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Research Article

# Effect of Hypnoanesthesia on Endogenous Opioids (Beta Endorphin and Enkephalin)

**Ihyan Amri[1], Abdul Hafid Bajamal[2,*], David S. Perdanakusuma[3]**

## ABSTRACT

**Background:** Hypnoanesthesia is a state of anesthesia achieved through hypnosis techniques. Meanwhile, hypnosis is a condition in which the mind receives information without analyzing it. Hypnoanesthesia has been empirically utilized in surgery since the 18th century, but the mechanism remains unclear. **Objective:** This study aims to prove nociceptive pain relief due to hypnoanesthesia in minor surgery, with indications of changes in the serum levels of several excitatory and inhibitory neurotransmitters in the physiological mechanism of pain. **Methods:** The study subjects included patients with benign soft tissue tumors consisting of 40 people who were divided into 2 groups (treatment and control). The treatment group underwent minor surgery using hypnoanesthesia, while the control group underwent minor surgery using 2% lidocaine local anesthesia. Pain in both groups was measured by FPS (Face Pain Scale) and monitored by a vital sign monitor. Changes in the serum levels of glutamic acid, substance P, beta-endorphin and enkephalin neurotransmitters in both groups before and after the intervention were analyzed using ELISA. **Results:** There were no significant changes in serum levels of neurotransmitters, pre and post intervention in both groups, both excitatory neurotransmitters Glutamic Acid and Substance P and inhibitory neurotransmitters Beta Endorphins and Enkephalin ($p > 0.05$). **Conclusion:** Based on the results of the data analysis, it can be concluded that the empirical nociceptive pain relief occurred in patients who underwent minor surgery with hypnoanesthesia and there was no significant change in beta-endorphin, enkephalin, glutamate acid, and substance P serum levels before and after hypnoanesthesia.
**Key words**: Hypnoanesthesia, Beta-Endorphin, Enkephalin, Glutamic Acid, Substance P.

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## INTRODUCTION

Hypnosis is a state of increased concentration and focal acceptance in which the suggested perception can change or replace the actual perception. Hypnosis is also described as a change in consciousness, dissociation, and regression states.[1] Hypnosis is a condition in which the mind receives information without analyzing it.[2] The information in the hypnotic process is often referred to as a suggestion. A suggestion is an idea that is accepted without critical analysis on it.[3] According to James Braid, in a hypnotic state when one of the five senses receives continuous stimulation from the outside, such as the eye seeing a shiny object or the ear hearing the same sound continuously, a concentration of nerve power will occur. When this concentration has occurred, even with the simplest suggestion, someone will easily be directed toward that suggestion.[2]

Hypnoanesthesia is an anesthetic state achieved by administering anesthetic suggestions. Anesthetic and analgesic suggestions start working at the third-order nerve level in the central nervous system where the perception of pain is changed to disappear or relief. Thus, pain stimuli transmitted by the spinothalamic tract are perceived as numb at the amygdala, limbic system, hypothalamus, thalamus, and cerebral cortex levels.[4,5] Hypnoanesthesia is preceded by changes in the perception of pain. Suggestions given in the form of visual, sound, and/or tactile stimuli penetrate the Reticular Activating System (RAS) in the reticular formation of the brain stem and are received by the dorsolateral prefrontal cortex as stimuli which then undergo a selection and interpretation process to become a new perception, from perception of pain to perception of pain relief.[6] RAS in a relaxed state becomes inactive so that the suggestions given are not critically thought or not analyzed.[2-3] These new perceptions are stored in the ventromedial prefrontal cortex as short-term memory and can be tagged using "anchors" as "passwords" to be reused when needed.[4,7]

