication on arterial oxygen saturation (SaO_2) and heart rate (HR), 11 children (ages three to seven years) scheduled for elective repair of cyanotic congenital heart defects were studied. Patients were premedicated with oral or rectal pentobarbitone 2 mg·kg^{-1} 90 minutes prior to induction of anaesthesia followed by intramuscular morphine 0.2 mg·kg^{-1} and atropine 0.02 mg·kg^{-1} 60 minutes prior to induction. The SaO_2 and HR of each child were monitored continuously using a Nellcor® pulse oximeter during two 90 minute periods: a control period commencing 25.5 hours preoperatively (day 1) and a post premedication period commencing 1.5 hours preoperatively (day 2). Data were compared at time 0 (corresponding to the time of administration of pentobarbitone on day 2), 30 (corresponding to the administration of intramuscular morphine and atropine on day 2), 60 and 90 minutes (the latter corresponding to the time of induction on day 2) after the administration of pentobarbitone. There were no significant differences in SaO_2 or HR between day 1 and day 2 at time 0, 60, and 90 minutes. The SaO_2 (mean ± SD) decreased significantly immediately following intramuscular premedication at time 30 minutes on day 2 (72.7 ± 5.9 per cent) compared to the corresponding time on day 1 (83.9 ± 2.9 per cent) ($p < 0.05$). The duration of this desaturation was 2.5 ± 1.9 minutes. Heart rate (mean ± SD) increased from 109.2 ± 21.3 beats·min^{-1} at time 30 minutes on day 1 to 142 ± 20.4 beats·min^{-1} on day 2 ($p < 0.05$). We conclude that administration of intramuscular premedication preceded by oral or rectal pentobarbitone causes transient arterial desaturation and tachycardia in children with cyanotic congenital heart disease.

Key words
ANAESTHESIA: premedication; CONGENITAL HEART DISEASE: cyanotic; MONITORING: pulse oximetry; OXYGEN: saturation.

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Premedication decreases the partial pressure of oxygen in arterial blood (PaO_2) and the arterial oxygen saturation (SaO_2) in adults.[1] Similar decreases in SaO_2 have been postulated in children with cyanotic congenital heart disease (CCHD) although this remains unproven.[2] Despite the lack of evidence, the administration of supplemental oxygen for a brief period after premedication has been recommended for this group of children.[3] Heavy premedication is recommended for children with CCHD to prevent arterial desaturation during induction of anaesthesia.[3,4] SaO_2 has been shown to increase during preoxygenation and induction of anaesthesia in children with CCHD.[5] One might conclude from the latter study that premedication either decreases SaO_2 in children with CCHD and that preoxygenation and induction of anaesthesia restores the SaO_2 to pre-premedication values or greater, or that despite premedication, struggling and crying occurs immediately prior to induction and produces a decrease in SaO_2 which is reversed by induction. Because the SaO_2 in children with CCHD lies on the steep portion of the oxyhemoglobin dissociation curve, small changes in the PaO_2 are proportional to changes in the SaO_2. This supports the use of pulse oximetry as an accurate non-invasive estimate of the SaO_2 in these patients.[6,7] We used pulse oximetry to monitor the effects of premedication on the arterial oxygen saturation of children with cyanotic congenital heart disease during the preoperative period including the time of induction of anaesthesia.

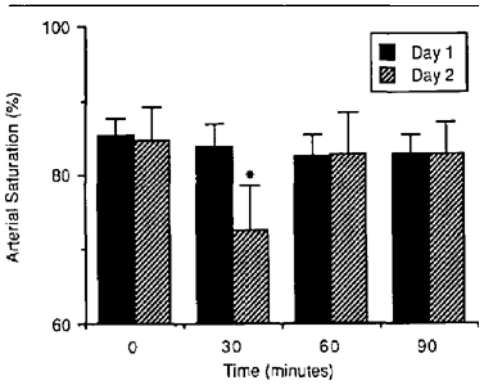

FIGURE 1 SaO_2 (mean ± SD) in children with CCHD at 0, 30 minutes, 60 minutes, and 90 minutes on day 1 and day 2. Arterial oxygen saturation decreased significantly (* $p < 0.05$) at 30 minutes on day 2 (the time of intramuscular premedication).

Methods

Following approval by the Human Research Committee of The Hospital for Sick Children and informed parental consent, 11 children with CCHD were studied. All children were admitted for elective surgical correction of cyanotic congenital heart defects.

To account for the diurnal variation in SaO_2, each child served as his/her own control and was studied during two 90 minute periods: on day 1, a control period commencing 25.5 hours prior to induction, and on day 2, a study period commencing 1.5-hours prior to induction. During each 90 minute period, SaO_2 and heart rate (HR) were monitored continuously and recorded using a Nellcor® pulse oximeter (model N 100, Nellcor Inc., Hayward, CA). The oximeter sensor was firmly applied to the great toe of one foot. During these periods, the behaviour of each child was recorded as: asleep; calm and cooperative; anxious or upset; or non-cooperative. On day 2, oral or rectal pentobarbitone 2 $mg \cdot kg^{-1}$ was given 90 minutes preoperatively (time 0). Thirty minutes later morphine 0.2 $mg \cdot kg^{-1}$ and atropine 0.02 $mg \cdot kg^{-1}$ were given intramuscularly (time – 30 minutes). Each child breathed room air during the study. On day 2, SaO_2 and HR were recorded continuously during the 90 minute study period which included transport to the operating room and induction of anaesthesia.

