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Dexmedetomidine Combination with Fentanyl on Brain Oxygenation using Invos in Intubed ICU Patients at Adam Malik Hospital, Medan

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Abstract:-

Introduction: Brain consumes 20% of the total oxygen within our body. The most of it used to produce ATP. High consumption of oxygen and absence of oxygen reserves will cause the cessation of active brain perfusion in an unconscious state. If blood flow does not return within 3-8 minutes, ATP reserves will decrease and causing permanent cell damage. NIRS is a non-invasive monitoring technique to measure cerebral oxygen saturation, the product of this cerebral oximeter is INVOS 4100. Dexmedetomidine (DEX) is a selective 2-adrenoreceptor agonist that has sedation effect, analgesics, anti-anxiety and sympathetic nervous inhibition, DEX also has a neuroprotective effect by producing 2 receptor that is mediated by tyrosine kinase receptor. An increase in rSO2 was found after administration of dexmedetomidine sedation. The combination of dexmedetomidine and fentanyl was found to have a good sedative effect and more controlled hemodynamics in CPB surgery.

Methods: This study is an observational analytic study with a prospective cohort design. The sample were patients who were treated by ventilator on the ICU of general hospital Haji Adam Malik Medan. This study uses consecutive sampling technique to gain the total of the sample. All of the sample will be given dexmedetomidine and fentanyl then we will monitor the sample every 6 hours for 24 hours then the data will be analyzed.

Result: The result of the paired T-test and Wilcoxon test are there is a significant difference between before and after dexmedetomidine administration in MAP (p-value = 0.012), left rSO2 (p-value = 0.000) and right rSO2 (p-value = 0.000) and there is no significant difference in systole (p-value = 0.169), diastole (p-value = 0.129), HR (p-value = 0.974) and SpO2 (p-value = 0.405).

Conclusion: there is a significant difference between before and after dexmedetomidine administration in MAP, left rSO2 and right rSO2 and there is no significant difference in systole, diastole, HR and SpO2.

Keywords:- *Dexmedetomidine, Fentanyl, INVOS, oxygenation, combination.*

I. INTRODUCTION

The brain is responsible for 20% of the body's total oxygen consumption. Most of the brain's oxygen consumption (60%) is used to produce adenosine triphosphate (ATP) to support nerve electrical activity. The metabolic rate of the brain, commonly referred to as brain oxygen consumption (CMRO2), is about 3-3.8 ml/100 g/min in adults. CMRO2 is highest in the cerebral cortex and usually corresponds to electrical activity in the cortex. Because of the high consumption of oxygen and the absence of oxygen reserves, cessation of cerebral perfusion will result in unconsciousness within 10 seconds when oxygen falls rapidly below 30 mmHg. If blood flow does not return within 3-8 minutes, ATP stores will decrease and permanent cell damage occurs (Butterworth et al., 2013; Parnia et al., 2016).

Dexmedetomidine (DEX) is a selective 2-adrenoceptor agonist that has sedative, analgesic, antianxiety, and sympathetic nerve inhibitory effects. Patients given DEX can be awakened easily without causing respiratory arrest. Therefore, DEX is considered an ideal sedative and analgesic agent for use in ICU patients. DEX is a dextro-isomer of medetomidine which exerts a sedative and analgesic effect. In addition, DEX also produces neuroprotective effects by producing 2 receptors mediated by receptor tyrosine kinases. Furthermore, DEX initiates the release of various growth factors by producing astrocytes to participate in nerve cell protection. DEX is able to activate survival enzymes by activating 2-adrenoceptors that produce cardioprotective effects by controlling protein kinase pathways, protein kinase B, and extracellular nitric oxide synthesis in endothelium (Yoo et al., 2015).

In a study conducted by Jyrson and colleagues on hemodynamic comparisons given Dexmedetomidine-Fentanyl with Midazolam-Fentanyl in children who had heart surgery with cardiopulmonary bypass, the results showed that patients in the DEX group experienced an increase in systolic, diastolic, and lower heart rates. and required less isoflurane administration than the other groups (Klamt et al., 2010).

NIRS is a non-invasive monitoring technique that measures regional cerebral oxygen saturation (rSO2) and detects changes in cerebral oxyhemoglobin, deoxyhemoglobin, and oxidized cytochrome aa3 concentrations in brain tissue. In brain tissue, the vascular compartment is dominated by veins (70%-80% v 20-30%

arterial). Cerebral venous blood oxygen saturation is about 60% compared to 98-100% in arterial blood. Based on this assumption, the mean value of rSO2 is 60% to 70%. During cardiovascular surgery, the downward trend in rSO2 may reflect a decrease in cerebral oxyhemoglobin saturation (Green & Kunst, 2017).