Signals from the prefrontal cortex will be responded to by the brain through the HPA (Hypothalamic Pituitary Adrenal) axis which stimulates the prohormone POMC (Pro-opiomelanocortin) in the pituitary which undergoes hydrolysis to become beta-endorphin and periaquaductal gray to produce enkephalin. Beta-endorphin is a powerful endogenous opioid peptide neurotransmitter found in the neurons of the peripheral nervous system as well as the central nervous system. As long as there is severe pain, beta-endorphin provides an analgesic effect to reduce pain.[8] Beta-endorphin will activate μ receptors, while enkephalin will activate δ receptors found in neurons in the spinal cord, brain stem, and mesencephalon. The activation of these three receptors will inhibit two neurotransmitters, namely glutamate acid and substance P, which transmit nociceptive pain so that the pain decreases until it relieves. At this stage, any action in the area subjected to surgical procedure cannot be felt because they already have a new perception.[9]

Although hypnosis in the field of anesthesia has been extensively studied and discussed, the mechanism of pain relief in hypnoanesthesia for minor surgery remains unexplained.[10-14] The originality of this study was to examine the effect of hypnoanesthesia on several neurotransmitters, including beta-

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endorphin, enkephalin, glutamic acid, and substance P which play a role in the mechanism of pain in a comprehensive manner.

## METHOD

This quasi-experimental study applied the randomized control group pretest-posttest Design. The study was conducted at Dr. Moh. Soewandhie Regional Public Hospital (RSUD dr. Moh. Soewandhie), Surabaya. The population in this study included patients who were clinically diagnosed with benign soft tissue tumors treated at the surgical clinic. The subjects for each group amounted to 20 people so the overall size of the study subjects in the two groups amounted to 40 people. This study has received an Ethical Eligibility Letter No.001/KE/KEPK/2020 dated 27 October 2020 from the Clinical Research Ethics Commission.

The inclusion criteria of this study included male or female patients (18-60 years) who were able to communicate in Indonesian (not deaf-mute) and were cooperative with a minimum education level of a six-year elementary school. Surgical excisions were performed for clinically benign tissue tumors located on the ventral part of the patient's body, and surgical excisions in a supine position were performed for benign tumors no more than 5 cm in size. Meanwhile, patients who did not reach the optimal degree of anesthesia when hypnoanesthesia was performed and/or those who experienced abreaction were included in the exclusion criteria.

Prior to the procedures, the patient signed an informed consent form to participate in the study process. Pain scale and vital signs were measured and recorded on the Hypnoanesthesia Research Form. The pain scale (Face Pain Scale) is measured by tweezing the area subjected to surgical procedures and the area around it using a tweezer. Furthermore, blood samples were collected by the laboratory nurse from peripheral blood intravenously to check beta-endorphin, enkephalin, glutamic acid, and substance P levels. Local anesthesia was performed in the control group with 2% lidocaine in the area subjected to surgical procedures until the anesthetic level was achieved. As for the treatment group, hypnoanesthesia was performed (the script is attached) in the area subjected to surgical procedures. An indicator of achieving the required level of hypnoanesthesia was obtained by tweezing the area subjected to surgical procedures and the area around it using a tweezer to find out whether the pain has gone, and see the pain scale (Face Pain Scale) in the patients until it reaches a scale value of 0 (Figure 1). Pain in both groups was monitored with a vital sign monitor. The intervention process was recorded using a camcorder.

The surgical procedures were performed by the operator while maintaining the level of hypnoanesthesia and monitored by a vital sign monitor during the operation until the surgical procedure was completed. Then, after the 10-minute incision, a pain scale and vital sign measurement, as well as a second intravenous blood sampling was carried out. Data on the results of the first and second blood sampling were analyzed using the ELISA test at the Laboratory of Dr. Soetomo Regional Public Hospital. The data collected was processed manually and using SPSS 17. The significance level of the statistical test applied in this study was 0.05.


