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Comparison Between Hypnoanesthesia and Local Anesthesia in Minor Surgery

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ABSTRACT

Background: Hypnoanesthesia is a state of anesthesia achieved through hypnosis techniques. Meanwhile, local anesthesia is anesthesia carried out by injecting local anesthetic drugs in or around the surgical site which causes temporary obstruction to the conduction of afferent impulses. Objective: This research was conducted to prove that there is no difference between hypnoanesthesia and local anesthesia in minor surgical procedures, with the indication of pain relief.
Methods: The research subjects were patients with benign soft tissue tumors consisting of 40 people who were divided into 2 groups, namely treatment and control. Minor surgical procedures with hypnoanesthesia were performed in the treatment group, while in the control group, minor surgical procedures were performed with local anesthesia using 2% lidocaine. Pain is measured with FPS (Face Pain Scale) and monitored with a vital sign monitor. The intervention process was recorded with camcorder video. Changes in serum levels of excitatory neurotransmitters (Glutamic Acid and Substance P) and inhibitory (Beta Endorphin, Enkephalin, and Serotonin) before and after intervention were analyzed using ELISA (Enzym-Link Immunosorbent Assay) in both groups.
Results: In the treatment group, it was discovered that patients did not feel pain after undergoing hypnoanesthesia intervention by providing suggestions for the relief of pain in the area where surgery would be performed. In the control group, patients also did not feel pain after local anesthetic intervention in the area to be operated on. However, the results of research and statistical tests showed that there was no significant difference in changes in Beta Endorphins, Enkephalin, and Serotonin as inhibitory neurotransmitters ($p > 0.05$) or Glutamic Acid and Substance P as excitatory neurotransmitters ($p > 0.05$) before and after the intervention in both groups of research subjects.
Conclusion: the results of this study show that there is no significant difference between the treatment and control groups, they have equality in the final result, namely the relief of pain.

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Introduction

Pain relief can occur with hypnoanesthesia for minor surgical procedures. This phenomenon has been known empirically in the medical field since the 18th century. Every surgical procedure, both minor and major, requires anesthesia to relieve pain during the surgical procedure. In minor surgical procedures, local anesthesia using 2% lidocaine is standardly used. The state of anesthesia achieved through hypnosis techniques is called hypnoanesthesia. Hypnoanesthesia was first used in the medical field by James Esdaille, a Scottish surgeon, for surgical procedures, both minor and major. The hypnoanesthesia process does not require anesthetic drugs so the negative impact of anesthetic drugs can be eliminated.[1,2] The mechanism for the anesthesia process in the area undergoing surgical procedures in hypnoanesthesia is due to the obstruction of nociceptive pain impulses which are transmitted to the brain, however, the scientific explanation of the mechanism of obstruction of the transmission of nociceptive pain impulses is still unclear and requires further research.[2–7]

In 1957, hypnoanesthesia was used for the first time in obstetrics and gynecology during section and hysterectomy procedures at Chicago Lying-in Hospital.[3] In the same year in the field of Dentistry, the Michigan State Board of Dentistry recognized the use of hypnosis in dental practice as legal.[8] In the period between April 1994 and June 1999, the United States National Institute of Health reported 197 thyroidectomy surgical procedures and 21 cervical exploration procedures for hyperparathyroidism using hypnoanesthesia.[9] This study examines the differences between hypnoanesthesia and local anesthesia, regarding neurotransmitters that play a role in pain mechanisms comprehensively, both excitatory neurotransmitters, Glutamic Acid and Substance P, and inhibitory ones, Beta Endorphins, Enkephalin, and Serotonin.