D'Agostino's test was used to determine whether the SaO_2 data were normally distributed.[8] SaO_2 and HR for all patients at times 0, 30, 60 and 90 minutes on days 1 and 2 were calculated and compared using analysis of variance for repeated measures, the Student-Newman-Keuls multiple range test and Student's paired t test. Behavioural data were compared using the Wilcoxon paired rank test. Statistical significance was accepted as $p < 0.05$. Data are presented as means ± SD.

Results

The demographic data and type of surgery is presented in Table I. All children were cyanotic with a haematocrit of 53.1 ± 4.2 per cent. Nine of the 11 children in this study were diagnosed as having tetralogy of Fallot.

Premedication was administered according to the protocol with the exception of one child who was given diazepam 0.22 $mg \cdot kg^{-1}$ (PO) instead of pentobarbitone.

The SaO_2 and HR at the 30 minute measurement on day 2 (the time of administration of intramuscular premedication) differed significantly from the 30 minute measurement on day 1 and from times 0, 60 and 90 minutes on both days (Figures 1 and 2). Arterial oxygen saturation decreased from 83.9 ± 2.9 per cent on day 1 to 72.7 ± 5.9 per cent on day 2 and the mean duration of the arterial oxygen desaturation was 2.5 (± 1.9) minutes. Heart rate increased from 109.2 ± 21.3 $beats \cdot min^{-1}$ on day 1 to 142.9 ± 20.4

TABLE I Demographic data

Patient number	*Age (months)*	*Weight (kg)*	*Haematocrit (%)*	*Previous surgery*	*Procedure*
1	62	16.6	50	Nil	Repair TOF
2	44	14.0	60	Nil	Repair TOF
3	55	15.2	48	Right B-T	Repair TOF
4	73	25.7	57	B-H, PA band	Mustard repair
5	69	20.1	59	Left B-T	Fontan repair
6	60	16.3	54	Right B-T	Repair TOF
7	89	19.5	50	Pott's anast	Repair TOF
8	42	14.5	55	Right B-T	Left B-T for TOF
9	27	12.6	51	Right B-T	Repair TOF
10	38	15.9	53	Nil	Repair TOF
11	74	16.2	48	Nil	Repair TOF

TOF = tetralogy of Fallot; B-T = Blalock-Taussig shunt; B-H = Blalock-Hanlon shunt; anast = anastamosis; PA = pulmonary artery.

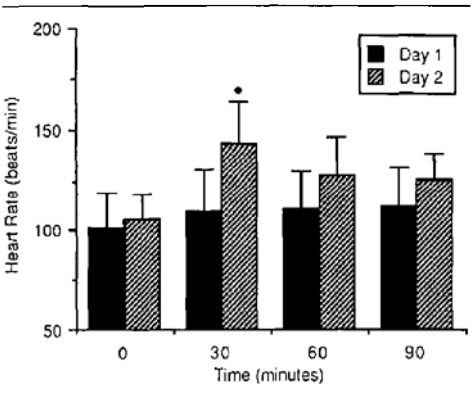

FIGURE 2 Heart rate (mean ± SD) at 0, 30 minutes, 60 minutes and 90 minutes. Heart rate is increased significantly (* $p < 0.05$) only at 30 minutes on day 2 (the time of intramuscular premedication).

beats·min^{-1} on day 2 ($p < 0.05$). These changes in SaO_2 and HR at the 30 minute measurement on day 2 occurred in all patients.

Behavioural data for children at times 0, 30, 60 and 90 minutes on days 1 and 2 are shown in Table II. The behavioural scores differed at the 30 minute period on day 2 (corresponding to the intramuscular injection of morphine and atropine) compared to day 1. At this time the children were more anxious and upset than at any other time during the study.

Discussion

We previously reported that the administration of 100 per cent oxygen before induction of anaesthesia in children with CCHD caused a significant increase in SaO_2.[5] We postulated that a non-cardiac, pulmonary mechanism secondary to the administration of premedication might have contributed to the preoperative hypoxaemia. Several plausible explanations might be invoked. Atropine has been shown to increase physiological dead-space[9] and cause preoperative hypoxaemia in adults.[10] Furthermore, morphine has been shown to reduce the ventilatory response to carbon dioxide and reduce functional residual capacity, thereby increasing ventilation–perfusion mismatch.[11] These effects have not yet been substantiated in children with CCHD. Patients with CCHD demonstrate a blunted response to hypoxaemia and therefore may not increase minute ventilation sufficiently to fully compensate for a worsening hypoxaemia induced by premedication.[12]

TABLE II Behaviour score

Behaviour score	*Day 1 time (mins)*				*Day 2 time (mins)*			
	0	*30*	*60*	*90*	*0*	*30**	*60*	*90*
1	6	5	1	1	3	1	2	2
2	4	5	10	10	6	2	9	7
3	1	1	0	0	1	1	0	2
4	0	0	0	0	1	7	0	0

Data are the number of children with each score at each time. Score: 1. asleep, 2. calm/cooperative, 3. anxious/upset, 4. uncooperative.
*Different from day 1 ($p < 0.05$).

