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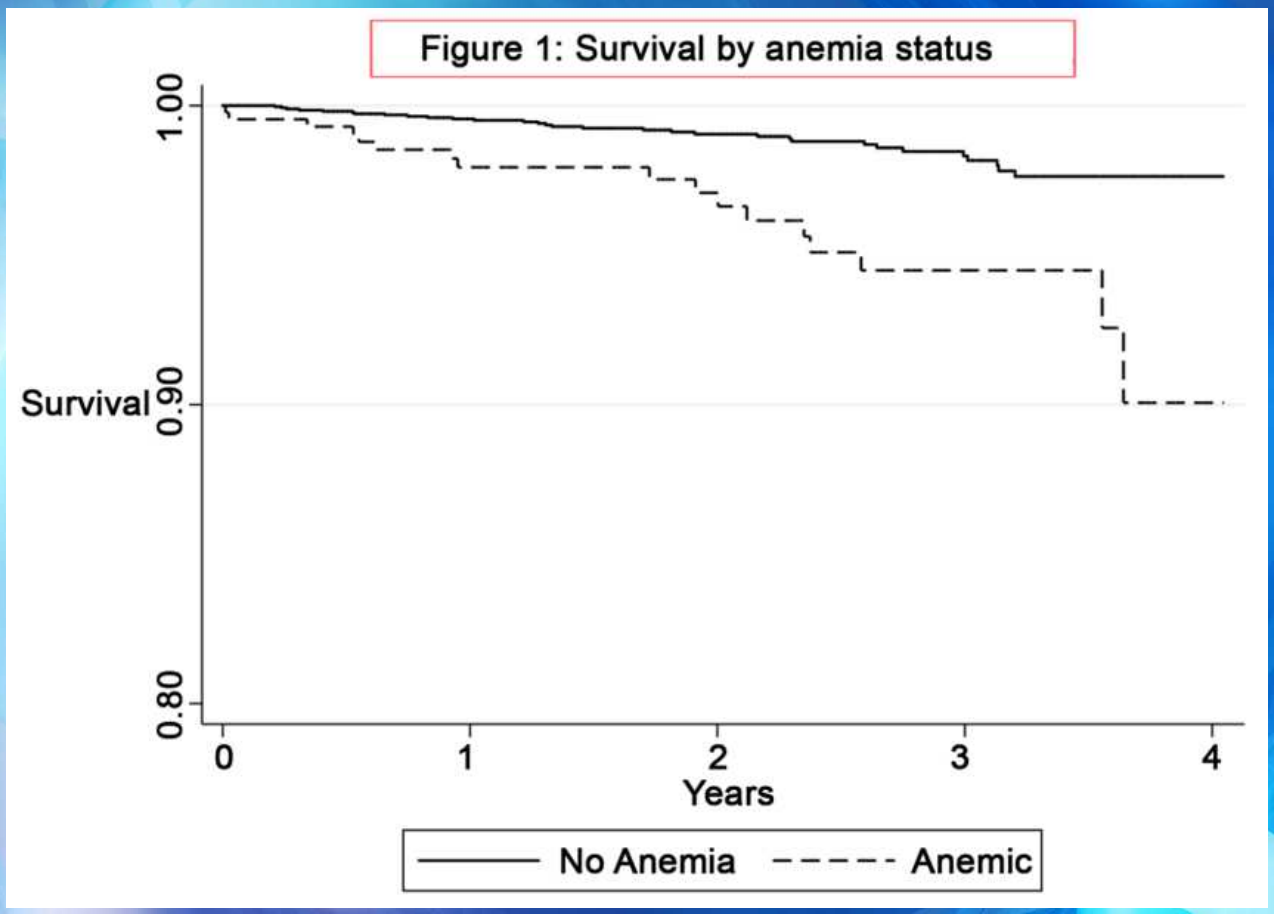

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The figure on the front cover is from the article published in Open Journal of Anesthesiology, 2016, Vol. 6, No. 6, pp. 91-96 by Michael D. Herrick, Brian D. Sites, Melissa M. Masaracchia and Wayne E. Moschetti.

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The Effect of Nitrous Oxide on the Intraocular Pressure in Patients Undergoing Abdominal Surgery under Sevoflurane and Remifentanil Anesthesia

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Abstract

Introduction: Although inhalational anesthesia and nitrousoxide (N_2O) are known to affect the intraocular pressure (IOP), little is known about the effect of nitrousoxide on the IOP during sevoflurane and remifentanil anesthesia. In the present study, we examined the effect of balanced anesthesia on the IOP. Materials and Methods: After obtaining informed consent, the patients undergoing abdominal surgery under general anesthesia were divided into two groups: N_2O group (n = 10) and control group (n = 12). General anesthesia was maintained with remifentanil (0.05 - 0.3 μg/kg/min), 33% O_2 and 1.2% sevoflurane to keep $ETCO_2$ of 35 - 40 mmHg following tracheal intubation. After baseline measurement of IOP (T0, 20 minutes after injection of anesthesia), the patients in the N_2O group received 67% nitrousoxide, and the patients in the control group received air, with O_2 and 1.2% sevoflurane. Then, IOP was measured at 1 hour (T1), 2 hours (T2), and 3 hours (T3) after anesthesia induction in the supineposition. Blood pressure and heart rate were recorded at the same time interval. Results: There was no significant difference in the IOP at any period between the two groups. In both groups, the IOP at the T3 was significantly higher than that at T0. Conclusion: These results suggest that N_2O does not affect the IOP in patients undergoing abdominal surgery under sevoflurane and remifentanil anesthesia.