Hypnoanesthesia is preceded by changes in pain perception. Suggestions given in the form of visual, sound, and/or tactile stimuli, penetrate the Reticular Activating System in the Reticular Formation in the Brain Stem and are received by the Dorsolateral Prefrontal Cortex as a stimulus which then undergoes a process of selection, interpretation until it becomes a new perception from the perception of pain to the perception of pain relief.[10] The Reticular Activating System in a state of relaxation becomes inactive so that the suggestions given are not criticized or analyzed.[3,11] This new perception is stored in the Ventromedial Prefrontal Cortex as short-term memory and can be marked using an “anchor” as a “password” to be used again when needed.[12,13]

In local anesthesia, local anesthetic drugs work by blocking sodium channels, thereby preventing the entry of sodium into nerve cells. The inhibition of sodium causes no depolarization to occur so that no action potential can be initiated or continued. Depolarization barriers cause the flow of impulses through these nerves to stop, so that all kinds of stimuli or sensations do not reach the central nervous system. The above process shows that local anesthesia will inhibit excitatory neurotransmitters, both glutamate and Substation P so that the pain relief.[14]

Methods

This research is a Quasi-Experimental study with a Randomized Control Group pre-test and post-test design. The research was conducted at Dr. Mohamad Soewandhie Regional General Hospital, Surabaya, Indonesia. The population in this study were patients who were clinically diagnosed with benign soft tissue tumors and patients who required an incision biopsy without inflammation who came for treatment at the surgical clinic. The number of research subjects for each group was 20 people, consisting of a treatment group and a control group, so there was a total of 40 people. This research has received a Letter of Clinical Research Ethics Eligibility No.001/KE/KEPK/2020 dated 27 October 2020.

Inclusion criteria are male or female patients (16-18 years), able to communicate in Indonesian (not deaf-mute), and cooperative with a minimum education level of Elementary School. Surgical action (excision) is carried out on clinically benign soft tissue tumors without any signs of inflammation (calor, dolor, rubor, functional laesa) and the location of the tumor is in the ventral part of the patient's body and surgery can be performed in the supine position with a benign tumor size of no more than 5 cm. Patients who do not achieve optimal levels of anesthesia when hypnoanesthesia is performed and/or patients who experience abreaction are included in the exclusion criteria.

Patients signed an informed consent form to participate in the research process. The pain scale and vital signs were measured and recorded on the research form. The pain scale (Face Pain Scale) is determined by clamping using tweezers on the area where the surgery will be performed and its surroundings. Next, blood samples are taken from peripheral blood intravenously, to check Beta Endorphins, Enkephalins, Glutamic Acid, Substance P, and Serotonin by the nurse. Local anesthesia was performed in the control group with 2% lidocaine in the area to be operated on until the level of anesthesia was achieved. Meanwhile, the treatment group underwent hypnoanesthesia in the area where surgery would be performed. The indicator of achieving the level of hypnoanesthesia is clamping using tweezers on the area where the surgical procedure will be carried out and its surroundings to determine whether the pain has been relieved and looking at the patient's face pain scale (Face Pain Scale) until it reaches a scale value of 0. The research process in both groups was monitored using a vital sign monitor and recorded using a camcorder.

The surgical procedure is carried out by the operator while maintaining the level of anesthesia until the surgical procedure is completed. Then the pain scale, vital signs, and a second blood sample were taken 10 minutes after the incision was made. Data from the results of the first and second blood draws were analyzed using ELISA examination at the Laboratory of Dr. Soetomo General Academic Hospital. The data collected was processed manually using SPSS 17 software. The level of significance of the statistical test used in this research was 0.05.

Results

Table 1. Characteristics of Research Subjects

Characteristics	Control (n = 20)	Treatment (n = 20)
Age		
Mean ± SD	30,4 ± 13,220	28,15±2,076
Sex [n(%)]		
Male	12 (60%)	5 (25%)
Female	8 (40%)	15 (75%)
Last Education [n(%)]		
Elementary	2 (10%)	2 (10%)
Junior high school	1 (5%)	3 (15%)
Senior high school	14 (70%)	14 (70%)
Bachelor	3 (15%)	1 (5%)
Pain scale		
Before	10	10
After	0	0
Sistole Blood Pressure		
Before	137,45±27,810	133,60±9,672
After	132,70±23,622	130,75±20,047
Diastole Blood Pressure		
Before	77,80±15,419	71,80 ± 13,320
After	76,55±14,376	69,90 ± 9,597
Heart rate (x/minute)		
Before	92,15±20,399	84,85 ± 16,813
After	82,9 ± 18,894	81,30 ± 15,755
Respiratory rate (x/minute)		
Before	23,35 ± 2,084	21,05 ± 3,103
After	20,60 ± 1,465	20,00 ± 1,864
Awareness (before/after)		
Compos mentis	20 (100%)	20 (100%)

All research subjects in the control group and treatment group during the research were in composmentis conditions.

