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Oxygen Administration Prevents Hypoxemia during Post-anesthetic Transport in Children

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Arterial oxygen desaturation frequently occurs in healthy patients during transport from the operating room (OR) to the post-anesthetic recovery room.[1,2] Because infants and children have a higher basal metabolic rate and a higher ratio of alveolar ventilation to functional residual capacity (FRC) than adults, pediatric patients are probably at a greater risk of post-anesthetic arterial desaturation. This prospective study examines changes in arterial oxygen saturation in healthy children who are breathing room air during and immediately after transport following routine surgery and documents the effectiveness of added inspired oxygen in the prevention of arterial oxygen desaturation.

MATERIALS AND METHODS

The protocol was approved by our hospital's Institutional Review Board, and parents were informed about the study. Two hundred ASA physical status I or II patients between 2 months and 9 yr of age scheduled for elective surgery were studied. Patients undergoing bronchoscopy or thoracotomy, as well as those with a history of cardiovascular or pulmonary disease, symptoms of an upper respiratory infection within 2 weeks of surgery, or an axillary temperature below 35.5° C prior to transport, were excluded from the study. Patients did not receive pre-anesthetic sedation or atropine. The anesthesiologist was free to use any anesthetic technique that seemed appropriate for the surgical procedure. At the end of surgery, the anesthetic agents were discontinued and 100% oxygen was administered for at least 60 s in the operating room. A Nellcor® pulse oximeter (Model N-100) set in Mode 3 was applied for continuous recording of SaO_2 during transport. Immediately before leaving the operating room, the patients were assigned to one of two study groups based on a number sequence generated by a randomization table. Patients in group A (n = 100) were transported to the recovery room while breathing room air. Those in group B (n = 100) received oxygen at 6 l/m by face mask during transport to the recovery room. The duration of 100% oxygen administered prior to transport was recorded. SaO_2 and Steward's recovery score[4] in the operating room, duration of transport time before onset of arterial desaturation (arbitrarily defined as oxygen saturation of less than 90% for more than 30 s), recovery room scores, and SaO_2 upon admission to the recovery room were compared between the two groups. The difference in the percentage of patients whose saturations were below 90% was tested using Chi-square analysis. Student's *t* test was used to compare mean values of age, anesthesia time, and transport time. The Wilcoxon test was used for comparison of Steward's recovery scores and lowest oxygen saturation recorded during transport and in the recovery room. The results are expressed as mean ± standard deviation. *P* value less than 0.05 was considered statistically significant in all cases.

RESULTS

The two groups were comparable in age, types of surgical procedures, and anesthesia time (table 1). There was no difference between patients in group A and B in the degree of wakefulness, as measured by Steward's recovery scores at the beginning of transport in the operating room and at the end of transport in the recovery room. The median transport time was 109 s (range 90–227 s) in group A (air) and 112 s (range 89–247 s) in group B (O_2). Twenty-one patients in group A showed arterial oxygen desaturation during transport, compared with only three patients (all infants) in group B ($P < .001$). The range of SaO_2 in patients who had arterial oxygen desaturation is shown in table 2. The mean of the lowest recorded oxygen saturations during transport in group A was 91.7 ± 10.1, *versus* 94.9 ± 5.8 in group B ($P < .01$). When the patients arrived in the recovery room, the mean levels were 93.8 ± 9.8 and 97.8 ± 4.0 ($P < .001$), respectively.

DISCUSSION

The occurrence of hypoxemia in adult and pediatric patients recovering from general anesthesia has been well documented.[1,3,5] Infants and young children normally have higher oxygen consumption, larger closing capacity,

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TABLE 1. Perioperative Variables (Mean ± SD)

Group	Age (Months)	Anesth Time (Min)	Transp. Time (s)	Recovery Score		SaO_2%	
				OR*	PARR*	Transp.	PARR
A (room air) (n = 100)	41.0 ± 27.4	53.3 ± 29.2	117.3 ± 27.0	4.0 (2–6)	5.0 (3–6)	91.7 ± 10.1	93.8 ± 9.8
B (oxygen) (n = 100)	34.4 ± 24.5	51.5 ± 31.4	119.7 ± 28.4	5.0 (2.5–6)	6.0 (4–6)	94.9† ± 5.8	97.8‡ ± 4.0

OR = operating room; PARR = post-anesthetic recovery room; SaO_2 = arterial oxygen saturation.
* Median and interquartile range.
† $P < .01$.
‡ $P < .001$.

and proportionately higher alveolar ventilation and basal metabolic rate than adults. Because inspired gases equilibrate more rapidly with capillary blood in the lung, arterial oxygen desaturation would be expected to occur more readily in children than adults with the change from supplemental oxygen to room air. In addition, the incompletely developed elastic support structure of the child's chest wall and lung allows earlier closure of small airways and places pediatric patients at greater risk for atelectasis and ventilation-perfusion mismatch, further exacerbating arterial desaturation.

Arterial oxygen desaturation reportedly occurs in 23% of pediatric patients during postoperative transport to the recovery room.[2] Since the degree of wakefulness does not correlate with oxygen desaturation,[3] recovery scores cannot be used to identify patients who are at risk for arterial oxygen desaturation. Routine use of supplemental oxygen has been suggested to prevent hypoxemia during transport; however, its efficacy has heretofore been unproven. We have shown that supplemental oxygen reduces the incidence of hypoxemia during transport and results in higher SaO_2 upon admission to the recovery room.

