EFFECT OF BOLUS DOSE REMIFENTANIL ON

HAEMODYNAMIC AND INTRAOCULAR

PRESSURE RESPONSES TO

TRACHEAL INTUBATION AND EXTUBATION

DURING RAPID SEQUENCE INDUCTION

AND RECOVERY OF ANAESTHESIA

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Abstract

*The effect of bolus dose of rernifentanil on the pressor responses to laryngoscopy*

*and tracheal intubation or extubation during rapid sequence*

*induction and recovery of anesthesia was assessed in a randomized,*

*double- blind, placebo- controlled study in two groups of 20 patients*

*each. No premedication was given. Anesthesia was induced with thiopental*

*5mg / kg. Followed by saline placebo or remifentartil 1.5 pg /kg and*

*suxamethonium. After the trachea get intubated, anesthesia maintained*

*with isoflurane* and *nitrous oxide in oxygen, at the end of surgery irthalational*

*anesthesia discontinued, the study* drug *readministered and residual*

*muscle relaxant was reversed. Mean arterial blood pressure,* heart

*rate and intra-ocular pressure were recorded immediately before induction,*

*before irttubatthn, at interval until* 5 min *after treachecd intubation,*

*before extubation and then at interval until 5 min after tracheal extubation.*

*There was a significant decrease in mean arterial pressure (MAP),*

*heart rate (1-IR) and irttraocular pressure in remifentand* group *(group R)*

compared *with saline placebo group (group S) from time of administration*

*of the study drugs to the end of the study at* 5th *min after extubation (Pc*

*0.001). we conculoded that remifentanil 1.5 pg/ kg administered during*

*induction and recovery, prevented the pressor response and increase* in

*lOP after irttubation or extubation.*

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**Introduction**

Tracheal intubation and extubation

may be associated with increases

in arterial pressure, heart

rate and development of myocardial

ischaemia (Shribman et al.,

1987 and Edwards et al., 1994).

Plasma concentrations of catecholamines

are increased (Derbyshire

et at, 1983 and Lowrie et

al.. 1992).

Suxamethonium is used to facilitate

rapid tracheal intubation

in patients who are at risk of

aspiration of gastric contents.

However, administration of suxamethonium,

tracheal intubation

or extubation are associated

with an increase in intra-ocular

pressure (10P) (Mirakhur et al.,

1987).

These responses may be attenuated

by several methods, including

administration of i. v.

opioids. vasodilators, B-blockers,

local anesthetics or by deeping

of anesthesia (Crawford et at,

1987; Stoeling, 1979; Vucevic et

at. 1992 and Miller et al.,

1995). The action of agents used

at intubation and extubation

should be brief to allow rapid return

of spontaneous respiration,

protective reflexes and avoid residual

hypotension, respiratory

depression or sedation (McAtamney

et al., 1998 and Shajar et al.,

1998).

Renaifentanil is a new opioid

agent that is structurally unique.

An ester bond renders it subject to

rapid hydrolysis by non specific

blood and tissue esterases and

thus it has a short half-life, resulted

in rapid onset and offset of action

(Thompson and Rowbotham,

1996). Speed of onset of effect is

rapid (1-2 min) and similar to that

of alfentanil (Egan et al., 1996).

Bolus doses of remifentanil have a

very short duration of action (9.5

min) (Glass et al., 1993). Therefore,

remifentanil may be appropriate

for attenuation of pressor

and IOP responses to brief but

noxious stimuli.

The aim of this study was to

assess the effectiveness of a bolus

dose of remifentanil on the pressor

and IOP responses to tracheal

intubation and extubation under

the conditions of rapid sequence

induction and recovery of anesthesia.

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**Patients and Methods**

Forty patients of ASA grade I or

H undergoing elective eye surgery

and requiring tracheal intubation

were studied. They were aged between

18 and 50 years. weighed

within 75-125% of ideal for height.

Exclusion criteria included a history

of glaucoma, oesophageal reflux

or hiatus hernia, cardiovascular

disease, taking vasoactive

medication, allergies to any of the

study drugs and if a difficult intubation

was anticipated. All patients

received no premedication.

Patients were allocated randomly

to one of two groups (n= 20

each): group R, received remifentanil

1.5 p.g/kg diluted to 5 ml

with normal saline and group S.

received normal saline 5m1. Monitoring

of ECG. heart rate (HR), peripheral

arterial hemoglobin oxygen

saturation (5P02) and arterial

blood pressure by automated,

noninvasive oscillotomometry

(Dinamap) were commenced. Tetracine

0.5% drops were administered

to the non-operated eye and

IOP was measured using an applanation

tonometer.

After 3 min period of preoxygenation.

anesthesia was induced

with a bolus dose of thiopentone 5

mg/kg adminsitered over 30 s.