Decreases in PaO_2 following premedication have been documented in acyanotic adults with SaO_2 values as low as 90 per cent.[1] The results of the present study, however, do not support the hypothesis that premedication with pentobarbitone, morphine and atropine in children with CCHD causes a progressive decrease in SaO_2 preoperatively. SaO_2 decreased transiently during and immediately after intramuscular premedication (Figure 1) but then returned to pre-premedication values for the remainder of the preoperative period. Therefore, heavy premedication does not cause a progressive worsening arterial desaturation in children with CCHD. This difference from the adult studies may be due to a greater sensitivity of the study method in the adult study in which arterial blood gas samples were analyzed or alternatively, arterial desaturation due to breath holding or straining by the patients in anticipation of the arterial puncture as has been demonstrated in children.[4]

Analysis of the behavioural data indicated that there was no difference in the behaviour of the children on day 2 compared to day 1 (Table II) except at the 30-minute time period. This time corresponded to the administration of intramuscular morphine and atropine. At this time, the children were more upset, anxious and uncooperative compared to the same time on day 1. There was however no difference in the behaviour scores at the 90-minute period (time of induction) indicating the premedication was successful in producing a calm patient at induction with no decrease in SaO_2.

Heavy premedication in children with CCHD is recommended to minimize crying and struggling during induction of anaesthesia.[13,14] We have shown that the benefit of premedication occurs at the expense of a significant, albeit transient, acute arterial desaturation at the time of administration of the intramuscular premedication associated with transient increased anxiety state. In our study this desaturation was not clinically significant.

The optimal premedication for children is one which can be administered with minimal distress, respiratory or cardiovascular depression but which confers adequate sedation. Oral or rectal premedication with avoidance of intramuscular injections may be ideal.[15] We were unable to identify any arterial desaturation after the administration of oral or rectal pentobarbitone.

In conclusion, we found that SaO_2 does not progressively decrease after premedication in children with CCHD. However, SaO_2 does decrease transiently during periods of struggling and crying after intramuscular premedication. Arterial saturation and behaviour scores did not change significantly after rectal or oral pentobarbitone or at induction.

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Résumé

Afin de déterminer les effets de la prémédication sur la saturation artérielle en oxygène (SaO_2) et la fréquence cardiaque (HR), 11 enfants (âgés de trois à sept ans) cédulés pour une réparation élective de maladie cardiaque congénitale cyanotique ont été étudiés. Les patients ont été prémédiqués avec du pentobarbitone 2 $mg \cdot kg^{-1}$ par voie orale ou rectale 90 minutes avant l'induction de l'anesthésie suivi par l'injection intramusculaire de morphine 0.2 $mg \cdot kg^{-1}$ et d'atropine 0.02 $mg \cdot kg^{-1}$ 60 minutes avant l'induction. La SaO_2 et HR pour chaque enfant ont été surveillées continuellement par le pulse oxymeter de Nellcor® pendant deux périodes chacune de 90 minutes: une période de contrôle débutant à 25.5 heures préop (premier jour) et une période post-prémédication débutant à 1.5 heures préop (deuxième jour). Les données ont comparées au temps 0 (correspondant au temps de l'administration du pentobarbitone le deuxième jour), 30 (correspondant à l'administration intramusculaire de morphine et d'atropine au deuxième jour), 60 et 90 minutes (ce dernier correspondant au temps de l'induction au deuxième jour) après l'administration de pentobarbitone. Il n'y avait aucune différence statistiquement significative concernant la SaO_2 et le HR entre le premier jour et le deuxième jour au temps 0, 60 et 90 minutes. La SaO_2 (moyenne ± SD) a diminué significativement immédiatement après la prémédication intramusculaire au temps 30 minutes le deuxième jour (72.7 ± 5.9 pour cent) comparativement au temps correspondant de la première journée (83.9 ± 2.9 pour cent) ($p < 0.05$). La durée de cette désaturation était de 2.5 ± 1.9 minutes. La fréquence cardiaque (moyenne ± SD) a augmenté de 109.2 ± 21.3 $batt \cdot min^{-1}$ au temps 30 minutes du premier jour à 142 ± 20.4 $batt \cdot min^{-1}$ au deuxième jour ($p < 0.05$). On conclut que l'administration d'une prémédication intramusculaire précédée par du pentobarbitone par voie rectale ou orale produit une désaturation artérielle transitoire et une tachycardie chez les enfants atteints de maladie cardiaque congénitale cyanogéne.