Patients with an axillary temperature below 35.5° C prior to transport were excluded from the study. Shivering due to hypothermia causes continuous movement of the finger probe and the pulse oximeter may misinterpret such movements as a pulse resulting in erroneous SaO_2. Three of our infants developed hypoxemia in spite of supplemental oxygen administration. The reason for this is difficult to explain. They were not hypoventilating by clinical evaluation. Their body temperatures were above 36° C before leaving the operating room. None of these infants was noted to have any risk factors predisposing to hypoxemia, such as airway obstruction, shivering, or obesity. They were all awake and crying. It is possible that the intermittent breath-holding that is often associated with crying was responsible for the drop in oxygen saturation. SaO_2 had returned to normal by the time each of these patients arrived in the recovery room.

TABLE 2. Lowest Arterial Oxygen Saturations Recorded during Postoperative Transport

SaO_2 Level (%)	71–80	80–85	86–90	>90
Group A (room air) n = 100	3	6	12	79
Group B (oxygen) n = 100	2	0	1	97

SaO_2 = arterial oxygen saturation.

The clinical significance of a SaO_2 level of 90% or less that does not manifest as detectable cyanosis is unknown. Low oxygen saturation decreases the total amount of oxygen carried in the blood. Low SaO_2 level in our patients may have been partially compensated by increased cardiac output to maintain minimal oxygen delivery (oxygen delivery = O_2 content × cardiac output).[6] In any individual patient, however, it would be difficult to predict the degree of such compensation and its effectiveness, especially at low oxygen saturation. We arbitrarily chose a SaO_2 level of 90% or less for more than 30 s as being significant. Normal transcutaneous oxygen saturation in the sleeping infant ranges from 91–95%.[7] An oxygen saturation of 90% (which corresponds to a Pa_{O_2} of about 60 mmHg) can forwarn about impending severe hypoxemia. The role of such forewarning in reducing morbidity is not yet established.

Our study was limited to healthy infants and children undergoing relatively simple surgical procedures. In sicker patients or those undergoing extensive surgery, the incidence of low arterial oxygen saturation during transport is likely to be higher than that detected in this study.

In summary, infants and children are at risk of developing arterial oxygen desaturation if they are allowed to breathe room air during post-anesthesia transport to the recovery room. Administration of oxygen significantly reduces the incidence of desaturation. We recommend that infants and children recovering from general anesthesia be transported to the recovery room while breathing supplemental oxygen. Because some infants may still become hypoxemic while breathing supplemental oxygen, monitoring of saturation and the availability of equipment to provide positive pressure ventilation may occasionally be necessary to assure normoxia during transport.

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Perioperative Management for Transplant of Autologous Adrenal Medulla to the Brain for Parkinsonism

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Parkinson's disease was originally described as "paralysis agitans" or the shaking palsy by James Parkinson in 1817. Parkinsonism (lower case "p"), the generic term for the disease, is characterized by tremor, bradykinesia, rigidity, and postural changes. The pathophysiology is dopamine deficiency in the basal ganglia associated with decreased numbers of melanocytes in the substantia nigra.[1]

The only treatment before 1967 was placing lesions in the basal ganglia, although it was not very effective.[2] Since 1967, dihydroxyphenylalanine (DOPA) and other dopaminergic drugs[3,4] have become the mainstay of therapy, owing to their ability to raise the central nervous system (CNS) concentration of dopamine.

Recently, surgical therapy has again been suggested. Transplantation of autologous adrenal medulla to the caudate nucleus of the brain has resulted in reduced symptoms and decreased drug requirements in two reports.[5,6] Several of these operations have been performed at our institution. This report describes perioperative management of these patients.

METHODS AND MATERIALS

Twelve patients have undergone this procedure, which was approved by the local ethics committee. Informed consent was obtained from each patient. Ages were 39 to 49 yr and parkinson disability was Stage II to Stage IV.[7] All patients had been with l-DOPA, carbidopa, bromocriptine, amantadine, trihexyphenidyl, in various combinations and were experiencing worsening of symptoms.

Patients had nothing by mouth for at least 6 h before the operation. A Brown-Roberts-Wells stereotactic base and frame was placed on the head after sedation with midazolam 1–2 mg iv and infiltrating the scalp with a 50/50 mixture of 1% lidocaine with 1:100,000 epinephrine and 0.5% bupivacaine (total volume 10–15 ml). Computerized tomography was performed and target point coordinates determined.[8,9]

After transport to the operating room, arterial blood pressure cuff, electrocardiograph, precordial stethoscope, and pulse oximeter were placed. Further monitoring after induction of anesthesia included nasopharyngeal temperature, esophageal stethoscope, radial arterial catheter, urinary catheter, and mass spectrometry.

Additional midazolam was given in 1-mg increments as the upper airway was anesthetized with nebulized 4% lidocaine (4–5 ml) and viscous 2% lidocaine (15 ml) in the nose.[10] After spraying with 0.5% phenylephrine (1 ml total), a warmed tracheal tube was inserted through the

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