Table 2. Differences in Changes in Beta Endorphins, Enkephalins, Serotonin, Glutamic Acid and Substance P Between Groups

Variable	**Groups**	**n**	**Mean ± SD** **Median (min-max)**	**P value**
Beta Endorphins	Control	20	-1,50 ± 24,121	0,758
	Treatment	20	0,86 ± 23,944	
Enkephalins	Control	20	-0,30 ± 0,858	0,220
	Treatment	20	0,11 ± 1,173	
Serotonin	Control	20	4,06 ± 14,990	0,799
	Treatment	20	2,48 ± 23,136	
Glutamic Acid	Control	20	0,04 ± 0,160	0,937
	Treatment	20	0,035 ± 0,230	
Substance P	Control	20	-8,7 (-56,3 – 113,9)	0,507

Treatment	20	0,05 (-135 – 156)

The results of the 2 independent sample t-tests showed that there were no significant differences in Beta-Endorphins, Enkephalin, Serotonin, and Glutamic Acid between groups ($p > 0.05$), while the results of the Mann-Whitney test showed that Substance P between groups was not significantly different ($p > 0.05$).

Treatment Group Analysis (Hypnoanaesthesia)

Based on Table 3, the Pearson correlation shows that variable Y1 is not correlated with Y2 because the p-value is > 0.05 (r = 0.082). Variable Y1 is very strongly correlated with Y3 with a correlation value of 0.992 ($p = 0.000<0.01$). Variable Y1 is very strongly correlated with Y4 (Glutamic acid) with a value of 0.990 ($p = 0.000<0.01$). Variable Y1 has a very strong correlation with Y5 (Substance P) with a correlation coefficient = 0.992 ($p<0.01$). Variable Y2 is not correlated with Y3 because $p = 0.748>0.05$. Variable Y2 is not significantly correlated with Y4 because $p = 0.679>0.05$. Variable Y2 is not correlated with Y5 because $p = 0.761>0.05$. Variable Y3 has a very strong correlation with Y4 with a correlation coefficient of 0.985 ($p<0.01$). Variable Y3 is very strongly correlated with Y5 (r = 0.985) with $p<0.01$. Variables Y4 and Y5 have a very strong correlation with a correlation coefficient of 0.994 ($p<0.01$).

Table 3. Correlation Between X1, Y1, Y2, Y4, Y5 and Y6

		Correlation						
		X1	Y1	Y2	Y3	Y4	Y5	Y6
X1	Pearson Correlation	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]
	Sig. (2-tailed)	0	0	0	0	0	0	0
	N	20	20	20	20	20	20	20
Y1	Pearson Correlation	.[a]	1	.082	.992**	.990**	.992**	.[a]
	Sig. (2-tailed)	.	.	.731	.000	.000	.000	.
	N	20	20	20	20	20	20	20
Y2	Pearson Correlation	.[a]	.082	1	.077	.099	.072	.[a]
	Sig. (2-tailed)	.	.073		.748	.679	.761	.
	N	20	20	20	20	20	20	20
Y3	Pearson Correlation	.[a]	.992**	.077	1	.985**	.985**	.[a]
	Sig. (2-tailed)	.	.000	.748		.000	.000	.
	N	20	20	20	20	20	20	20
Y4	Pearson Correlation	.[a]	.990**	.099	.985**	1	.994**	.[a]
	Sig. (2-tailed)	.	.000	.679	.000		.000	.
	N	20	20	20	20	20	20	20
Y5	Pearson Correlation	.[a]	.992**	.072	.985	.994	1	.[a]
	Sig. (2-tailed)	.	.000	.761	.000	.000		.
	N	20	20	20	20	20	20	20
Y6	Pearson Correlation	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]
	Sig. (2-tailed)	.	.	.	.	.	.	
	N	20	20	20	20	20	20	20

**. Correlation is significant at the 0.01 level (2-tailed)

a. Cannot be calculated because at least one of the variables is constant

Notes:

