Comparison of the Efficacy of Intravenous Lignocaine and Sublingual Nifedipine in Attenuating the Haemodynamic Response to Laryngoscopy and Intubation - A Clinical Study

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ABSTRACT *Aim- To asses the hemodynamic response to laryngoscopy and endotracheal intubation in normal patients of ASA grade-1 and 2.To compare the efficacy of sublingual nifedipine and intra venous lignocaine in attenuating the hemodynamic response in intubation against a control where no drug was given. Materials and Methods-Randomised controlled study was conducted on 45 patients who were ASA grade 1 and 2 who were to undergo elective surgeries were randomly allocated to 3 groups.Group A with 15 patients served as control recieveing no drugs.Group B 15 patients received IV 2 % lignocaine 1.5mg/kg prior to pentothal sodium.Group C 15 patients given sublingual nifedipine 10mg 3 minutes prior to pentothal sodium.Pulse and Blood pressure was noted immediately after intubation and at intervals of 1,2,3, and 5 minutes. Statistical Analysis was done using paired t test in each group. <0.05 considered statistically significant.For comparison between the groups chi square test was used. Result-In the control group there was significant rise in pulse rate which is statistically significant.This rise persistant even after 5 minutes.Systolic,diastolic as well as Mean arterial pressure were also elevated.In group B,lignocaine was partially effective in attenuating the pulse rate,but very effective in attenuating rise in systolic,diastolic and MAP which is most evident after 5 mins.In group C,Nifedipine was more effective in attenuating the rise in systolic BP and very significant in reducing diastolic blood pressure,maximum effective at 5 minutes and failed completely to attenuate the rise in pulse rate. Conclus ion-From the study it can be concluded that there is only partial protection from increase in heartbeat in case of iv lignocaine,but has proved to be very effective in controlling the rise in blood pressure.Nifedipine was more effective than lignocaine in controlling rise in both systolic and diastolic Blood pressure.*

Key words *- nifedipine,intra venous lignocaine,intubation.*

Introduction

Technique of general anaesthesia using thiopental for induction and endotracheal intubation after muscle relaxant was introduced my McEwan,A Scottish surgeon in 1880.Laryngoscope was used first by Kirstein in 1895. Endotracheal intubation produces reflex cardiovascular responses,particularlytachycardia,increase in cardiac output,transient rise in CVP.This reflex is mediated via sympathetic nervous activity.This reflex rise in heart rate and CVP could be life threatening for patients with hypertension,Ischemic heart disease and raised intra cranial pressure.

To attempt to maintain normal physiology during anaesthesia and surgery,there are several methods used.Like 4% lignocaine spray or IV lignocaine 1.5 mg/kg 90 seconds before laryngoscopy,IV Sodium nitroprusside 1-2 microgram/kg 15 seconds before scopy,IV fentanyl 8 microgram/kg 2-4 minutes before scopy,Betablocker orally 2 hours before or IV esmolol,Volatile agents like halothane,intranasal and NTG spray,Nifedipine sub-lingual,IV clonidine 1.25 microgram/kg 15 minutes before scopy etc.

In our centre, it has been a practise to administer intra venous lignocaine in any patient considered at risk from hypertensive response.In this study,anassesement of efficacy of sub-lingual nefidine in attenuating these hemodynamic responses to intubation was studied against a control,where no drug has been given and another using intravenous lignocaine.We included only normotensives.

Aims and Objectives

Aim of this study is to compare the efficacy of lignocaine in a dose of 1.5mg/kg intravenously with nifedipine 10mg sublingually in attenuating the haemodynamic response to laryngoscopy and intubation.

Materials and methods

This randomized controlled study was conducted on 45 patients who were to undergo elective gastroenterology surgeries,belonging to ASA 1 and 2.Informed consent was obtained.Routine investigations were within normal limits, randomly allocated to group-A, group-B and group-C.

Group-A

15 patients served as control who received no drug to attenuate pressor response to laryngoscopy and intubation.

Group-B
15 patients were included in group B and were given IV 2% lignocaine 1.5mg/kg 90 seconds before laryngoscopy.

Group C
15 patients were given sublingual Nifedipine 10milligram 3 minutes prior to pentathol Sodium.

All patients received pre-medications of INJ.Pethidine,1 mg/kg and Promethazine 0.5 mg/kg 1 hour before surgery.