Keywords

Intraocular Pressure, Nitrous Oxide, Balanced Anesthesia

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1. Introduction

Intraocular pressure (IOP) is known to change during perioperative period due to inhalational anesthetic agents like halothane, isoflurane and sevoflurane [1] [2], and opioids such as fentanyl, alfentanil and remifentanil [3]-[7]. Schäfer *et al*. have shown that IOP more reduces during anesthesia with propofol than with sevoflurane, both combined with remifentanil [8].

Although nitrous oxide (N_2O) may affect IOP [9]-[12], one report indicates that IOP with desflurane and N_2O does not differ compared with desflurane alone in dogs [11]. Moreover, N_2O has been shown to have no influence in healthy volunteer [13]. On the other hand, detrimental effect of N_2O is reported, indicated that the use of N_2O in patients, who undergo vitreoretinal procedures cause retinal or optic nerve ischemia, results in visual loss [14]-[16].

Sevoflurane combined with remifentanil anesthesia is not known to influence IOP. In addition, it is not well known about the effect of N_2O on IOP in patients receiving sevoflurane and remifentanil anesthesia in patients with abdominal surgery. Therefore, we examine the effect of N_2O on the IOP in patients undergoing abdominal surgery under sevoflurane and remifentanil anesthesia.

2. Materials and Methods

The study was approved by the Ethics Committee of Akita University Hospital and registered with the UMIN clinical trials registry (ID: UMIN000020241). After obtained informed consent, 22 ASA physical status I or II patients scheduled for elective abdominal surgery were studied. We excluded patients with allergies, unstable angina, congestive heart failure, glaucoma and other ophthalmic disease and past history of eye surgery. The patients were allocated to either of two groups; N_2O group (n = 10) and control group (n = 12). All patients were premeditated with ranitidine150 mg 90 min before general anesthesia. Anesthesia was induced with propofol 1 mg/kg, continuous infusion of remifentanil and rocronium 1mg/kg. The trachea was intubated, and lung ventilation was adjusted to maintain end-tidal CO_2 at 35 - 40 mmHg with 33% oxygen, 1.2% sevoflurane and remifentanil (0.05 - 0.3 μg/kg/min). The patients in the N_2O group received 33% oxygen and 67% N_2O, and the patients in the control group received air instead of oxygen and N_2O. IOP was measured at 20 min after induction of anesthesia (T0), 1 hour after T0 (T1), 2 hours after T0 (T2), and 3 hours after T0 (T3) at the supine position using PT100 portable non-contact tonometer (Reichert, INC, Depew, NY, USA). Blood pressure and heart rate were recorded at the same time interval. We measured the IOP three times in each epoch, and then calculated the mean value.

We defined hypotension as a SBP (systolic blood pressure) ≤ 80% of the preinduction baseline SBP, hypertension as a SBP > 140% of the preinduction baseline SBP, and bradycardia as HR < 40 bpm. Hypotension was treated with an intravenous bolus of phenylephrine 50 μg or ephedrine 5 mg and bradycardia was treated with an intravenous bolus of atropine 0.5 mg.

Data were expressed as mean ± SD. Student t-test was used to compare the data between two groups, and analysis of variance for repeated measures was performed to access differences within the groups. $P < 0.05$ was considered as statistically significant.

3. Results

The patients in the two groups were comparable with regards to demographic and hemodynamic data (**Table 1** and **Table 2**).

Although there were no significant differences between the two groups in IOP at any measuring points, IOP at T3 was significantly higher than that at T0 in both groups (**Figure 1**).

Table 1. Patients' demographic data.

	N_2O group	Control group	*P* value
Age (years)	59 ± 12	55 ± 15	0.64
Gender (male/femal)	4/6	3/9	0.65
Height (cm)	157 ± 13	159 ± 12	0.63
Weight (kg)	56 ± 14	58 ± 12	0.65
ASA (grade1/2)	4/6	3/9	0.65

Values are mean ± SD or numbers. No significant difference.