X 1: Hypnoanesthesia

Y 1: Beta-endorphin

Y 2: Enkephalin

Y 3: Serotonin

Y 4: Glutamic acid

Y 5: Substance P

Y 6: Pain relief

Path Analysis between Y1, Y2, Y3 and Y4

In the Summary Model of the path regression test Y1, Y2, and Y3 against Y4, it was found that the R square was 0.982, thus the path coefficient ε (variable outside the model) was 0.134.

$$\rho_{Y5\varepsilon} = \sqrt{(1 - 0{,}982)} = \sqrt{0{,}018} = 0.134$$

Meanwhile, in the coefficients in the Anova test, it was found that the path coefficients Y1, Y2, and Y3 were 0.800, 0.019, and 0.190. And the only significant correlation is Y1 (0.009 < 0.05). This shows that only Beta-endorphin has a significant correlation with Glutamic acid of 0.800.

Path Analysis between Y1, Y2, Y3 and Y5

In the Summary Model of the path regression test Y1, Y2, and Y3 against Y5, the R square is 0.984, so the path coefficient ε (variable outside the model) is 0.126. Meanwhile, in Figure 1 below, it can be seen that the path coefficients Y1, Y2, and Y3 are 0.912, -0.009, and 0.081. And the only significant correlation is Y1 (0.002 < 0.05). The correlation from Y1, Y2, and Y3 to Y4 is shown in red correlation numbers with magnitudes of 0.800, 0.019, and 0.190 respectively. Meanwhile, the correlation of Y1, Y2, and Y3 with Y5 is shown in the blue correlation figures with respectively 0.912, -0.009, and 0.081 (Figure 1).

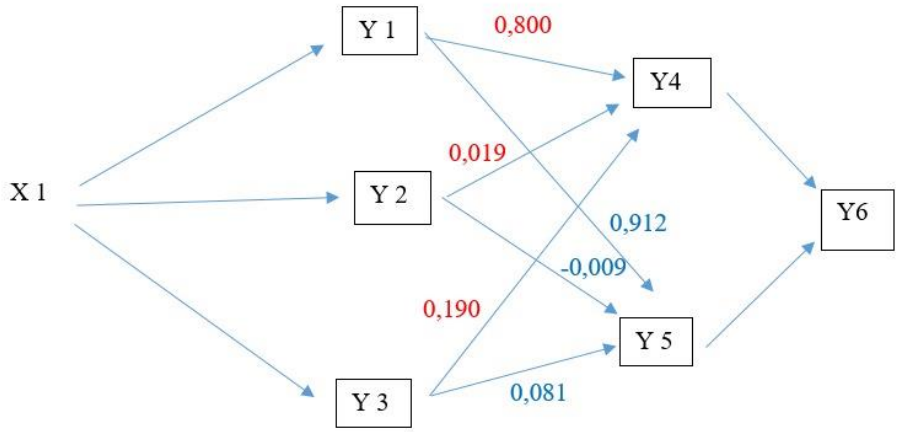

Figure 1. Treatment Group Path Analysis Model

In Figure 1, X1 will suppress Y4 and Y5 which are excitatory neurotransmitters so that painful stimuli are not transmitted to the brain or do not feel pain (Y6). If Y4 and Y5 are suppressed, Y1, Y2, and Y3 as inhibitory neurotransmitters are also suppressed or not released in the brain's nervous system. Y1, Y2, and Y3, when stimulated, will inhibit pain so that the pain relief.

Control Group Analysis Results

The control group in this study had their data analyzed to see differences in the results of each variable after administering local anesthetic drugs to the research subjects.