All were induced with 5-6 mg/kg thiopental after 3 minutes of pre-oxygenation.Intubation done after 2mg/kg of succinylcholine was given,usingmacintosh curved blade laryngoscope.Within 90 seconds,by the same person ventillated manually with nitrous oxide and oxygen.Pulse and BP were noted immediately after intubation and at intervals of 1 minute,2,3 and 5 minutes.During this time,no surgical stimulation was allowed.Anaestesiawas maintained with 0.1 mg/kg vercuronium,oxygen,nitrous oxide and isoflurane.BP,pulse recorded every 10 minutes.Reversal was given 0.05 mg/kg neostigmine and 0.02 mg/kg of atropine and extubated.

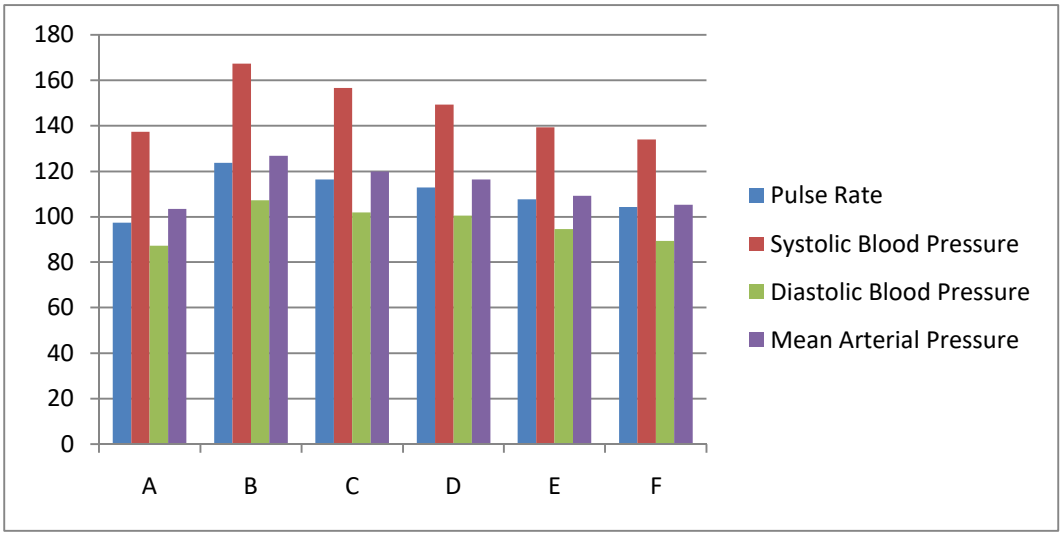
Statistical analysis of the data obtained was done using paired t test in each group. Below 0.05 was considered statistically significant.

For comparison between 2 groups, chi square test was used.

Observation-

Control Group
Mean values

Number		Pulse Rate	Systolic Blood pressure	Diastolic Blood pressure	Mean arterial Blood pressure.
A	Before Induction(after starting i/v line attaching cardioscope)	97.4	137.33	87.2	103.5
B	Immediately after laryngoscopy and intubation	123.8	167.3	107.3	126.8
C	One minute after intubation	116.4	156.6	102.0	119.9
D	Two minutes after intubation	112.9	149.3	100.6	116.5
E	Three minutes after intubation	107.7	139.3	94.6	109.2
F	Five minutes after intubation	104.4	134.0	89.3	105.3