The use of NIRS (Near-infrared Spectroscopy) technology has been shown to correlate with the results of brain tissue oxygenation. The availability of this non-invasive technology allows estimation of cerebral oxygenation during conditions where there may be changes in oxygen delivery due to anemia, decreased cardiac output or changes in oxygen saturation (Ameloot et al., 2015).

In a study of patients undergoing carotid endarterectomy with local anaesthesia, it was found that the use of a cerebral oximeter (INVOS 4100) in conscious patients gave good and reliable results. Cerebral oximetry is also an efficient tool that does not require a special expert to operate it, just a surgeon or an anesthesiologist. Cerebral oximetry can also be used by anesthetists to manipulate blood pressure to get the desired perfusion before the patient loses consciousness (Ritter JC et al., 2010).

Research conducted in America in 2000 aimed at examining the ability of Invos to detect cerebral ischemia. From this study, it was found that changes in rSO2 in carotid endarterectomy surgery can help predict the possibility of cerebral ischemia, but are limited. It is not possible for the operator to see a critical value where there is a higher probability of cerebral ischemia. Monitoring rSO2 has a sensitivity and specificity that has a low false negative rate (2.6%), but a high false positive rate (66.7%) (Samra s. et al., 2000).

Therefore, based on the background and references of the above study, the investigators wished to assess cerebral saturation in intubated patients receiving dexmedetomidine combined with fentanyl in the ICU of RSUP. H. Adam Malik Medan.

II. RESEARCH METHODS

This study is an observational analytic study with a prospective cohort design to examine the effects of sedation and analgesics dexmedetomidine and fentanyl on brain oxygenation using INVOS in mechanically ventilated patients in the ICU H. Adam Malik Hospital.

A. Place and time of research

This research was conducted in the ICU RSUP. Haji Adam Malik Medan. This research takes place from January until the sample is fulfilled, since the researcher determines the title of the research, prepares a research proposal, collects research data, and makes a report on the research results until the research results seminar, which takes place from January 2022 until the number of samples is met.

B. Research Population and Sample

The study population was intubated patients who were treated in the ICU of H. Adam Malik Central General Hospital Medan. The research sample was patients who were treated with a ventilator in the ICU of the H. Adam Malik General Hospital Medan who met the research criteria. The technique of obtaining samples is by consecutive sampling, namely looking for patients who meet the inclusion and exclusion criteria until the required number of samples is met.

C. Inclusion Criteria

The inclusion criteria in this study were intubated patients who were admitted to the ICU and aged 18-64 years

D. Exclusion Criteria

Exclusion criteria in this study were the patient's family refused to participate in the study, had a history of hypersensitivity to dexmedetomidine, had a history of hypersensitivity to fentanyl, patients with head trauma or intracranial problems, and post cardiac arrest patients.

E. Dropout Criteria

Patients will be dropped out of the study if the patient experiences cardiac and pulmonary emergencies after drug administration, and the patient experiences cardiac arrest less than 24 hours after drug administration.

F. Informed Consent

After obtaining approval from the Ethics Committee, the patient received an explanation of the procedure to be carried out and stated his willingness in writing in the informed consent form.

G. Tool

This research will use tools and materials as well as working methods as described below. The tools used in this study are non-invasive automatic monitoring devices (blood pressure, heart rate, respiratory rate, ECG, oxygen saturation) (Infinity), invasive cerebral saturation monitor (INVOS), 50 cc syringe (B-Braun), extension tube (B-Braun), and stationery and research forms.