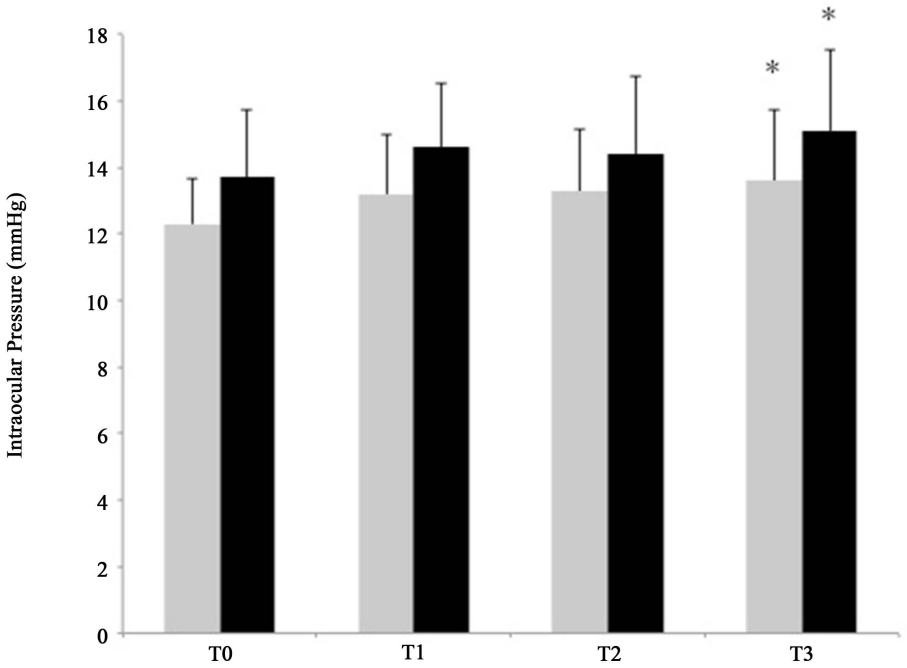

Figure 1. Changes of the intraocular pressure (IOP) during study period. IOP did not differ between the N_2O groups (gray bar) and the control group (black bar) at any measuring points. IOP values at T3 in both groups were higher than those at T0. T0 = 20 min after induction of anesthesia, T1 = 1 hour after T0, T2 = 2 hours after T0, T3 = 3 hours after T0. Values were mean ± SD. $^{*}P < 0.05$ versus T0.

Table 2. Changes of intraocular pressure.

	Group	T0	T1	T2	T3	*P* value
Mean Blood Pressure (mmHg)	N_2O group	69 ± 11	68 ± 9	68 ± 8	69 ± 10	0.84
	Control group	70 ± 14	73 ± 10	67 ± 7	72 ± 11	
Heart Rate (beats/min)	N_2O group	71 ± 8	69 ± 10	68 ± 13	69 ± 11	0.09
	Control group	64 ± 11	63 ± 12	62 ± 10	67 ± 9	
$ETCO_2$ (mmHg)	N_2O group	36 ± 0.8	36 ± 0.9	36 ± 0.8	37 ± 1.4	0.13
	Control group	35 ± 0.4	36 ± 0.9	36 ± 0.9	36 ± 1.2	

T0 = 20 minutes after induction, T1 = 1 hour after T0, T2 = 2 hour after T0, T3 = 3 hour after T0. Values = mean ± SD. There were no significant different between two groups.

There was no patient who developed hypertension, hypotension and bradycardia during the study period.

4. Discussion

We conducted a prospective, randomized study to evaluate the effects of N_2O on IOP during sevoflurane and remifentanil anesthesia. Our results demonstrated that N_2O did not affect IOP during 3 hours in patients under general anesthesia with sevoflurane and remifentanil. However, IOP increased at 3 hours after induction of anesthesia compared with starting point in both patients with and without N_2O.

Intraocular pressure (IOP) is known to changing at perioperative period due to anesthetic maneuvers [3] [17], anesthetic agents [4] [18]-[21], and patient's position [22]-[27] and hemodynamics [28]. Tracheal intubation [29], succinylcholine [5]-[7], inhalational anesthesia [1] [2] [8], and nitrous oxide (N_2O) [21] may influence IOP. Inhalational anesthetics and propofol have been shown to decrease IOP [8], whereas succinylcholine can increase IOP [5]-[7]. Previous studies demonstrated that general anesthesia with halothane, enflurane, propofol,

and fentanyl would decrease IOP after tracheal intubation [2]-[5]. These range of reduction varied with type of anesthesia. Tracheal intubation could lead to elevate IOP [17], however, this effect can be minimized by various method [5]-[7]. In this study, we did not measure IOP before anesthesia. Therefore, we could not compare the IOP values between before and after tracheal intubation. Moreover, none of the previous reports that showing the changes of the IOP during sevoflurane and remifentanil anesthesia in patients undergoing abdominal surgery was existed.

Although major determinants for IOP include the production rate of aqueous humor, vitreous volume, sclera rigidity, choroidal blood volume, and orbicularis oculi muscle tension [12], there have been few studies to assess the effect of nitrous oxide on IOP [10]-[13]. Lalwani *et al*. have shown that nitrous oxide inhalation did not significantly change IOP from baseline values in a population of healthy adults [13]. Our result of present study was consistent with their result.

IOP at T3 in both groups were significant greater than IOP at T0 in this study. Because hemodynamics and anesthesia was similar during the study period, the possibility of blood pressure and anesthesia can be excluded. However, it remains unknown what was the effect of IOP at T3. IOP at T3 in both group were within normal range and did not differ between the two groups. Therefore, it is clear that N_2O does not affect the elevation of IOP at T3.