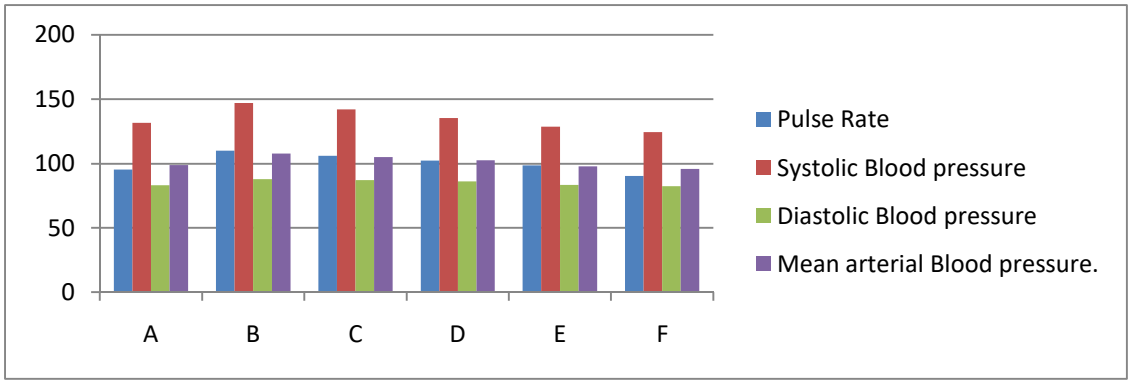


A – Before induction B - Immediately after intubation
C - One minute after Intubation D - Two minutes after intubation
E - Three minutes after intubation F - Five minutes after intubation

Mean Values of Intravenous lignocaine group

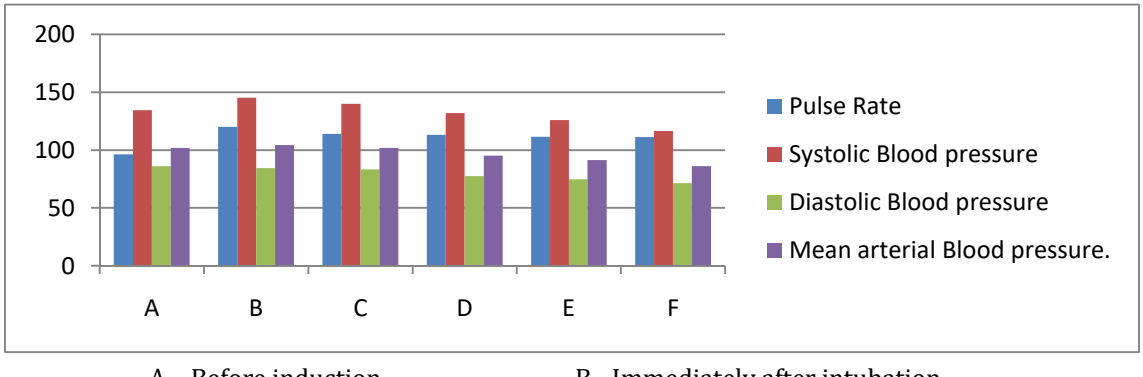
	Pulse Rate	Systolic Blood pressure	Diastolic Blood pressure	Mean arterial Blood pressure.
Before Induction(after starting i/v line attaching cardioscope)	95.4	131.8	83.0	99.0
Immediately after laryngoscopy and intubation	110.0	147.0	88.0	107.8
One minute after intubation	106.0	142.0	87.3	105.2
Two minutes after intubation	102.4	135.3	86.0	102.5
Three minutes after intubation	98.6	128.6	83.3	98.0
Five minutes after intubation	90.4	124.5	82.13	95.8

A – Before induction B - Immediately after intubation
C - One minute after Intubation D - Two minutes after intubation
E - Three minutes after intubation F - Five minutes after intubation



Mean values of sublingual Nifedipine group.

	Pulse Rate	Systolic Blood pressure	Diastolic Blood pressure	Mean arterial Blood pressure.
Before Induction(after starting i/v line attaching cardioscope)	96.5	134.6	86.2	101.8
Immediately after laryngoscopy and intubation	120.13	145.3	84.6	104.5
One minute after intubation	114.0	140.0	83.3	102.0
Two minutes after intubation	113.3	132.13	77.3	95.3
Three minutes after intubation	111.6	126.0	74.6	91.4
Five minutes after intubation	111.2	116.6	71.3	86.26



A – Before induction B - Immediately after intubation
C - One minute after Intubation D - Two minutes after intubation
E - Three minutes after intubation F - Five minutes after intubation

Comparison of mean values of pulse rate in control,lignocaine and nifedipine groups.