H. Ingredient

The research material consisted of the sedating drug dexmedetomidine (Precedex), the analgesic drug Fentanyl (Fentanyl citrate), normal saline 0.9% fluid (B-Braun), and emergency medicine Ephedrine (Vasodrine) 5 mg/ml and Atropine Sulphate (Atropine). Sulfate) 0.25 mg/ml, Inotropic drugs: Dobutamine, and Norepinephrine vasopressor drugs

I. Procedure

The method of work carried out in this research is as follows. This research was conducted after obtaining approval from the Health Research Ethics Committee, Faculty of Medicine, University of North Sumatra / Haji Adam Malik Hospital, Medan. After obtaining informed consent and being approved by the health research ethics committee, Faculty of Medicine, University of North Sumatra, all samples were reassessed and included in the inclusion and exclusion criteria. Samples that were determined to meet the inclusion and exclusion criteria were

ISSN No:-2456-2165

recorded and all patients were given dexmedetomidine with a loading infusion of 1 mcg/kgBW in 10 minutes followed by 0.5 mcg/kgBW hour and 0.5 mcg/kgBW/hour fentanyl with a syringe pump. Patients were monitored for hemodynamics and peripheral and cerebral saturation using INVOS. Each patient studied maintained a MAP >65 mmHg. Each change was assessed every 1 hour T1, T2 6 hours, T3 12 hours, T4 18 hours, T5 24 hours. The collected data was processed and analyzed statistically using the statistical product and service solution (SPSS) windows program. For numerical data presented in the mean ± standard deviation and statistical tests to compare between statistically significant or significant.

J. Data analysis

After the required data has been collected, it will then be re-checked for completeness before being tabulated and processed. So, the next step is the coding process to make it easier to tabulate. The data that has been collected is analyzed using a computer program. Numerical data is shown in mean ± SD (standard deviation), while categorical data is shown in sum (percentage).

K. Ethical Issues

This research was conducted after obtaining permission from the health research ethics commission, Faculty of Medicine, University of North Sumatra. The patient's family was previously explained about the objectives, benefits, and risks and matters related to the study. Then asked to fill out a form of willingness to be a research subject (informed consent).

III. RESEARCH RESULT

This study is an observational analytic study using a prospective cohort design to examine the effects of sedation and analgesics dexmedetomidine and fentanyl on brain oxygenation using INVOS in mechanically ventilated patients in the ICU. This research was carried out after passing the Ethical Clearance and the number of samples was fulfilled at the Haji Adam Malik General Hospital Medan.

This study was followed by 29 samples that have met the inclusion criteria and exclusion criteria. Descriptive data before treatment, 1 hour, 6 hours, 12 hours, 18 hours, and 24 hours after treatment are displayed in the form of medians with minimum and maximum values if not normally distributed, and the mean with standard deviation if normally distributed based on the Sapphiro Wilk normality test. . The data presented include systolic blood pressure, diastolic blood pressure, MAP (Mean Arterial Pressure), HR (Heart Rate), left rSO 2 , right rSO 2, and SpO 2 . The results of the descriptive statistical analysis before treatment (T0) can be seen in table 4.1 below.

Data	median	Min	Max	*mean*	±SD
Systolic (mmHg)	121	100	165	126.66	16.73
Diastole (mmHg)	70	64	100	72.21	7.07
MAP (mmHg)	88	79	99	88.83	5.16
HR (x/minute)	89	72	112	89.69	11.69
rSO_2 Left (%)	53	50	57	53.07	1.85
rSO_2 Right (%)	55	51	60	55.62	2.56
SpO_2 (%)	98	97	99	98.28	0.80

Table 1: Descriptive Data Test Results Before Treatment (T0)

Before treatment, the median value of systolic blood pressure was 121 mmHg with a minimum value of 100 mmHg and a maximum of 165 mmHg. The median diastolic blood pressure of our study subjects was 70 mmHg and ranged from 65 mmHg to 100 mmHg. In our study the mean MAP value was 88.83 ±5.16 mmHg. The average heart rate before treatment was 89.69 ±11.69 times per minute. The median left cerebral regional oxygen saturation was 53% (50-57%) and the median right cerebral regional oxygen saturation was 55% (51-60%). The median peripheral oxygen saturation is 98% (97-99%).

Table 2 below is the results of descriptive statistical analysis 1 hour after treatment (T1)

Data	median	Min	Max	mean	±SD
Systolic (mmHg)	120	106	153	126.28	15.25
Diastole (mmHg)	69	62	81	69.93	3.92
MAP (mmHg)	88	79	99	88.83	5.16
HR (x/minute)	90	70	118	89.24	11.58
rSO_2 Left (%)	65	55	75	66,10	5.22
rSO_2 Right (%)	69	54	79	68,10	4.60
SpO_2 (%)	98	97	99	98.28	0.80

Table 2: Descriptive Data Test Results 1 Hour After Treatment (T1)

One hour after treatment, the median systolic blood pressure was 120 mmHg (106 - 153 mmHg) and the diastolic blood pressure was 69 mmHg (62 - 81 mmHg). The mean MAP one hour after treatment was 88.83 ±5.16 mmHg. The average heart rate one hour after treatment was 89.24 ±11.58 beats per minute. The median left brain regional oxygen saturation increased to 65% (55-75%) and the right brain regional median oxygen saturation also

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increased to 69% (54-79%). Her median peripheral oxygen saturation remained 98% (97-99%).

