cognitive, and motor skills. ANESTHESIOLOGY 40:453–458, 1974

3. Bruce DL, Stanley TH: Research replication may be subject specific. Anesth Analg 62:617, 1983

4. Bruce DL, Bach MJ: Effects of trace anaesthetic gases on behavioural performance of volunteers. Br J Anaesth 48:871–876, 1976

(Accepted for publication March 4, 1991.)

Anesthesiology
74:1161, 1991

Postanesthetic Hypoxemia and Oxygen Administration

To the Editor:—The results and conclusions recently described by Moller *et al.*[1] largely confirm our findings[2] regarding hypoxemia in the early postoperative period. Among the similarities between these two studies are the number of patients studied (200), the high incidence of postoperative hypoxemia observed, the relationship of hypoxemia to age and type of anesthesia, and the lack of relationship between hypoxemia and other factors such as preexisting pulmonary disease or obesity.

An important point of disagreement between the two studies, however, is in the effect of oxygen therapy to prevent postoperative hypoxemia. Fifty-five percent of the hypoxemic episodes (arterial oxygen saturation [Sp_{O_2}] ≤ 90%) Moller *et al.* observed in 32% of their patients occurred when patients were receiving oxygen. By contrast, fewer than 2% of patients in our study were hypoxemic when they were receiving oxygen on arrival at the recovery room as well as 1 h later. Although we performed single measurements of Sp_{O_2} after 10 min of oxygen therapy, while Moller *et al.* monitored Sp_{O_2} continuously, we do not believe this to be the cause of such a discrepancy in the incidence of hypoxemia during oxygen therapy.

The difference may lie in the way in which oxygen was administered. The patients of Moller *et al.* received at least 3 l/min nasal oxygen, and depending on the presence of hypoxemia, they might have had the oxygen flow increased. Eight percent of the hypoxemic episodes occurred because of accidental interruption of oxygen administration. In our study, we administered oxygen with a 35% Venturi mask. It is widely recognized that the inspired concentration of oxygen varies greatly when the gas is given through nasal catheter.[3] This variation in oxygen concentration is related to inspiratory flow rate, tidal volume, and inspiratory to expiratory times ratio.[4] An increase in all of these parameters decreases inspired oxygen concentration. The early postoperative period is characterized by variations in breathing pattern due to residual effects of anesthetics and neuromuscular blocking agents, pain, and stimulation of respiration by the staff while patients are regaining consciousness and resuming spontaneous ventilation. Each of these factors affects breathing in different ways which, in turn, increase or decrease the inspired concentration of a fixed flow of oxygen.

For these reasons, we think that in the postoperative period a fixed oxygen concentration device is more advisable to ensure a stable concentration of inspired oxygen. The disagreement between the two studies seems to favor our belief. In any case, further research is necessary to prove the relative efficiency of the different devices for administering oxygen during the early postoperative period.

JAUME CANET, M.D.
MONTSERRAT RICOS, M.D.
FERNANDO VIDAL, M.D.

Servei d'Anestesiologia i Reanimació
Hospital Universitari "Germans Trias i Pujol"
Badalona, 08916 Barcelona, Spain

REFERENCES

1. Moller JT, Wittrup M, Johansen SH: Hypoxemia in the postanesthesia care unit: an observer study. ANESTHESIOLOGY 73: 890–895, 1990
2. Canet J, Ricos M, Vidal F: Early postoperative arterial oxygen desaturation: Determining factors and response to oxygen therapy. Anesth Analg 69:207–212, 1989
3. Leigh JM: Variation in performance of oxygen therapy devices. Anaesthesia 25:210–222, 1970
4. Canet J, Sanchis J: Performance of a low flow O_2 Venturi mask: diluting effects of the breathing pattern. Eur J Respir Dis 65: 68–73, 1984

(Accepted for publication March 8, 1991.)

Anesthesiology
74:1161–1162, 1991

In Reply:—The large discrepancy between our findings[1] and those of Canet *et al.*[2] concerning hypoxemia and supplemental oxygen does not surprise us. When comparing two studies with different objectives, methodology, and material, one often finds disagreement. To illustrate the most obvious major differences in methodology used in the two studies, we have reanalyzed a part of our study.

Our study was a blinded observer study using continuous measurement of oxyhemoglobin saturation (Sp_{O_2}) with the pulse oximeter.[1] Canet *et al.*[2] measured Sp_{O_2} at two fixed single points, 10 or 20 min after arrival in the postanesthesia care unit (PACU) and again after 1 h. We have now analyzed our original data using their time schedule for measurements.

Table 1 illustrates a considerable reduction in the incidence of hypoxemia if we had recorded hypoxemia only 10 and 60 min after arrival in the PACU. Actually, only 9% of the patients would have been identified as hypoxemic if the study was performed with single measurement of Sp_{O_2}. Of these, only half (corresponding to 5% of the patients) occurred during oxygen administration.