**Figure 1:** Face pain scale (Wong Baker).

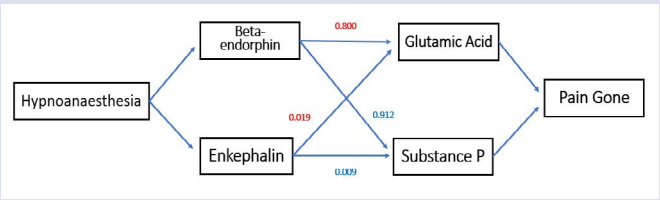


**Figure 2:** Path analysis model.

## RESULTS

Table 1 below presents the characteristics of the subjects in this study.

All the study subjects in the control and treatment groups were under compos mentis conditions during the study.

The results of the Shapiro-Wilk test showed that the difference in glutamic acid level differences of the two groups was normally distributed ($p > 0.05$). So, data differences before and after the treatments were analyzed using a paired t-test, and the difference in glutamic acid level differences of the two groups was analyzed using the two-sample t-test. Meanwhile, the data of the substance P level differences of the two groups were not normally distributed ($p < 0.05$). Thus, data differences before and after the treatments were analyzed using the Wilcoxon test, and the difference in glutamic acid level differences of the two groups was analyzed using the Mann-Whitney test.

The paired t-test indicated no significant difference between beta-endorphin and enkephalin levels before and after the intervention ($p > 0.05$), both in the control and treatment groups.

The results of the two-sample t-test revealed no significant differences between the beta-endorphin, enkephalin, and glutamic acid levels of the two groups ($p > 0.05$), while the results of the Mann-Whitney test showed that substance P levels in the two groups were not significantly different ($p > 0.05$).

Y1 *does not correlate* to Y2 because the p-value is > 0.05 (r = 0.082). Y1 significantly correlates to Y4 (glutamic acid) with a correlation coefficient of 0.990 ($p = 0.000 < 0.01$). Y1 significantly correlates to Y5 (Substance P) with a correlation coefficient of 0.992 ($p < 0.01$). Y2 does not significantly correlate to Y4 because the p-value is 0.679 > 0.05. Y2 does not correlate to Y5 because the p-value is 0.761 > 0.05.

The Model Summary table of the following test presents that the R square reached 0.982, thus the path coefficient ε (variable outside the model) is 0.134 (Table 6).

$$\rho_{Y5\varepsilon} = \sqrt{(1 - 0.982)} = \sqrt{0.018} = 0.134$$

Meanwhile, the coefficient table below shows that the path coefficients of Y1 and Y2 reach 0.800 and 0.019. Only Y1 has a significant correlation ($0.009 < 0.05$). It indicates that only beta-endorphin has a significant correlation with glutamic acid of 0.800.

The Model Summary table of the following test presents that the R square reached 0.984, thus the path coefficient ε (variable outside the model) is 0.126.

$$\rho_{Y5\varepsilon} = \sqrt{(1 - 0.984)} = \sqrt{0.016} = 0.126$$

Meanwhile, table 5.14 depicts that the path coefficients of Y1 and Y2 reach 0.912, -0.009, and 0.081. Only Y1 has a significant correlation ($0.002 < 0.05$).

The correlation between Y1 & Y2 and Y4 is indicated with red correlation numbers with respective magnitudes of 0.800, 0.019, and 0.190. Meanwhile, the correlations between Y1 & Y2 and Y5 is indicated