Table 4: Correlation of X1, Y1, Y2, Y3, Y4, Y5, and Y6

		Correlation						
		X1	Y1	Y2	Y3	Y4	Y5	Y6
X1	Pearson Correlation	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]
	Sig. (2-tailed)		.	.	.	.	.	.
	N	20	20	20	20	20	20	20
Y1	Pearson Correlation	.[a]	1	-.316	.803**	.879**	.762**	.[a]
	Sig. (2-tailed)	.	.	.175	.000	.000	.000	.
	N	20	20	20	20	20	20	20
Y2	Pearson Correlation	.[a]	-.316	1	-.189	-.076	-.017	.[a]
	Sig. (2-tailed)	.	.175		.424	.749	.944	.
	N	20	20	20	20	20	20	20
Y3	Pearson Correlation	.[a]	.803**	-.189	1	.800**	.842**	.[a]
	Sig. (2-tailed)	.	.000	.424		.000	.000	.
	N	20	20	20	20	20	20	20
Y4	Pearson Correlation	.[a]	.879**	-.076	.800**	1	.810**	.[a]
	Sig. (2-tailed)	.	.000	.749	.000		.000	.
	N	20	20	20	20	20	20	20
Y5	Pearson Correlation	.[a]	.762**	-.017	.842	.810	1	.[a]
	Sig. (2-tailed)	.	.000	.944	.000	.000		.
	N	20	20	20	20	20	20	20
Y6	Pearson Correlation	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]	.[a]
	Sig. (2-tailed)	.	.	.	.	.	.	
	N	20	20	20	20	20	20	20

**. Correlation is significant at the 0.01 level (2-tailed)
a. Cannot be calculated because at least one of the variables is constant

Variable Y1 (Beta-endorphin) has a strong correlation with Y3 (Serotonin) of 0.803, Y4 (Glutamic Acid) of 0.879, and Y5 (Substance P) of 0.762. Meanwhile, Y1 is not strongly correlated with Y2 (Enkephalin), with a negative correlation direction. This negative correlation direction also occurs between Y2 and Y3, Y4 and Y5; If Enkephalin increases, Serotonin, Glutamic Acid and Substance P will decrease, and vice versa. Variable Y3 (Serotonin) has a strong correlation with Y4 (Glutamic Acid) and Y5 (Substance P), respectively 0.800 and 0.842.

Path Analysis between Y4 and Y5 towards Y1

It can be seen from the results of the coefficients in the Anova test for paths Y4 and Y5 that they are 0.763 and 0.143. The only significant correlation is Y4 (0.001 < 0.05), whereas in the Model Summary table, it can be seen that the R square is 0.754, so the path coefficient ε (variable outside the model) is 0.495.

$$\rho_{Y5\varepsilon} = \sqrt{(1 - 0.754)} = \sqrt{0.246} = 0.495$$

This means that the proposed hypothesis is not completely accepted because based on testing, only the path coefficient from Y4 to Y1 is statistically significant. This shows that the only influence on Y1 is Y4.

Path Analysis between Y4 and Y5 towards Y2

It can be seen from the Anova test coefficients that the path coefficients Y4, and Y5 are -0.183 and 0.132. There is no significant correlation (0.662 > 0.05 and 0.753 > 0.05), whereas in the Model Summary table, it can be seen that the R square is -

0.104, so the path coefficient ε (variable outside the model) is 0.947.

$\rho_{Y5\varepsilon} = \sqrt{(1 - (-0.104))} = \sqrt{0.896} = 0.947$

This means that none of the proposed hypotheses is accepted because based on testing, none of the path coefficients from Y4 and Y5 to Y2 are meaningful.

Path Analysis between Y4 and Y5 to Y3

It can be seen from the Anova test coefficients that the path coefficients Y4, and Y5 are 0.341 and 0.566. The only significant correlation is Y5 (0.014 < 0.05), whereas in the Model Summary table, it can be seen that the R square is 0.720, so the path coefficient ε (variable outside the model) is 0.529.

$\rho_{Y5\varepsilon} = \sqrt{(1 - 0.720)} = \sqrt{0.28} = 0.529$

This means that the proposed hypothesis is not completely accepted because based on testing, only the path coefficient from Y5 to Y3 is statistically significant. This shows that the only influence on Y3 is Y5.