Mean Values of pulse rate	Control	Lignocaine	Nifedipine
A – Before induction	97.4	95.4	96.5
B-Immediately after intubation	123.8	110.0	120.13
C-One minute after Intubation	116.4	106.0	114.0
D-Two minutes after intubation	112.9	102.4	113.3
E-Three minutes after intubation	107.7	98.6	111.6
F-Five minutes after intubation	104.4	90.4	111.2

Comparison of mean values of systolic Blood pressure in control,lignocaine and nifdipine group

Mean Values of systolic blood pressure	Control	Lignocaine	Nifedipine
A – Before induction	137.33	131.8	134.6
B-Immediately after intubation	167.3	147.0	145.3
C-One minute after Intubation	156.6	142.0	140.0
D-Two minutes after intubation	149.3	135.3	132.13
E-Three minutes after intubation	139.3	128.6	126.0
F-Five minutes after intubation	134.0	124.5	116.6

Comparison of mean values of diastolic Blood pressure in control,lignocaine and nifdipine group

Mean Values of diastolic blood pressure	Control	Lignocaine	Nifedipine
A – Before induction	87.2	83.0	86.2
B-Immediately after intubation	107.3	88.0	84.6
C-One minute after Intubation	102.0	87.3	83.3
D-Two minutes after intubation	100.6	86.0	77.3
E-Three minutes after intubation	94.6	83.3	74.6
F-Five minutes after intubation	89.3	82.13	71.3

Comparison of mean values of mean arterial Blood pressure in control,lignocaine and nifdipine group

Mean Values of mean arterial blood pressure	Control	Lignocaine	Nifedipine
A – Before induction	103.5	99.0	101.8
B-Immediately after intubation	126.8	107.8	104.5
C-One minute after Intubation	119.9	105.2	102.0
D-Two minutes after intubation	116.5	102.5	95.3
E-Three minutes after intubation	109.2	98.0	91.4
F-Five minutes after intubation	105.3	95.8	86.26

Results

1) Pulse rate

Group A-showed a range of rise of 4-63 beats per minute, with a mean rise of 26 beats per minute. Thereafter the pulse rate gradually decreased but did not come back to the basal pulse rate within 5 minutes, though it showed a downward trend.

Group B- there was a mean rise of 15 beats per minute and it ranged from 0-44. the rise in pulse rate immediately after intubation was less, but not statistically significant. After five minutes, the pulse rate came down in group B with mean decrease of 7.5 beats per minute with a range of 0-32 with increase in pulse rate from baseline into two cases only. This decrease was statistically significant with t value 3.06. This indicated that lignocaine is partially effective in attenuating pulse rate immediately after intubation but it is effective in reducing pulse rate 5 minutes after intubation.

Group C- there was a mean increase of 23 beats/minute and the fluctuations in the rise of pulse rate ranged between 18-32. Compared to Group A, the mean rise was almost similar. In group A it was 26 beats/mt and Group C 23 beats/mt. The rise in pulse rate in nifedipine group was statistically significant with t value of 15.8. When compared to group A, there was no statistical significance.

After five minutes the mean rise was 14 beats/minute with a range of 8-20. Here also the rise in pulse rate is statistically significant.So in group C, there was considerable rise in pulse rate which was persistent even after five minutes.

3) Mean Arterial Pressure

Group A- mean arterial pressure rise was 25mm of Hg and the range was 10-40mm of Hg. This rise was statistically significant with a t value of 10.3. After 5 minutes/ the mean rise was only 3.5mm of Hg which was also statistically significant with- t value of 2.7.

Group B- mean arterial pressure rise was 8mm of Hg ranged between 0 and 33 mm of Hg.When compared to control, where mean rise was 25mm of Hg, lignocaine was effective in attenuating the rise in mean arterial pressure. This was significant statistically also with t value of 3.44. After 5 minutes there was a reduction in mean arterial pressure with mean value of 3.6mm of Hg

Group C-Pressure response to intubation the mean rise was only 2mm of Hg. In two cases there was reduction in mean arterial pressure. The range was 0-10mm of Hg. This rise was statistically not significant.When compared to control group, nifedipine was found to be extremely effective in attenuating mean arterial pressure which was statistically significant with a t value of 7.9.After 5 minutes, there was significant reduction in mean arterial pressure with a mean decrease of 15.6mm of Hg. -This was also statistically significant.

4) Systolic Arterial Pressure

Group –A- There was a mean increase of. systolic arterial pressure by 30mm of Hg. The range of fluctuations varied from 10-50mm of Hg. The pressure slowly came down to reach the original value in five minutes but in two cases, remained high even after five minutes. On statistical analysis the rise in systolic blood pressure immediately after intubation was very significant with a t value of 7-35.After five minutes, most of the values came down to the base line value and statistically also, there was no significant rise with t value 0.27.