**Table 1: Characteristics of the subjects.**

| Characteristics | Control (n = 20) | Treatments (n = 20) |
|---|---|---|
| **Age** | | |
| Mean ± Standard Deviation | 30.4 ± 13.220 | 28.15 ± 12.076 |
| **Sex [n(%)]** | | |
| Male | 12 (60%) | 5 (25%) |
| Female | 8 (40%) | 15 (75%) |
| **Education [n(%)]** | | |
| Elementary School | 2 (10%) | 2 (10%) |
| Middle School | 1 (5%) | 3 (15%) |
| High School | 14 (70%) | 14 (70%) |
| Bachelor's degree | 3 (15%) | 1 (5%) |
| **Pain Scale** | | |
| Before | 10 | 10 |
| After | 0 | 0 |
| **Systolic Blood Pressure** | | |
| Before | 137.45 ± 27.810 | 133.60 ± 19.672 |
| After | 132.70 ± 23.622 | 130.75 ± 20.047 |
| **Diastolic Blood Pressure** | | |
| Before | 77.80 ± 15.419 | 71.80 ± 13.320 |
| After | 76.55 ± 14.376 | 69.90 ± 9.597 |
| **Pulse rate (x/minute)** | | |
| Before | 92.15 ± 20.399 | 84.85 ± 16.813 |
| After | 82.95 ± 18.894 | 81.30 ± 15.755 |
| **Respiratory Rate (x/min)** | | |
| Before | 23.35 ± 2.084 | 21.05 ± 3.103 |
| After | 20.60 ± 1.465 | 20.00 ± 1.864 |
| **Conciousnes level before/after** | | |
| CM | 20 (100%) | 20 (100%) |

**Table 2: Normal distribution test.**

| Variables | p-value | |
|---|---|---|
| | Control (n = 20) | Treatment (n = 20) |
| Differences in beta-endorphin levels | 0.967 | 0.052 |
| Differences in Enkephalin levels | 0.814 | 0.722 |
| Differences in glutamic acid levels | 0.068 | 0.537 |
| Differences in Substance P levels | 0.031 | 0.023 |

**Table 3: Differences in beta-endorphin and enkephalin levels before and after the intervention.**

| Groups | | | n | Mean ± Standard deviation | Mean ± Standard Deviation of the Differences | p-value |
|---|---|---|---|---|---|---|
| Beta-Endorfin | Control | Before | 20 | 194.84 ± 105.025 | -1.50 ± 24.121 | 0.784 |
| | | After | 20 | 193.34 ± 100.269 | | |
| | Treatments | Before | 20 | 187.42 ± 249.439 | 0.86 ± 23.944 | 0.874 |
| | | After | 20 | 188.28 ± 262.420 | | |
| Enkephalin | Control | Before | 20 | 14.66 ± 1.887 | -0.30 ± 0.858 | 0.141 |
| | | After | 20 | 14.36 ± 1.928 | | |
| | Treatments | Before | 20 | 13.36 ± 2.005 | 0.11 ± 1.173 | 0.680 |
| | | After | 20 | 13.47 ± 1.647 | | |

with blue correlation numbers with respective magnitudes of 0.912, -0.009, and 0.081 (Figure 2).

The correlation between beta-endorphin & enkephalin and glutamic acid is indicated with red correlation numbers with respective magnitudes of 0.800, 0.019, and 0.190. Meanwhile, the correlations between beta-endorphin & enkephalin and substance P is indicated with blue correlation numbers with respective magnitudes of 0.912, -0.009, and 0.081.

## DISCUSSION

The results of this study and the statistical tests in this study indicated no significant difference in beta-endorphin and enkephalin changes as inhibitory neurotransmitters ($p > 0.05$) as well as glutamate acid and substance P as excitatory neurotransmitters ($p > 0.05$) before and after the hypnoanesthesia intervention (Table 2). There was no statistically significant difference, but it turns out that pain relief can occur empirically. During the minor surgery, all study subjects in the treatment group did not feel pain after being given hypnoanesthesia (the data sourced from the video recordings of this study).

The mechanism of pain in the control group is the inhibition of the transduction process in minor surgical procedures by the intervention of local anesthetic administration with 2% lidocaine, thereby the pain is not passed on to the transmission, modulation, and perception processes so that the pain is not felt.[15,16] It is consistent with the results of the t-test in the control group proving that the control and treatment groups have the same end result, namely pain relief. It means that hypnoanesthesia can replace local anesthesia in situations and conditions where there are contraindications to local anesthetic drugs.