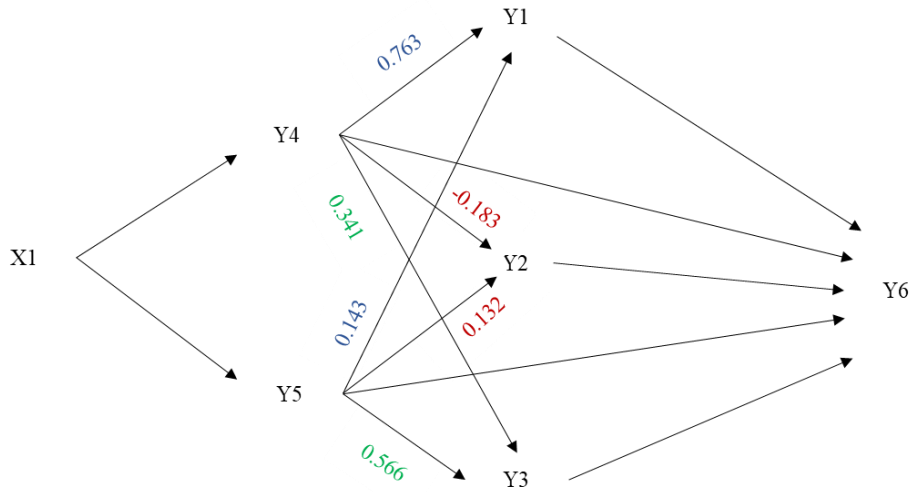

Figure 2. Control Group Analysis Model (X1: Local anesthetic; Y1: Beta Endorphin; Y2: Enkephalin; Y3: Serotonin; Y4: Glutamic Acid; Y5: Substance P; Y6: Pain relief)

In figure 2, X1 will suppress Y4 and Y5 which are excitatory neurotransmitters so that painful stimuli are not transmitted to the brain or do not feel pain (Y6). If Y4 and Y5 are suppressed, Y1, Y2, and Y3 as inhibitory neurotransmitters are also suppressed or not released in the brain's nervous system. Y1, Y2, and Y3, when stimulated, will inhibit pain so that the pain disappears.

Discussion

In the treatment group, it was discovered that patients did not feel pain after undergoing hypnoanesthesia intervention by providing suggestions for the relief of pain in the area where surgery would be carried out. Suggestions for the relief of pain or suggestions for numbness given through visual, sound, and/or touch stimuli are received by the sensory organs and then forwarded to the Thalamus and after breaking through the Reticular Activating System (RAS) alert system in the Formation Reticularis, it is passed on to the Dorso Lateral Prefrontal Cortex as a new perception. , namely pain-free feeling.[10] This pain-free perception is stored in the Ventro Medial Prefrontal Cortex.

However, the results of research and statistical tests showed that there were no significant differences in changes in Beta Endorphins, Enkephalin, and Serotonin as inhibitory neurotransmitters ($p > 0.05$) or Glutamic Acid and Substance P as excitatory neurotransmitters ($p > 0.05$) before and after hypnoanesthesia intervention. There is no statistically significant difference. It turns out that there can be an empirical relief of pain. At the time of the minor surgical procedure, all research subjects in the treatment group did not feel pain after being given hypnoanesthesia (data source in research video recording).

In this study, the mechanism of pain in the control group occurred in the peripheral nervous system, the transduction process during minor surgical procedures was inhibited by the intervention of local anesthesia with 2% lidocaine so that pain was not passed on to the transmission, modulation and perception processes so that pain was not felt.[15,16] This is in accordance with the results of the t-test in the control group; proving that the control and treatment groups have equality in the final result, namely pain relief.

Based on the results of statistical analysis of the treatment group, it was found that Beta Endorphin, Enkephalin, and Serotonin simultaneously had a significant effect on Substance P by 98.4% and Glutamic Acid by 98.2%. Of the three inhibitory neurotransmitters, the results obtained were that only Beta Endorphin had a strong significant correlation with Substance P ($r = 0.912$) and Glutamic Acid of 0.800.[17–19] These results are in accordance with Gate Control Theory[19] which states that ascending pain signals delivered by excitatory neurotransmitters (Glutamic Acid and Substance P) interact with the inhibition of descending signals by inhibitory neurotransmitters (Beta Endorphin, Enkephalin, Serotonin).

Conclusion

Based on the results of data analysis and discussion in this study, it can be concluded that there is no significant difference between hypnoanesthesia which mechanism is in the central nervous system, and local anesthesia in the peripheral nervous system in minor surgical procedures in both the control and treatment groups, wherein the control and treatment groups have equality in the final result, namely pain relief.

Conflict of Interest

The authors declare no potential conflicts of interest or competing interests. The authors received no financial assistance or grants from public, private, or non-profit funding agencies.

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