The study treatment was then given

over 30 S and followed by suxamethonium

1 mg/kg Laryngoscopy

and tracheal intubation were

performed using a standard Mcintosh

blade laryngoscope 1 min later.

After tracheal intubation and

cuff inflation, anesthesia was

maintained with 1% isoflurane

and 60% nitrous oxide in oxygen,

and the lungs were ventilated to

normocapnia. There was no further

stimulation of the patient

during this period of the study.

Five min after tracheal intubation

atracurium 0.4 mg/kg was

given and all patients received 20

mg feldene rectally. MAP during

surgery was controlled to within

10% of resting preoperative (baseline)

value by titration of inspired

isoflurane concentration. At the

end of surgery with the last suture

(time 0), the patient received remifentanil

1.5 p.g/kg or an equivalent

volume of saline over 30 S.

tetracine 0.5% drops were readministered

to the non-operated

eye and 1013 was measured using

an applanation tonometer.

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This was followed by neostigmine

2.5 mg with atropine 1.5 mg

to antagonize the residual effect of

the nondepolarizing neuromuscular

blocking agent. Mechanical

ventilation was continued with

100% oxygen, followed by oropharyngeal

suction. Extubation was

performed in a standard manner

when patients were able to open

their eyes and squeeze a hand on

command. Extubation time was

defined as the time between the

study drug administration (To)

and extubation.

Mean arterial blood pressure

(MAP), heart rate (HR) ad intraocular

pressure (I013) were measured

at the begining and end of preoxygenation

(averaged together) (baseline),

after injection of induction

agent (To). Just before laryngoscopy

(Ti) ant at 1 min intervals for

5 min after tracheal intubation

(T2- 6).

Another recording of MAP, HR

and IOR were measured after

readminstration of the study drug

(TEa) and evry 1 min from time

TEo to 5 min after extubation. The

incidence of coughing or gagging

at extubatMn was noted, sedation

score (0 = alert and responsive, 2

= drowsy but responsive to verbal

command and 3 = unresponsive to

verbal comand), ventilatory frequency

and nausea were assessed

on arrival in the recovery area and

at 15 min interval thereafter.

Data collected was statistically

analysed using two way and multivariate

analysis of variance for

repeated measures (ANOVA, MANOVA

with treatment group and

time as the between and within

group factors), and paired and unpaired

t-test and MannWhitney

tests as appropriate.

**Results**

There were no significant differences

in patient characteristics

between the groups (Table 1). Arterial

pressure, heart rate and intraocular

pressure during induction

are detailed in table 2. Heart

rate increased significantly in both

groups after induction of anesthesia

(Pc 0.01). The mean HR Just

before laryngoscopy was significantly

lower in group R (P 0.001)

compared with group S and compared

with preinduction values

(P<0.001) and was significantly

greater 1-5 min after intubation in

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group S compared with group R

(P< 0.001). There were no significant

differences in HR before

and after intubation in group R

(Fig. 1).

MAP was significantly lower in

group R (Pc 0.01) compared with

group S before intubation. MAP

increased after intubation significantly

in group S (Pc 0.01) and

decreased significantly (Pc 0.05)

in group R compared with preinduction

values. MAP was significantly

lower in group R (P.< 0.001)

in all times after intubation compared

with group S (Fig. 3).

There was a significant decrease

in TOP in group R at times

1-6 and increase in group S at

times 2-6 compared with baseline

(Pc 0.01). There was a significant

difference in LOP in group R compared

with group S from times 2-6

(P<0.001) (Fig. 5).

Two patients in group R expressed

bradycardia (HR< 55

beat/ min) or hypotension (MAP <

50 mm Hg), or both, requiring rescue

medication. No other side effects

were recorded during induction

of anesthesia.

MAP, HR and TOP changes during

extubation and recovery are

detailed in Table 3. There were

no significant differences in MAP,

HR and IOP between groups at

the end of surgery crime 0).

MAP in group S significantly increased

(P< 0.01) from baseline

at times 1-3 after extubation

(Fig. 4). There was a significant

difference (13.< 0.001) between

group R and S from time 1-4 and

at time 5 (Pc 0.01) after extubaton.

HR increased significantly-in

group S from baseline (Pc 0.05)

for 2 mm after extubation, and reduced

significantly from baseline

at time 1 in group R after extubation.

There was a significant difference

(Pc 0.001) between group R

and S at limes, 1 and 2 and significant

difference (Pc 0.01) at limes

3-5 (Fig. 2). Intraocular pressure

increased significantly (Pc 0.01)

from baseline at times 1-3 and at

times 4-5 (Pc 0.05) in group S.

while it decreased significantly

from baseline (P< 0.01) at times 1-

5 in group R. There was a significant

difference (13.< 0.001) between

group R and S at times 1-5

(Fig. 6).