Based on the present and previous similar study [11]-[13], the effect of N_2O would not affect the IOP during 3 hours sevoflurane and remifentanil anesthesia in patients undergoing abdominal surgery. Our study had the following possible limitations. We had recruited the small number of patients with ASA physical status I or II. It remains unknown if the results will be applicable to other populations such as patients with glaucoma, under head down position surgery or laparoscopy. The difference of the IOP before and after sevoflurane and remifentanil anesthesia was not clear from this study and warranted the additional studies. Future studies will be needed to clarify the effects of N_2O long exposure on IOP in other patient populations and surgery.

5. Conclusion

In conclusion, N_2O did not affect IOP during abdominal surgery under sevoflurane and remifentanil anesthesia. With or without N_2O, IOP at 3 hours after induction of anesthesia was significantly higher than that at 20 minutes after (T0).

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No one other than the authors contributed substantially to the performance of this study or to the drafting of the manuscript.

Competing Interest

The authors have no conflicts of interest to declare, financial or otherwise.

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Preoperative Anemia Is Associated with Increased Mortality Following Primary Unilateral Total Joint Arthroplasty

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Abstract

Background: Total joint arthroplasty is a commonly performed procedure for end-stage osteoarthritis. Preoperative anemia is a well-characterized and potentially modifiable risk factor for morbidity and mortality in non-cardiac surgery. This retrospective cohort study identified the prevalence of anemia in our total joint arthroplasty population and investigated if there was an association with all-cause mortality. Study Design and Methods: Using an electronic medical record, we examined all patients who underwent a primary unilateral total joint arthroplasty at Dartmouth-Hitchcock Medical Center from January 1, 2011 through July 1, 2015. Preoperative anemia thresholds were defined according to the World Health Organization as 12 mg/dl for women and 13 mg/dl for men. Kaplan-Meier survival analyses were performed to examine the relationship between preoperative anemia and all-cause mortality. Cox proportional hazards were calculated for various models adjusting for potential confounders. Results: 439 of the 3247 patients (13.5%) that underwent total joint arthroplasty met the preoperative definition for anemia. 48 patients (1.48%) died during 6470 patient years of follow up, generating an incidence rate of 7.4 deaths per 1000 patient years. The crude hazard ratio for death in anemic patients was 3.42 (95% CI 1.89, 6.82). In multiple models adjusting for various health related confounders, preoperative anemia was associated with a roughly two-fold increase risk of death compared to non-anemic patients. Conclusion: Preoperative anemia is prevalent in our population and it is associated with increased postoperative mortality in total joint arthroplasty patients, even when adjustments are made for significant co-morbidities.

Keywords

Anesthesia, Total Joint Arthroplasty, Anemia

*Corresponding author.

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1. Introduction

Total joint arthroplasty (TJA) is one of the most commonly performed procedures in the world. According to the Centers for Disease Control, there were over 1 million total knee and hip procedures performed in the United States in 2010 [1]. As of 2010, there were an estimated 7.2 million Americans living with a total knee or hip replacement, and, by 2020, two million Americans will undergo a hip or knee replacement each year [2]. Although total joint replacement is a highly effective treatment for end-stage osteoarthritis, patients can experience significant perioperative morbidity and mortality. According to one large retrospective analysis of 15,321 patients, between1.6% and 2.1% of patients will experience a major systemic complication following knee arthroplasty (TKA) [3]. Although rare, 30-day mortality is estimated to be 0.3% following total hip arthroplasty (THA) [4] and 0.18% following total knee replacement [3].

A robust body of literature has identified multiple risk factors associated with complications following TJA. Age and diabetes are independent risks factors for mortality following TKA [3]. Additionally, an increased risk of 90-day mortality following THR is associated with patients who have one of the following comorbid conditions: congestive heart failure, metastatic cancer, psychosis, renal disease, dementia, hemiplegia, cerebrovascular disease or chronic pulmonary disease [5]. Pre-existing conditions that have been associated with postoperative joint infections include rheumatologic disease, obesity, coagulopathy and preoperative anemia [5]. Of all the complications associated with TJR, death is the least studied. This is partially a function of the rare nature of the event, generating suboptimal power to understand the relationship to various exposures. Risk factors combined, with expert opinion, have been utilized to create multiple clinical pathways aimed at decreasing morbidity [6]-[8]. Decreasing mortality after TJA is an important outcome because over 1000 Americans will die each year within 30 days following a TJA [2]-[4]. Strategies to reduce this mortality are thus warranted.

In the past several years, preoperative anemia has been identified as a risk factor for morbidity and mortality in non-cardiac surgery [9]. Preoperative anemia is an attractive risk factor to understand because it can potentially be modified prior to elective surgery. In the orthopedic population, preoperative anemia has been linked to higher rates of transfusion, longer lengths of stay, and an increased risk of wound infection [10] [11]. However, long-term survival outcome data in the setting of preoperative anemia is limited, likely by both the rarity of the event and emphasis on 30 or 60-day survival metrics. As part of an exploratory quality improvement initiative, we conducted a retrospective cohort study with the following goals: 1) to identify the prevalence of preoperative anemia in our total joint population; 2) to determine whether preoperative anemia is associated with increased mortality in these patients.