Table 3 The following are the results of descriptive statistical analysis 6 hours after treatment (T2)

Data	median	Min	Max	mean	±SD
Systolic (mmHg)	120	105	151	125,90	15.88
Diastole (mmHg)	70	60	80	70.21	4.55
MAP (mmHg)	88	76	97	88,72	5.80
HR (x/minute)	87	69	109	87.79	11.23
rSO_2 Left (%)	69	54	77	68.21	5.35
rSO_2 Right (%)	72	61	79	71.07	4.52
SpO_2 (%)	98	97	99	98.28	0.70

Table 3: Descriptive Data Test Results 6 Hours After Treatment (T2)

Six hours after treatment, the median systolic blood pressure was 120 mmHg (105 - 151 mmHg) and the diastolic blood pressure was 70 mmHg (60-80 mmHg). The mean MAP six hours after treatment was 88.72 ±5.80 mmHg. The average heart rate six hours after treatment was 87.79 ±11.23 beats per minute. The mean regional left brain oxygen saturation increased to 68.2 ±5.35% and the right brain regional oxygen saturation also increased to 71.07 ±4.52%. Her median peripheral oxygen saturation remained 98% (97-99%).

The results of descriptive statistical analysis 12 hours after treatment (T3) can be seen in table 4.4 below:

Data	median	Min	Max	mean	±SD
Systolic (mmHg)	121	108	163	125.79	15.91
Diastole (mmHg)	71	64	88	73.17	5.44
MAP (mmHg)	91	79	110	90.69	6.32
HR (x/minute)	87	70	111	87.93	11.23
rSO_2 Left (%)	70	60	76	69.10	3.81
rSO_2 Right (%)	71	62	78	71.03	3.89
SpO_2 (%)	98	97	99	98.28	0.70

Table 4: Descriptive Data Test Results 12 Hours After Treatment (T3)

Twelve hours after treatment, the median systolic blood pressure was 121 mmHg (108 – 163 mmHg) and the diastolic blood pressure was 71 mmHg (64 – 88 mmHg). In this study, the mean MAP 12 hours after treatment increased to 90.69 ±6.32 mmHg. The average heart rate is 87.93 ±11.23 beats per minute. Left brain regional oxygen saturation increased with a median of 70 mmHg (60 – 76 mmHg) and right brain regional oxygen saturation tended to be stable with a mean of 71.03 ±3.89%. Her median peripheral oxygen saturation remained 98% (97-99%).

The results of descriptive statistical analysis 18 hours after treatment (T4) can be seen in table 4.5 below.

Data	median	Min	Max	mean	±SD
Systolic (mmHg)	122	107	161	127.44	14.98
Diastole (mmHg)	72	60	88	73.31	5.56
MAP (mmHg)	91	77	112	91.34	6.32
HR (x/minute)	89	72	109	88,72	10.24
rSO_2 Left (%)	70	62	77	69.52	3.47
rSO_2 Right (%)	70	62	79	70.79	5.16
SpO_2 (%)	98	97	99	98.41	0.57

Table 5: Descriptive Data Test Results 18 Hours After Treatment (T4)

The measurement results after 18 hours after treatment included: The median systolic blood pressure was 122 mmHg (107 - 161 mmHg) and the average diastolic blood pressure was 73.31 ±5.56 mmHg. The median MAP 18 hours after treatment was 91 mmHg (77 – 112 mmHg). The average heart rate 18 hours after treatment was 87.72 ±10.24 times per minute. The mean regional left brain oxygen saturation increased to 69.52 ±3.47% and the right brain regional oxygen saturation had an average of 70.79 ±5.16%. Her median peripheral oxygen saturation remained 98% (97-99%).

The results of descriptive statistical analysis 24 hours after treatment (T5) can be seen in table 4.6 below.