Group –B the mean rise in systolic arterial pressure was only 15mm of Hg and the rise ranged from 0-60mm of Hg . This rise was significant statistically with t value of 3.4. When compared to croup A where mean raise was 30mm of Hg, lignocaine attenuated the rise in systolic arterial pressure immediately after intubation. Statistically also this reduction in systolic blood pressure was significant with t value of 2.4. After five minutes there was a reduction in systolic arterial pressure below the base line value in most of the cases with mean value of -10 with a range of 0 to -22 with only 2mm of Hg rise in a single case. This reduction was also statistically significant with t value of 4.1.

Group –C- there was a mean rise of only 10mm of Hg with a range of -6 to -30mm of Hg and in two cases there was actual reduction in systolic blood pressure. This rise in systolic blood pressure immediately after intubation was statistically significant with t value of 4.75. When compared to group A where mean rise was 30mm of Hg nifedipine was more effective than lignocaine in attenuating the rise in blood pressure immediately after intubation and this was statistically significant with a t value of 3.9.After five minutes there was a reduction in systolic arterial pressure below the base line value with a mean of -18 and range of -10 to -30 and rise of 10mm of Hg in a single case. This reduction was statistically significant with t value of 8.4.

5) Diastolic Arterial Pressure

Group A- The mean rise in diastolic arterial pressure was 22mm of Hg with a range of 10-40mm of Hg. This significant rise is statistically also significant with t value of 6.3.After five minutes the mean rise was 4mm of Hg with a range of 0-10mm of Hg. This rise was also statistically significant with t value of 3.8. Therefore there was a rise in diastolic blood pressure immediately after intubation which is persistent after five minutes. The value did not come back to base line value even after five minutes.

Group B: In group B where intravenous lignocaine was used, the mean rise in diastolic pressure was only 4mm of Hg with a range of 0-20mm of Hg and in two cases there was reduction below the base line value. When statistically analysed this rise from base line value was not significant with t value of 1.97. When compared to control group, there was significant reduction in diastolic blood pressure when intravenous lignocaine was used to attenuate the pressure response. This was statistically proved with t value of 4.1.After five minutes in most cases the DBP came down to base line value with reduction below the base

line value in 6 cases. The range of fluctuations were -10 to 10 with rise in diastolic blood pressure in three cases. The mean change in blood pressure was -1.8mm of Hg. This showed that lignocaine is effective in attenuating the pressure Response to intubation compared to the no drug group by reducing diastolic blood pressure significantly immediately after intubation.

Group C: In group C the mean rise observed was -1.6mm of Ha or in other words diastolic blood pressure was below the base line value in most of the cases with a rise only in one case. This reduction was not statistically significant with t value of 0.6. Compared to control group where the mean rise in diastolic blood pressure was 22mm of Hg, in group C, there was significant reduction in pressure response which was statistically significant with t value of 6.5. After five minutes, there was a mean reduction in diastolic blood pressure of 14.9 which was statistically significant with a t value of 7.6.

Discussion

The aim of the present study was to assess the haemodynamic response to laryngoscopy and endotrachealintubation in normal patients of ASA Grade I and II, bynoting the rise in pulse rate and blood pressures – thesystolic arterial pressure, diastolic arterial pressureand mean arterial pressure. The trend of theseparameters during the first five minutes of intubationwas also noted.

In all the patients similar an anesthetic procedures wereemployed and the same laryngoscope blade was used asTakishima, Noda and Hijaki (1964) compared the effectsof laryngoscopy with different laryngoscope blades andconcluded that there were differences in thehemodynamic response with different blades[1]. Macintoshcurved blade laryngoscopy was used in all the patients.Another contributory factor influencing tachycardia andpressor response was the induction-intubation time. This was standardised at 90 seconds in all the cases. The stimulation of different areas of thelaryngopharynx and tracheobroncheal tree differ withindividual technique of laryngoscopy and intubation. In order to avoid variations due to this/ all the patients were intubated by the same anaesthesiologist.