2. Materials and Methods

Following approval and a consent waiver from the Dartmouth College Committee for the Protection of Human Subjects, (63 South Main Street, Hanover, NH 03755 USA; Study #00028930 approved on September 16, 2015) we examined 3247 patients undergoing primary unilateral TJA at a single institution from January 1, 2011 through July 1, 2015. Dartmouth-Hitchcock Medical Center is a tertiary care academic medical center located in Lebanon, New Hampshire, USA. Revision surgeries and bilateral procedures were excluded in the analysis. The data set generated from our electronic medical record (Epic Systems Corp.) included standard demographics, medical comorbidities, transfusion events, mortality, and laboratory data. The hemoglobin closest to the time prior to surgery was used for this study. Patients that did not have a hemoglobin value within six months prior to surgery were excluded. The decision to transfuse the patient was made by the primary anesthesia team.

Statistical Analysis

Stata Version 12 (StataCorp, College Station, TX, USA) was used for the statistical analysis. For the descriptive statistics, categorical variables are summarized as proportions and continuous variables are reported as means and standard deviations. The prevalence of preoperative anemia was measured, and defined as a hemoglobin concentration threshold of the World Health Organization's gender-based definition of 12.0 g/dl in women and 13.0 g/dl in men [12]. Univariate analysis, using chi-square statistic for categorical variables, and the t test for continuous variables, was performed to characterize the relationship of anemic status to patient characteristics (**Table 1**).

Kaplan-Meier curves were used to describe survival by preoperative anemia status (anemic versus not anem-

Table 1. Characteristics of patients having total joint arthroplastyby anemia status (2011-2015).

Characteristic	Mean (SD) or proportion		
	Anemic[∞] n = 439	**Not Anemic**[∞] n = 2801	**All** n = 3247
Age (years)	62.5 (10.9)[*]	63.4 (10.7)	63.9 (10.9)
Sex (male)	0.435	0.455	0.452
BMI	30.9 (6.9)	31.1 (7.5)	31.05 (7.7)
Race (white)	0.996	0.989	0.988
ASA III and higher[¶]	0.585[*]	0.362	0.392
Diabetes	0.196[*]	0.113	0.125
Cancer (any)	0.088	0.074	0.047
Coronary artery disease	0.164	0.134	0.138
Tobacco smoking	0.041	0.036	0.036
COPD	0.071[*]	0.044	0.047
Renal insufficiency	0.102[*]	0.023	0.033
Congestive heart failure	0.062[*]	0.011	0.018
Transfusion⊙	0.303[*]	0.061	0.938
Surgery type			
Anterior total hip	0.237[*]	0.329	0.317
Posterior total hip	0.243[*]	0.317	0.252
Total knee	0.447	0.427	0.430
Mortality (any day)	0.387[*]	0.111	0.148

[¶]Based on American Society of Anesthesiologists Health Classification; [∞]Anemia based on World Health Organization Classification of ≤12 mg/dl for women and ≤13 mg/dl for men; [*]p value < 0.05 comparing anemic to not anemic patients; ⊙Defined as any packed red blood cell transfusion during hospital admission.

ic). The log-rank test and Cox Proportional Hazards modeling were used for univariate and multivariate tests of statistical significance, respectively. Our a priori assumption was that there was co-linearity between anemic status and the occurrence of a postoperative transfusion. We thus plotted a separate survival curve for anemic patients by transfusion status. Transfusion was defined as administration of packed red blood cells at any time during the index hospital admission.

For all analyses, we set the p-value for statistical significance to 0.05 (2-sided).

3. Results

We identified 3247 patients that underwent primary TJA between January 1, 2011, and July 1, 2015. 530 patients had more than one joint replacement during this period. 48 patients (1.48%) died during 6470 patient years of follow up, generating an incidence rate of 7.4 deaths per 1000 patient years. Overall mortality as a function of age and time period is demonstrated in **Table 2**.

439 of the 3247 patients met the WHO criteria for anemia, generating a prevalence of 13.5%. A preoperative hemoglobin was unable to be obtained for seven patients. Anemic patients were slightly younger than the non-anemic patients (62.5 yrs. vs. 63.4 yrs.), and had higher rates of diabetes, COPD, renal insufficiency and congestive heart failure (**Table 1**). Patients with preoperative anemia were approximately five times more likely to receive a transfusion (30% vs. 6%).

The Kaplan-Meier curve (**Figure 1**) demonstrates an increased risk of mortality associated with patients who were anemic prior to their surgery (p value < 0.001). The crude hazard ratio (HR) for preoperative anemia was 3.42 (95% CI 1.89 - 6.2). To adjust for the American Society of Anesthesiologists (ASA) health score, as well as for several pre-operative comorbid conditions, we performed several regression analyses (**Table 3**). The HR decreased to 2.14 (95% CI 1.13, 4.07) when adjusted for ASA score and to 2.45 (95% CI 1.35, 4.46) when adjusted for a combination of diabetes mellitus, coronary artery disease, and COPD (**Table 3**).