ISSN No:-2456-2165

Data	median	Min	Max	mean	±SD
Systolic (mmHg)	120	100	162	125.52	16.40
Diastole (mmHg)	72	65	81	73.56	4.61
MAP (mmHg)	90	80	106	91.00	5.58
HR (x/minute)	90	70	108	89.66	10.04
rSO_2 Left (%)	71	60	79	71.21	3.64
rSO_2 Right (%)	73	64	79	71.97	4.41
SpO_2 (%)	98	97	99	98.41	0.57

Table 6: Descriptive Data Test Results 24 Hours After Treatment (T5)

At 24 hours after treatment, the median systolic blood pressure remained stable at 120 mmHg (100 - 162 mmHg) and the diastolic blood pressure was 72 mmHg (65-81 mmHg). In this study, the average MAP 24 hours after treatment increased to 91.00 – ±5.58 mmHg. The average heart rate six hours after treatment was 89.66 ±10.04 beats per minute. Regional left brain oxygen saturation 24 hours after treatment increased by a median of 71% (60-79%) and right brain regional oxygen saturation also increased with a mean of 71.97 ±4.41%. Meanwhile, the median peripheral oxygen saturation remained 98% (97-99%).

Statistical test analysis was conducted to determine whether there was a difference between before and after treatment. Paired T-test was performed on normally distributed data and Wilcoxon's test on data that were not normally distributed.

Data	P- *value*
Systole T0 & T5	0.169
Diastole T0 & T5	0.129
MAP T0 & T5	0.012
HR T0 & T5	0.974
rSO_2 Left T0 & T5	0.000
rSO_2 Right T0 & T5	0.000
SpO_2 T0 & T5	0.405

Table 7: Results of Data Analysis Before and After Administration of Dexmedetomidine

Based on the results of the analysis in Table 4.7, it is known that there are significant differences between before and after dexmedetomidine administration in MAP (p-value = 0.012), left rSO_2 (p-value = 0.000), and right rSO_2 (p-value = 0.000). It is known that there is no significant difference between before and after dexmedetomidine administration in systole (p-value = 0.169), diastole (p-value = 0.129), HR (p-value = 0.974), and SpO_2 (p-value = 0.405).

Statistical test analysis was conducted to determine whether there were differences in all types of data based on time. The Annova Repeated Measure test was performed on normally distributed data and Friedman's test on data that were not normally distributed.

Data	Mauchly's Sphericity	P-value Sphericity	P-value Greenhouse-Geisser
Systole T0-T5	0.069	**0.491**	0.470
T0-T5 . diastole	0.043	0.002	**0.006**
MAP T0-T5	0.000	0.000	**0.002**
HR T0-T5	0.054	**0.162**	0.181
rSO_2 Left T0-T5	0.003	0.000	**0.000**
rSO_2 Right T0-T5	0.606	**0.000**	0.000
SpO_2 T0-T5	0.000	0.804	**0.623**

Table 8: Results of Analysis of All Data Based on Time

Based on the results of the analysis in Table 4.8, it is known that there are significant differences in overall data on diastolic (p-value = 0.006), MAP (p-value = 0.002), left rSO_2 (p-value = 0.000), and right rSO_2 (p -value = 0.000). It is known that there is no significant difference between before and after dexmedetomidine administration in systole (p-value = 0.491), HR (p-value = 0.162), and SpO_2 (p-value = 0.623).

IV. DISCUSSION

In this study, the patient's hemodynamic status and brain oxygen saturation were monitored at one hour and every 6 hours to 24 hours after administration of dexmedetomidine and fentanyl. The hemodynamic status monitored included systolic, diastolic blood pressure, MAP, and HR. Prior to treatment, systolic blood pressure had a median of 121 mmHg (100 – 165 mmHg). Systolic and diastolic blood pressure changed with treatment, namely

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there was a decrease and increase but still within the normal range, with a median of 120-122 mmHg systolic and 69-72 mmHg diastolic. MAP also decreased and increased but remained stable within the normal range, where the lowest mean MAP was 88.72 ±5.80 mmHg at 6 hours after treatment and the highest average MAP was 90.69 ±6.32 mmHg at 12 hours after treatment. Along with the treatment, the heart rate was also stable with an average of 87.79 – 89.66 times per minute. This shows that there is a stable hemodynamic status in patients after administration of dexmedetomidine and fentanyl.

