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## 18 Mar-P069

### EVALUATION OF ADOPTED WEIGHT DROP DEVICE TO INDUCE CONTUSIVE SPINAL CORD INJURY IN RATS: BEHAVIORAL AND HISTOPATHOLOGICAL STUDIES

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Recently, many experimental devices have been designed to construct standardized animal spinal cord injury (SCI) models, because electromagnetic SCI devices are expensive. To evaluate adopted weight drop device inducing contusive SCI, the fixed weight was used and dropped down from varied heights, then followed by behavioral and histopathological studies. Fifteen adult male Sprague Dawley rats were divided into laminectomy (L), moderate injury (MI) and severe injury (SI) groups. The C5 hemicontusion injury was performed and resulted in the right side hemiplegia and forepaw deficits. Both MI and SI rats showed the clubbing forepaw at 24 h after injury. The skilled locomotion using the horizontal ladder test was analyzed. The SI showed a significant increase in error scores, percentage of total rungs used and decrease in percentage of correct placement when compared to L group, $p<0.05$. The normal recovered placement (type II) was shown at day 7 after injury but higher numbers in MI than SI group. The somatosensory function using sticker removal test was also analyzed. The SI group showed a significant somotosensory deficit at day 3, 7 when compared to L group, $p<0.05$. Behavioral deficits were related to histopathological study using H&E counterstained with luxol fast blue staining, the higher degree of injury, the larger area of lesion. The lesion was mostly in lateral funiculus related to rubrospinal and lateral corticospinal tract involving skilled movement of forepaw. Results indicate that this rat SCI model is more suitable, simple, reliable and it should be induced as moderate injury for allowing significant recovery of function.

**Keywords**: Hemicontusion injury, Spinal cord injury

## 18 Mar-P070

### HISTOPATHOLOGICAL APPEARANCE OF THE BRAIN DUE TO HEAD INJURY AND KETAMINE INTOXICATION IN *RATTUS NORVEGICUS*

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The brain, comprised of the cortex, cerebellum and brain stemn is the control board for all functions of the body. Traumatic or non-traumatic brain injury lead to impairments in physical, cognitive, speech or language and behavioral functioning. Traumatic brain injury are much more common due to head injuries, whereas non-traumatic injury is an injury occur as a result of stroke, tumours, infectious diseases, lack of oxygen or toxicity. Ketamin has used as an anesthetic, but it's as a recreational use in some countries. Overdose that results in a loss of consciousness or a lack of oxygen to the brain can lead to permanent brain damage.

To determine the effects of blunt head injury and ketamine intoxication to the brain tissue damage, we studied the hystological appearance of male Rattus Norvegicus brain after getting head injury and ketamin injection. Two groups of eight rats each, were head injury and the other two groups were treated with lethal dose ketamine intraperitoneal. The brain tissue were prepare for hystopathological examination directly after treatmen and four after treatment to asess hemmorhage necrosis, congestion and inflammation. This study was recomended by Research Ethic Committee of Andalas University.

The histopathological appearance of brain tissue in groups with head injury and ketamine intoxication shows a hemorrhage and congestion. Its appear that neuron cells become hypercromatic and perivasculer space were edema mainly in gray matte. The hemorrhage and congestion in groups with head injury was higher than ketamine intoxication. The location of hemorrhage in group with head injury were in subarachnoid and parenchym, with erythrocyte in the tissue, whereas in group with ketamine intoxications, the haemorhage were on intracerebral and subarachnoid. This is due to head injury causes direct trauma to the brains blood vessels, whereas ketamine poisoning causes increased CO2 pressure in the brains blood vessels. Autolysis, necrosis and inflammation was not seen in the brain tissue of all groups, because the tissue were taken immediately and three hours after treatment.

From study, we conclude that head injury and ketamine intoxication causes brain injury in different way

Keywords: brain injury, head injury, ketamine

# Histopathological appearance of the brain due to head injury and ketamine intoxication in *rattus norvegicus*

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## Background

The brain, comprised of the cortex, cerebellum and brain stemn is the control board for all functions of the body. Traumatic or non traumatic brain injury lead to impairments in physical, cognitive, speech or language and behavioral functioning. A traumatic brain injury are much more common due to head injuries, whereas non-traumatic injury is an injury occur as a result of stroke, tumours, infectious diseases, lack of oxygen or toxicity. Ketamin has used as an anesthetic, but its as a recreational use in some countries. Overdose that results in a loss of consciousness or a lack of oxygen to the brain can lead to permanent brain damage.

## Aim of Study

To determinethe effects ofbluntheadinjury andketamine intoxication to thebrain tissue damage

## Methods

Thirty two of male Rattus Norvegicus were divided into four groups, two groups were head injury, and the other two groups were treated with lethal dose ketamine. The brain tissue were prepared for histopathological examination directly after treatment and after death to assess hemmorrhage, congestion, necrosis and inflammation. The appearance is calculated using scores. This study was recommended by Research Ethic Committee of Andalas University.

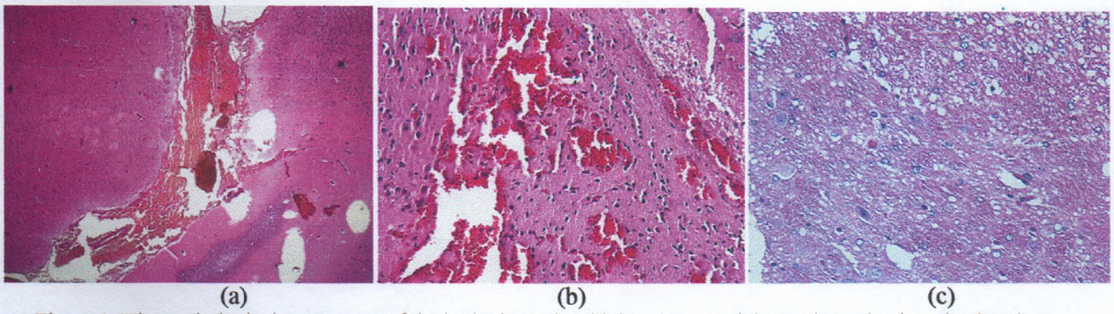
## Results:

The histopathological appearance of brain tissue:

- Groups with head injury shows that a hemorrhage and congestion was higher thanketamineintoxication. The location of hemorrhage were in subarachnoidand parenchym, with erythrocyte in the tissue
- Groups with ketamineintoxication shows the location of hemorrhageandcongestionwere intracerebraland subarachnoid
- Neuron cells become hypercromatic and perivasculer space were edema mainly in gray mate
- No autolysis, necrosis andinflammationseen inthe brain tissueofall groups, because the tissue were taken before inflammatory process occurs.
- Differences in locationhemorrhage is dueto head injurycauses direct trauma to the brains blood vessels, whereasketaminepoisoningcauses increasedCO2pressurein the brainsblood vessels

Table: Brain histopathological appearance in heaad injury groups and ketamin intoxication groups

| Groups | Hemorrhage scores | Congesti scores | Inflammation | Necrosis |
|---|---|---|---|---|
| Head Injury | 1 | 1 | 0 | 0 |
| Ketamin Intoxication | 2 | 2 | 0 | 0 |



(a) (b) (c)

Figure 1: Histopathological appearance of the brain due to head injury (a,), and due to ketamine intoxication (b,c)

## Conclusion

Head injury and ketamine intoxication causes brain damage duehemorrhageandcongestionin different places, in connection with a different mechanism


# Certificate of Attendance

This is to certify that

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