The results of the path analysis suggest that the whole hypnoanaesthesia process to produce pain relief cannot be performed because there are

**Table 4: Change differences in beta endorphin, enkephalin, glutamic acid, and substance P levels in the two groups.**

| Variables | Groups | n | Mean ± Standard Deviation Median (min–max) | p-value |
|---|---|---|---|---|
| Beta-endorfin | Control | 20 | -1.50 ± 24.121 | 0.758 |
| | Treatments | 20 | 0.86 ± 23.944 | |
| Enkephalin | Control | 20 | -0.30 ± 0.858 | 0.220 |
| | Treatments | 20 | 0.11 ± 1.173 | |
| Glutamic Acid | Control | 20 | 0.04 ± 0.160 | 0.937 |
| | Treatments | 20 | 0.035 ± 0.230 | |
| Substance P | Control | 20 | -8.7 (-56,3–113.9) | 0.507 |
| | Treatments | 20 | 0.05 (-135–156) | |

**Table 5: Correlation between X1, Y1, Y2, Y4, Y5, and Y6.**

| | | X1 | Y1 | Y2 | Y4 | Y5 | Y6 |
|---|---|---|---|---|---|---|---|
| **X1** | Pearson Correlation | [a] | [a] | [a] | [a] | [a] | [a] |
| | Sig. (2-tailed) | | | | | | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |
| **Y1** | Pearson Correlation | [a] | 1 | 0.082 | 0.99** | 0.992** | [a] |
| | Sig. (2-tailed) | | | 0.731 | 0.000 | 0.000 | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |
| **Y2** | Pearson Correlation | [a] | 0.082 | 1 | 0.099 | 0.072 | [a] |
| | Sig. (2-tailed) | | 0.731 | | 0.679 | 0.761 | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |
| **Y4** | Pearson Correlation | [a] | 0.99 | 0.099 | 1 | 0.994 | [a] |
| | Sig. (2-tailed) | | 0.000 | 0.679 | | 0.000 | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |
| **Y5** | Pearson Correlation | [a] | 0.992** | 0.072 | 0.994** | 1 | [a] |
| | Sig. (2-tailed) | | 0.000 | 0.761 | 0.000 | | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |
| **Y6** | Pearson Correlation | [a] | [a] | [a] | [a] | [a] | [a] |
| | Sig. (2-tailed) | | | | | | |
| | N | 20 | 20 | 20 | 20 | 20 | 20 |

Description: **: Correlation is significant at the 0.01 level (2-tailed); a: Cannot be computed because at least one of the variables is constant; Y1: Beta Endorphin, Y2: Enkephalin, Y4: Glutamic Acid, Y5: Substance P, Y6: Pain relief.

**Table 6: Regression test of Y1 and Y2 against Y4.**

| Model | R | R Square | Adjusted R Square | Standard Deviation Error of the Estimate |
|---|---|---|---|---|
| 1 | 0.991[a] | 0.982 | 0.978 | 0.4067 |

Description: a. Predictors: (Constant), Y1, Y2.

**Table 7: ANOVA test of Y1 and Y2 against Y4.**

| Model | | Unstandardized Coefficients | | Standardized Coefficients | t | Sig. |
|---|---|---|---|---|---|---|
| | | B | Standard Deviation Error | | | |
| 1 | (Constant) | -0.588 | 0.777 | | -0.757 | 0.460 |
| | Y1 | 0.008 | 0.003 | 0.8 | 2.955 | 0.009 |
| | Y2 | 0.031 | 0.057 | 0.019 | 0.545 | 0.593 |

**Table 8: Regression test of Y1 and Y2 against Y5.**

| Model | R | R Square | Adjusted R Square | Standard Deviation Error of the Estimate |
|---|---|---|---|---|
| 1 | 0.992[a] | 0.984 | 0.981 | 73.4711 |

Description: a. Predictors: (Constant), Y1, Y2.