After being given treatment, there was no desaturation in the brain but an increase in the value of regional brain oxygen saturation. In the left brain 1 hour after being given treatment the saturation increased to 65% (55-75%) and the highest was reached at 24 hours after the treatment was given with a median of 71% (60 - 79%). The same thing with the right brain, 1 hour after treatment the saturation increased to 69% (54 – 79%) and the highest mean of 71.97 ±4.41% was achieved 24 hours after treatment. A previous study by Metry*et al* also stated the same thing, namely there was an increase in regional brain oxygen saturation and there was no cerebral desaturation during the *cardiopulmonary bypass surgery procedure* with the anesthetic agents propofol and dexmedetomidine (Metry et al., 2019) . Propofol causes an increase in cerebrovascular resistance as well as a decrease in CBF (*cerebral blood flow*) and CMR (*cerebral metabolic rate*). Dexmedetomidine also has the effect of causing a decrease in CBF but the effect is dose dependent.

The results of the analysis in this study showed that there was a significant difference between before and after administration of dexmedetomidine on the hemodynamics of patients, namely the *mean arterial pressure* (MAP) which increased 24 hours after being treated ($p<0.05$). Other hemodynamic markers such as systolic pressure, diastolic, and heart rate did not experience a significant difference ($p>0.05$) and were stable at the reference value. This is in accordance with previous research, namely research by Hashemian*et al.* who investigated hemodynamic changes in CABG surgery with dexmedetomidine administration. In that study, postoperative MAP values were significantly higher in patients receiving dexmedetomidine ($p=0.01$) compared to controls (Hashemian et al., 2017) . A previous study looking at the hemodynamic effects of multiple doses of dexmedetomidine in healthy subjects found that dexmedetomidine at doses of 1 and 2 mcg/kg transiently increased MAP that peaked 3 minutes after administration, then decreased MAP up to 5.5 hours post-administration in dose 0.25 – 2 mcg/kg (Bloor et al., 1992) .

The absence of significant differences in systolic blood pressure, diastolic blood pressure, and heart rate before and after dexmedetomidine administration may indicate that dexmedetomidine infusion can decrease the hemodynamic response to intubation, reduce the incidence of anesthesia-related emergencies by decreasing hemodynamic response and ETT release time . Tanskanen et al., 2006).

Based on the results of the analysis in our study, it is known that the distribution of data between before and after treatment found a significant decrease and increase in diastolic blood pressure ($p<0.05$), a significant increase in MAP ($p<0.05$), and regional oxygen saturation. left brain ($p<0.05$) and right ($p<0.05$).

The results in this study where the changes in systolic blood pressure were not significant, diastolic blood pressure and MAP had significant changes but all three were still at normal reference values in accordance with previous studies which stated that dexmedetomidine can provide hemodynamic stability as well as effective sedation and analgesia, also inhibiting the response. mediated by the sympathetic system at critical moments during surgery. (Easley & Tobias, 2008; Gerlach & Dasta, 2007; Klamt et al., 2010; Mukhtar et al., 2006).

In this study, there was a significant increase in right and left brain regional oxygen saturation ($p<0.05$) after administration of dexmedetomidine and fentanyl. Based on previous research, Dexmedetomidine is known to possibly reduce blood flow to the brain due to a decrease in cerebral perfusion pressure. However, it does not affect brain oxygenation despite hyperventilation. The decrease in cerebral blood flow by dexmedetomidine is dose dependent (Cetin et al., 2016; Metry et al., 2019).

The limitations of this study include that in our study we did not randomize the administration of dexmedetomidine with a combination of fentanyl and there were no controls, so all patients were given dexmedetomidine with a combination of fentanyl. In addition, in our study there was a limited research sample.

V. CONCLUSION

There was a significant difference between before and after administration of Dexmedetomidine in MAP (p - *value* = 0.012), left rSO2 (p - *value = 0.000), and* right rSO2 (*p-value* = 0.000). There was no significant difference between before and after administration of Dexmedetomidine combined with fentanyl in systole (p -*value* = 0.169, diastole (p - *value* = 0.129), HR (p - *value* = 0.974), and SpO2 (p - *value* = 0.405). There was a significant difference in overall data on diastolic (p - *value* = 0.006), MAP (p - *value* = 0.002), left rSO_2 (p- *value* = 0.000), and right rSO_2 (p- *value* = 0.000) before and after administration of dexmedetomidine combined with fentanyl There was no significant difference overall between before and after administration of dexmedetomidine combined with fentanyl in systole (p - *value* = 0.491), HR (p - *value* = 0.162), and SpO2 (p - *value* = 0.623).

ISSN No:-2456-2165

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