Responses to laryngoscopy and intubation in the control group (Group A)

These patients showed a mean rise of 26 beats perminute which is in quite agreement with the findings ofLojiTokishima, Kango Noda and Nasakihgaki (1960) whonoted a mean rise of pulse rate of greater than20beats/mt in 9 patients out of 50 patients studied[1]. In most of the previous studies done,simultaneous measurement of the plasma adrenaline andnor adrenaline levels were done, which were found to beparallel to the rise in the pulse rate and bloodpressure. Hence the stress response is interpreted asdue to reflex sympathetic adrenal stimulation. Thiswas proved in a study in anaesthetised cats wheremechanical stimulation of upper respiratory tract wasshown to increase nervous activity in .cervical sympathetic afferent fibers[2] (Tomori and Widdicombe 1969).A significant increase in plasma concentrationof nor adrenaline in response to tracheal intubation had been established[3,4]. (Russell et al 1981, Dubyshei 1983). These exaggerated sympathetic responses mightcause little harm in normotensive patients. PriyaRobert et al (1971) had shown that these vasopressors and tachycardia reflex were much more aggravated in patients with hypertension, myocardial ischemia, inotropic failure, ischemic arrhythmias and substantial worsening of left ventricular wall function[5].

Response to laryngoscopy and intubation inthe intravenous lignocaine group (Group B)

Regarding dose Abu Madi, Bngo Kessler and Joseph M. Yacoub (1977) found that a dose less than 1.5mg/kg was ineffective in protecting against cardiac arrythmias, while larger doses caused borderline protection against hypertension and tachycardia, the smaller dose prevented only a rise in systolic blood pressure[6]. In respect to the route of administration, intravenous lignocaine has been compared to orotracheal lignocaine spray[7] (D.R.Dubyshire, G. Smith and K.J. Achala"1987). Both wereeffective in attenuating the hemodynamic response to intubation but the intravenous route was found to be better.The mechanism ofprotective action of intravenous lignocaine against thestress response are by its anti arrhythmic (Class II)action that means by cardiac and central nervous system depression.The lignocaine dose used in the present studydid not cause any toxic symptoms. Direct depression of myocardium withlignocaine does not occur below plasma concentration of5 microgram/ml (Grosman et al 1969)

Responses to laryngoscopy and intubation in the sublingual nifedipine group (Group C)

Nifedipine, a calcium channel blocker when used to attenuate was found to be effective incontrolling the pressor response. But the sublingual routerequired 3-5 minutes for its peak effect (R. M. JonesRecent advances, anaesthesia and analgesia 1985).Hence it had to be given three minutes

beforeinduction. This produced tachycardia in almost allpatients. It failed completely to attenuate the rise in pulse rate.Systolic blood pressure rise was statisticallysignificant in nifedipine group with a mean rise of10mm of Hg. When compared to control where mean risewas 30mm of Hg, nifedipine was more effective thanlignocaine in attenuating rise in systolic bloodpressure.. Nifedipine has a greater coronary andperipheral arterial vasodilator property (Runs 1984).There was nominal effect on venous capacitance vessels (Robinson et al 1980). Nifedipine had little or nodirect depressant effect on sino atrial or atrioventricular nodal activity (Rowland et al 1979).Nifedipine may produce myocardial depression especially in patients with aortic stenosis, pre existing left ventricular dysfunction or thes'e onadrenergic antogonist therapy. Nifedipine is 3-10times more effective in inhibiting contraction ofcoronary artery smooth muscle than the myocardialcontractile cells I- Thus nifedipine is able to dilatecoronary arteries in doses that does not decrease themyocardial contractility. This drug is thus of particular value in prinz metal angina. It also has a place in the treatment of hypertension, myocardial protection subsequent to infarction and cardiac surgery. This may be safely added to beta adrenergicantagonist.

Conclusion

1.Endotracheal intubation causes significant rise in pulse rate, systolic/ diastolic and mean arterial pressure in control group. After 5 minutes rise in pulse rate, diastolic and mean arterial pressure is persistant but systolic blood pressure came down to base line value.

2.Intravenous lignocaine in a dose of 1.5 mg/kg given 90 sec. before laryngoscopy attenuates pressor response significantly immediately and 5 minutes after intubation. But attenuation in pulse rate is significant after 5 minutes but not immediately after intubation. Rise in pulse rate in lignocaine groups is Less compared to nifedipine group.

I. Sublingual nifedipine does not attenuate the rise in pulse rate but effectively attenuates the systolic, diastolic as well as mean arterial pressure. There is a reduction in diastolic pressure, instead of rise, which is more evident after 5 minutes.

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