**Table 9: ANOVA Test of Y1 and Y2 against Y5.**

| Model | | Unstandardized Coefficients | | Standardized Coefficients | t | Sig. |
|---|---|---|---|---|---|---|
| | | B | Standard Deviation Error | Beta | | |
| | (Constant) | 54.243 | 140.427 | | 0.386 | 0.704 |
| 1 | Y1 | 1.862 | 0.512 | 0.912 | 3.634 | 0.002 |
| | Y2 | -2.785 | 10.279 | -0.009 | -0.271 | 0.790 |

constant numbers in hypnoanaesthesia and pain relief. However, the analysis can still be carried out on the intervening variables, namely beta endorphin, enkephalin, glutamic acid, and substance P. The results of the regression and ANOVA analysis indicates that beta-endorphin and enkephalin simultaneously had a significant effect on substance P at 98.4% and glutamate acid at 98.2% (Table 8). Thus, the study hypothesis is fulfilled, but not completely. Among the three inhibitory neurotransmitters, only beta-endorphin significantly correlated to substance P (r = 0.912) and glutamate acid with a correlation coefficient of 0.800 (Table 7 and Table 9).[17-19]

The results of the above study are in similar with the Gate Control Theory, which states that ascending pain signals delivered by excitatory neurotransmitters (glutamate acid and substance P) interact with descending signal barriers by inhibitory neurotransmitters (beta-endorphin and enkephalin).[19] Another finding of this study is that beta-endorphin significantly correlates with substance P and glutamate acid. It is inconsistent with the results of a study conducted by Spiegel, stating that there was no correlation between endogenous opioids (including beta-endorphin and enkephalin) and hypnoanesthesia.[20]

## CONCLUSION

Based on the results of the data analysis and discussion in this study, it can be concluded that the empirical nociceptive pain relief occurred in patients who underwent minor surgical procedures with hypoaesthesia, and statistically, there was no significant change in beta-endorphin, enkephalin, glutamate acid, and substance P serum levels before and after hypoaesthesia. However, beta-endorphin and Enkephalin, as inhibitory neurotransmitters, simultaneously have a significant effect on the two excitatory neurotransmitters, namely glutamate acid and substance P on the mechanism of nociceptive pain relief with hypoaesthesia.

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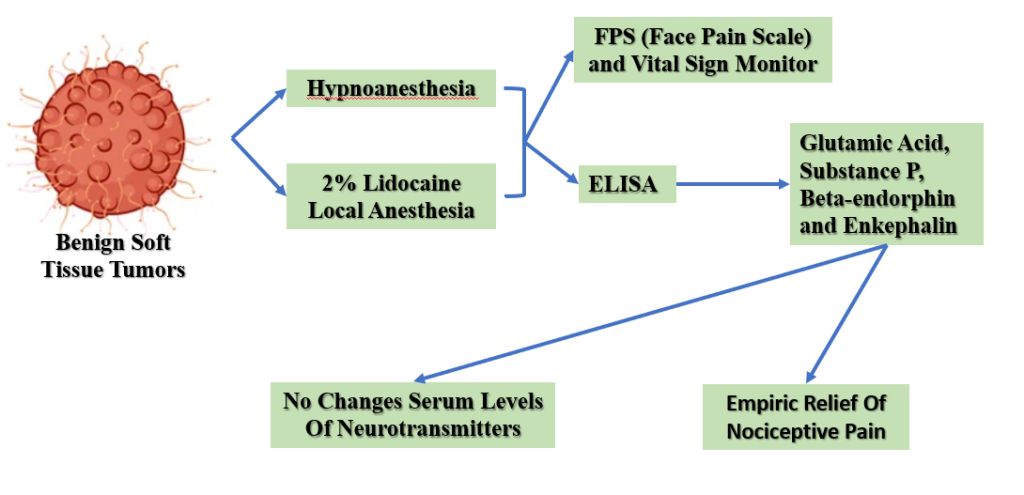
## GRAPHICAL ABSTRACT

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