TABLE 1. Number of Patients with Hypoxic Episodes in the PACU

Sp_{O_2} (%)	Measured at Two Fixed Points: 10 min after arrival, Number	10 min after arrival, %	60 min after arrival, Number	60 min after arrival, %	Original Data (Continuous Observation during the whole PACU Stay[1]), Number	Original Data, %
≤90	12	6	7	3.5	111	55
≤85	2*	1	0	0	56*	28
≤80	1†	0.5	0	0	26†	13

* Also counted among the patients at $Sp_{O_2} \leq 90\%$.
† Also counted among the patients at $Sp_{O_2} \leq 85\%$.

The incidence of hypoxemia in our study is still somewhat greater than that found by Canet *et al.*, and several factors, including the way in which oxygen was administered, may explain the difference.

JAKOB TRIER MOLLER, M.D.
Department of Anesthesia
University of Copenhagen
Herlev Hospital
DK-2730 Herlev, Denmark

REFERENCES

1. Moller JT, Wittrup M, Johansen SH: Hypoxemia in the post-anesthesia care unit: an observer study. ANESTHESIOLOGY 73: 890–895, 1990
2. Canet J, Ricos M, Vidal F: Early postoperative arterial oxygen desaturation. Determining factors and response to oxygen therapy. Anesth Analg 69:207–212, 1989

Anesthesiology
74:1162–1163, 1991

Anesthetic Management of a Patient with Cornelia de Lange Syndrome

To the Editor:—Recently, I was confronted with a patient with Cornelia de Lange Syndrome. This syndrome was described in 1933, and initially Cornelia de Lange called it "typus degenerativus Amstelodamensis".[1] It later was called "Amsterdam dwarfism". Reference to the recent review article on anesthetic management of disproportionate dwarfism by Berkowitz *et al.*[2] will not be helpful in a patient with Cornelia de Lange Syndrome since these patients have a normal proportion in build of body and limbs[3] and dwarfism is not invariable[1]; i.e., the term "Amsterdam dwarfism" lacks accuracy.[1] These patients have some growth and mental retardation, microbrachycephaly, and hirsuitism.[4] The syndrome was not found in American literature until 1963. It affects 1 in 30,000 to 1 in 60,000 live births.* The basic pathology is believed to be hypoplasia of the mesenchyme.† The etiology is believed to be genetic, with suggestions of autosomal recessive or dominant inheritance.[1,5,6] A most important feature is a striking delay in the maturation of structure and function of most organ systems, including the central nervous system.[6] These patients' "leprechaunoid" facies is striking.[7] Two thirds die before age 1 yr.[8] Death occurs from aspiration in infancy,[5,6] and from infections and bowel obstruction later on.[5–8,†] Depression of the immune system is reported.[6,†] Our patient, at age 30¾, is one of the oldest. They may have self-destructive tendencies,[5,8,*] and it was this characteristic that brought our patient to the hospital for amputation of an infected deformed distal upper extremity.

Preanesthetic considerations include immaturity of organ systems with possible unpredictable responses to drugs. This may be anticipated from some limited endocrine studies and autopsy findings, suggesting hypopituitarism with abnormal function and structure of the thyroid and adrenal glands.[3] Schlesinger *et al.*[3] suggested that antibiotics and steroids be given to cover infections and the stress of surgery. Problems during induction include the potential for aspiration due to frequent pressure of hiatal hernia and difficulty with tracheal intubation. Intubation may be difficult due to a short neck, often webbed; a high-arched palate, sometimes clefted; and a small mouth with micrognathia. Care must be taken in resumption of oral intake due to chewing and swallowing difficulties and the potential for aspiration.[5,8,*] In infants, apneic episodes are not uncommon.[8,9]

Reported anesthetic experience with these patients is sparse and sometimes fraught with problems, including death.[1,10,†] Our patient exhibited some unpredictable or sensitive responses to drugs. He had a past history of agitation after haloperidol and chlorpromazine, and hypotension when first given chlordiazepoxide. He became somnolent after 10 mg metoclopramide given preoperatively. His anesthetic requirements appeared minimal. Although severely retarded and uncooperative in surgery, he was well sedated, and his trachea was intubated with little technical difficulty after only 0.5 mg midazolam and 1 ml fentanyl. Ten milligrams dexamethasone was given. Hypnosis resulted after 60% nitrous oxide in oxygen. An additional 1 mg midazolam, 1 ml fentanyl, and 3 mg vecuronium provided uneventful general anesthesia for the hour-long orthopedic procedure. Relaxant reversal was accomplished with 0.3 mg glycopyrrolate and 1.5 mg neostigmine. Tracheal extubation and recovery were uneventful.

WILLIAM W. SARGENT, M.D., M.S.
Staff Anesthesiologist
St Luke's Hospital
915 East First Street
Duluth, Minnesota 55805

* O'Donnell D, Davis PJ, King NM: Management problems associated with Cornelia de Lange syndrome. Special Care in Dentistry Jul–Aug 5(4):160–163, 1985

† Takeshita T, Akita S, Kawahara M: Anesthetic management of a patient with Cornelia de Lange syndrome. Anesthesia Progress 34:63–65, 1987

REFERENCES

1. Filippi G: The de Lange syndrome: Report of 15 cases. Clin Genet 35:343–363, 1989
2. Berkowitz ID, Raja SN, Bender KS, Kopits SE: Dwarfs: patho-