The influence of transfusion on all-cause mortality was also explored. Patients having at least one packed red blood cell transfusion at anytime during their hospital stay were approximately two times more likely to die within the follow up period(HR 2.19; 95%CI 1.12, 4.27). **Figure 2** demonstrates that anemic patients who received a transfusion were 4.3 times more likely to die compared to anemic patients who did not get a transfusion (p value < 0.001HR 4.3; 95% CI: 1.5, 12.2). Additionally, non-anemic patients who received a transfusion were approximately 3 times more likely to die compared to non-anemic patients who did not get a transfusion (p value = 0.005, 95% CI: 1.43, 7.76).

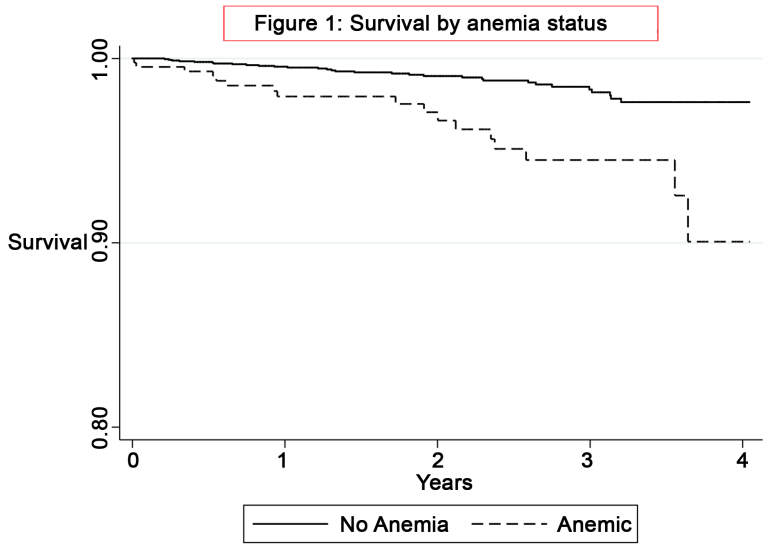

Figure 1. Kaplan-Meier survival curves by preoperative anemia status.

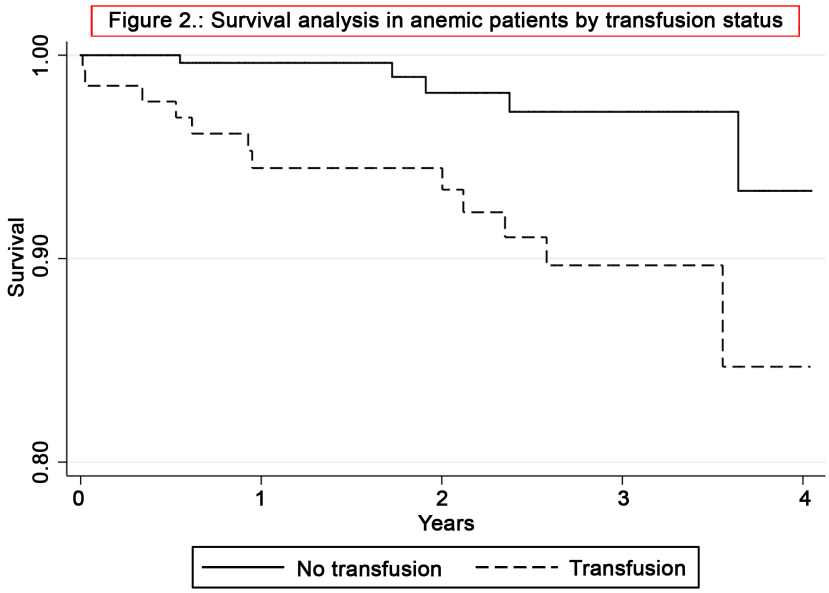

Figure 2. Kaplan-Meier survival curves for preoperative anemic patients by transfusion status.

Table 2. Mortality by age strata (April, 2011-March, 2015)[€] in total joint arthroplasty patients.

	N, (%)				
Age Category[¥] (N)	**30-day**	**60-day**	**90-day**	**One-year**	**Any-day**
<55 (601)	0, (0.00)	0, (0.00)	0, (0.00)	2, (0.33)	3, (0.50)
55 - 65 (1294)	0, (0.00)	0, (0.00)	1, (0.08)	3, (0.25)	7, (0.58)
>65 (1442)	2, (0.14)	2, (0.14)	3, (0.12)	12, (0.83)	38, (2.64)[*]
Any age	2, (0.06)	2, (0.06)	4, (0.12)	17 (0.52)	48, (1.48)

[€]Includes both unilateral primary total knee and total hip arthroplasty; [¥]Years; [*]p value <0.05 when compared to <55 years old.

Table 3. Regression analysis of anemia model predicting mortality.

	Hazard Ratio	**95% CI**	**P value**
Anemia (unadjusted)	3.42	1.89, 6.2	0.000
Model A[∞]	2.14	1.13, 4.07	0.019
Model B[⊙]	2.45	1.35, 4.46	0.003
Model C[¥]	2.97	1.64, 5.40	0.000
Model D[€]	2.85	1.52, 5.32	0.001
Model C[µ]	2.19	1.12, 4.27	0.021

[∞]Adjusted for age, body mass index; [⊙]Adjusted for advanced ASA status (≥3); [¥]Adjusted for diabetes mellitus, coronary artery disease, and COPD; [€]Adjusted for diabetes mellitus, congestive heart failure, and renal insufficiency; [µ]Adjusted for receiving a transfusion at any time during hospital postoperative course.

4. Discussion

We identified a high prevalence of preoperative anemia in patients undergoing TJA at our medical center. Preoperative anemia was associated with an increased risk of death as determined by a formal time to event analysis over the 4-year study period. The risk of death appeared independent of the presence of co-morbid medical conditions. We further identified that both preoperative anemic and non-anemic patients who received a blood transfusion were at an additional increased risk of death.

To our knowledge, this is the first observational data set including all patients undergoing TJA that has demonstrated an increased risk of mortality associated with preoperative anemia. Previous TJA outcomes research has focused on other metrics, partially because many large publicly available datasets such as the 5% Medicare sample do not track hemoglobin levels [3] [5] [13].

Our data is observational in nature and a causal relationship between anemia and death cannot be proven. Additionally, the observed relationship between anemia and death may be confounded by unmeasured variables. Given that our total death event number is 48, we are limited in our ability to model and control for additional confounders. Moreover, even if we assume a cause and effect relationship between anemia and death, it is unclear if correcting preoperative anemia will improve on mortality.

Despite these limitations, these data allow us to better educate patients and the care team regarding risks associated with TJA. Given that TJA is an elective procedure, our health system is collaboratively creating a care pathway based on an algorithm from The Network for Advancement of Transfusion Alternatives [14]. This process will allow us to further investigate patients who have been identified as anemic. Patients found to have reversible causes of anemia, such as iron deficiency, can be treated prior to the surgical intervention. We are also exploring the use of erythropoietin in certain anemic patient populations. Our future work will be to track mortality and the prevalence of anemia to assess the impact of our various interventions.

Disclaimer

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Spinal Anesthesia in Infant with Ventriculoperitoneal Shunt: A Case Report of Inguinal Hernia Repair

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Abstract

We are describing a case of a female infant with ventriculoperitoneal shunt scheduled for inguinal hernia repair under spinal anesthesia. The child was a premature newborn who, in a recent past, underwent surgery in general anesthesia for retinopathy correction with subsequent difficult mechanical ventilation weaning. The benefit of spinal anesthesia in high-risk infant was described and the risks of spinal anesthesia in the presence of a ventricular shunt device-especially dural leakage and infections were briefly discussed.

Keywords

Spinal Anesthesia, Prematurity, Ventriculoperitoneal Shunt, Inguinal Hernia

1. Introduction

Premature infants represent a challenge for pediatric anesthesiologists in fact about six percent of living born infants are delivered prematurely and many of them present clinical complications such as respiratory distress syndrome (RDS), bronchopulmonary dysplasia (BPD), chronic oxygen dependence and chronic airways hyper-reactivity [1]. The structure and function of immature lung predisposes to obstruction, difficulty with ventilation and hypoxia. Apnea of prematurity (AOP) is also a common respiratory pattern in this population, and appears to be related to immaturity of the central respiratory control mechanism, hypothermia, immature musculature and anemia [2]. Apneic episodes are frequent in the preterm infant although they may be precipitated by abrupt changes in pulmonary mechanics, oxygenation, airway stimulation or failure to maintain a patent airway,

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usually occur without a precipitating event (*i.e.* idiopathic). Anesthetic drugs may further decrease pharyngeal muscle tone and respiratory center output with a difficult return of spontaneous ventilation and complicated mechanical ventilation weaning.

Other two serious complications are retinopathy of prematurity (ROP) and intra-ventricular hemorrhage (IVH); the latter has an incidence of about 25% - 32% and often develops subsequent hydrocephalus that needs a shunt device insertion, mainly ventriculoperitoneal shunt (VPS) [3] [4]. Drugs used in general anesthesia often hold neurotoxic properties in this neurologically affected population, thus modern approach for emergency surgery is to limit general anesthesia and improve regional anesthesia whenever possible.

We present a case of a child with VPS scheduled for inguinal hernia repair who undergoes low abdominal surgery under spinal anesthesia (SA) without complications.

2. Case Description

The patient was a female born at 24th weeks gestational age (GA) after spontaneous vaginal delivery. Birth weight (BW) was 0.640 kg. She was immediately mechanically ventilated in neonatal intensive care unit (NICU) which was continued for fifteen days because of RDS, anemia and tetraventricular hemorrhage with subsequent obstructive hydrocephalus.

At 28th weeks gestational age she underwent VPS insertion. At 46th weeks GA the newborn developed severe ROP and therefore underwent urgent laser therapy and vitrectomy under general anesthesia with post-operative respiratory distress followed by sixteen days of mechanical ventilation with difficult ventilatory weaning. After three months the baby had hospital admittance because of an incarcerated right inguinal hernia with abdominal pain.

Pediatric surgeons decided for a urgent surgical procedure and after a fast pre-operative screening (blood count, coagulation tests and electrocardiogram) the neonate was moved to the operating theatre.

The anesthesists decided to perform a spinal anesthesia to preserve spontaneous breathing and avoid mechanical ventilation.

The baby girl weighted 5.1 kg and hematocrit was 35% with a normally functioning VPS (neurosurgical consultation was performed). The baby received antibiotic prophylaxis (50 mg/kg of ceftriaxone i.v.), and SA was performed according to our institutions guidelines [5] [6]. Before the surgical procedure the operating room was prepared to improve patient comfort with air temperature above 22 Celsius degrees, ambient dim lights, and soft classical music to promote natural sleeping.

She received a 0.8 mg/kg spinal 0.5% hyperbaric bupivacaine in lateral decubitus and she was monitored with intermittent non invasive blood pressure (left leg cuff position), three leads electrocardiography and continuous oxygen blood saturation [7] [8]. SA was performed at L4-L5 lumbar level of puncture with a 25 gauge-25 mm length pencil point needle. A little amount of lidocaine anesthetic cream (about 3 cm2 surface) was applied to the back skin 30 minutes before the puncture. The surgical procedure lasted 43 minutes with a good anesthesia quality (T5 sensory block level) and without intra-venous sedation/analgesia request. Post-operative pain management started with 20 mg/Kg rectal acetaminophen.

The baby presented normal cardiovascular parameters (no systolic pressure below 83 mmHg) with normal respiratory rate. No oxygen support was necessary with no arterial oxygen desaturation during medical procedure. The post-anesthesia recovery was performed in post-anesthesia care unit without complications with a fast and full sensory/motor recovery after 65 minutes from SA and with a continuous urine output above 1.5 ml/Kg/h.

No central temperature above 37.3 Celsius degrees was recorded.

The baby was discharged home 48 hours later without complications.

3. Discussion

VPS placement is the mainstay in the treatment of infants hydrocephalus (congenital or acquired) and the main indication in premature population is non-responding IVH to medical treatment with obstructive hydrocephalus [1].

Premature infants may require various surgical procedures and nearly 30% have inguinal hernia, which are bilateral in about 20% of patients [9].

Inguinal hernia is more frequent in premature infant with VPS (47%), probably because the amount of drained

cerebrospinal fluid (CSF) in abdomen increases intra-abdominal pressure [10]. One of the most important VPS complication is VPS infection, with an infection rate as high as 38% and four-fold increase rate in premature neonates [11] [12]. The most common pathogens involved are *Staphylococcus epidermidis*, *Staphylococcus aureus* and GRAM negative bacteria [12] [13]. Although no controlled studies show a significant decreasing infection rate with antibiotic prophylaxis, meta-analysis and experts opinion suggest perioperative antibacterial prophylaxis, particularly targeted on *Staphylococcus* spp. [14]. Many regimens are described including vancomycin, rifampicin, nafcillin and cephalosporins as in our institution [15].

Abdominal surgery in premature infants with VPS is usually performed under general anesthesia, Although defined contraindications to SA are not mentioned, many anesthesiologists refrain from using SA in this patients because of the fear of VPS infections.

In literature, we can find the same incident of meningitis after SA as after general anesthesia.

In adult population, the overall incident of bacterial infection after lumbar puncture is estimated 0.002% [16]). In children, the exact incidence of meningitis after SA is unknown, and the large study of Giaufre *et al.* (24.409 patients) reveals that only 2% of all regional anesthesias performed in children are spinal anesthesias and that only 30 patients are prematures [17]. Luz *et al.* report a case of fatal bacterial meningitis after SA in a preterm infant, but cannot exactly determine if the infection is consequent or just coincident with the lumbar puncture [18]. Easley *et al.* describe a case of aseptic meningitis after SA in a 2.5 month former premature but no causal relationship with SA is possibly proved [19].

Concerns that dural leakage following SA in children with VPS may compromise CSF flow and shunt performance seem to be overestimated [20]. During SA, we note a less CSF flow through the pencil point needle (about one drop of CSF/2.5 seconds versus normally one drop of CSF/1 second) probably due to a different CSF shunted flow into the spinal chord.

Preterm infants appear to represent a very high risk population for anesthesia in fact, most of them present chronic lung dysfunctions and the risk of a difficult ventilatory weaning after general anesthesia is very high.

In our institution, we decide to use SA as the first choice anesthesia to manage preterm newborns trying to limit perioperative respiratory complications avoiding general anesthesia.

In conclusion, we think that future studies comparing respiratory, surgical and neurological outcomes in preterm patients with VPS in general vs spinal anesthesia are indicated. Antibacterial prophylaxis is recommended and a strict aseptic technique is mandatory.

SA in experienced hands may be considered as a safe alternative to general anesthesia in selected patients. A further mandatory effort to improve children's safety is to build a very harmonious working team, in fact a not perfect integration of surgeons, anaesthesiologists and nurses staff may compromise the outcomes of those little patients.

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- Obstetrical Anesthesiology
- Ophthalmologic Anesthesiology
- Pain Medicine
- Pediatric Anesthesiology
- Plant Derivatives
- Regional Anesthesia
- Thoracic Anesthesiology
- Vascular Anesthesiology

We are also interested in: 1) Short reports—2 - 5 page papers where an author can either present an idea with theoretical background but has not yet completed the research needed for a complete paper or preliminary data; 2) Book reviews—Comments and critiques.

Notes for Intending Authors

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