

The course of respiration and circulation in death due to typical hanging

Noriaki Ikeda, Akira Harada, and Tsuneo Suzuki

Department of Forensic Medicine, Yamagata University School of Medicine, Yamagata 990-23, Japan

Received June 3, 1991 / Received in revised form September 2, 1991

Summary. Experiments were carried out on 15 dogs to investigate the course of respiration and circulation during the agonal period of death due to typical hanging. Animals were asphyxiated by a method simulating typical hanging. Electrocardiogram (ECG), electroencephalogram (EEG), blood pressure (BP) in the femoral artery and intrathoracic pressure (ITP) were registered.

In typical hanging, the course of respiration was characterized by shorter stages of dyspnoea and initial apnoea and a longer stage of the terminal respiration when compared with obstructive asphyxia. However, the whole time of the course of respiration and circulation in typical hanging was almost the same as that in obstructive asphyxia. The BP increased rapidly, was maintained during the dyspnoea stage, and then decreased gradually. EEG disappeared with, or a short time after, the end of the dyspnoea stage. The increased heart rate in the dyspnoea stage remained until circulatory collapse. The analysis of the ECG complexes revealed that the heart muscle adapted to oxygen deficiency during typical hanging.

Key words: Typical hanging – Obstructive asphyxia – Obstruction of the neck arteries – Respiration and circulation

Zusammenfassung. An 15 Hunden wurden Experimente durchgeführt, um den Verlauf der Atmung und des Kreislaufs während der Agonie-Phase bei typischem Erhängen zu untersuchen. Die Tiere wurden erstickt mit Hilfe einer Methode, welche das typische Erhängen simulierte. Elektrokardiogramm (EKG), Elektroencephalogramm (EEG), Blutdruck in der Femoralarterie und intrathorakaler Druck wurden registriert.

Bei typischem Erhängen war der Verlauf der Atmung charakterisiert durch kürzere Phasen der Dyspnoe und der initialen Apnoe und eine längere Phase der terminalen Atmung, wenn man diese Parameter mit der obstruktiven Asphyxie vergleicht. Jedoch war der gesamte Zeitverlauf für die Atmung und den Kreislauf bei typischem Erhängen nahezu dieselbe, wie bei obstruktiver Asphyxie. Der Blutdruck stieg rasch an, wurde während der

Dyspnoe-Phase aufrecht erhalten und sank dann graduell ab. Das EEG war entweder am Ende der Dyspnoe-Phase oder kurze Zeit hiernach nicht mehr nachweisbar. Die in der Dyspnoe-Phase angestiegene Herzfrequenz blieb hoch bis zum Kreislaufkollaps. Die Analyse der EKG-Komplexe zeigte, daß der Herzmuskel sich während des typischen Erhängens an den Sauerstoffmangel adaptiert.

Schlüsselwörter: Typisches Erhängen – Obstruktive Asphyxie – Verschuß der Hals-Arterien – Atmung und Kreislauf

Introduction

Hanging is due to constriction of the neck as a result of suspension in such a manner that the weight of the body, or a part of the body of the victim pulls upon the ligature. The neck veins, arteries and the air way may all be obstructed in typical hanging [1, 2].

The course of respiration and circulation during obstructive asphyxia has been described by Brinkmann et al. [3] and Suzuki et al. [4]. However, the pathophysiology of respiration and hemodynamics during the agonal period of death due to typical hanging has not been investigated.

In the present work, experimental animals were asphyxiated by a method simulating typical hanging, and the course of respiration and circulation in death due to typical hanging was investigated and compared to those due to obstructive asphyxia.

Materials and methods

Experiments were carried out on 15 healthy mongrel dogs weighing 8–10 kg. The dogs were anaesthetized with an intravenous administration of sodium pentobarbital 25.0 mg/kg body wt and were placed in a supine position with the legs fastened to a table. The trachea, bilateral common carotid arteries, vagus nerves, internal jugular veins, and cervical vertebral column were exposed. The dogs were asphyxiated by occlusion of the trachea with a ligature

of the bilateral common carotid arteries, vagus nerves and internal jugular veins. Bilateral vertebral arteries were ligated at the same time with the rope running horizontally round the exposed cervical spine at the level between the fourth and the fifth transverse process of the cervical vertebrae, by applying tractive forces in the range between 50–60 kg. To avoid variable results from general anaesthesia, the experiment was begun when the blood pressure (BP), intrathoracic pressure (ITP), electrocardiogram (ECG) and electroencephalogram (EEG) became stable. At that stage, the animals were still unconscious and they did not react to the pain stimulation.

The BP, ITP, ECG and EEG were recorded by the same methods as those described in our previous papers [5, 6]. These recordings were preserved in a data recorder, and the ECG complexes were later analyzed in detail.

Results

The respiration and circulation of the experimental animals during the agonal period of death due to typical hanging were as follows. Just after the ligation of the trachea, arteries, veins and nerves, the dyspnoea stage consisted of large inspiratory movements with a slight

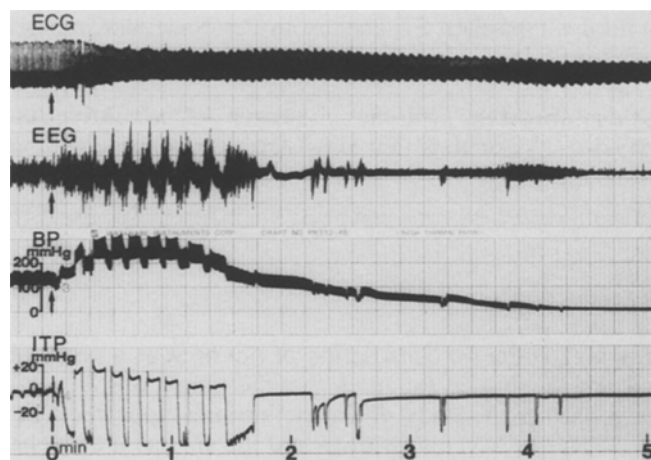


Fig. 1. A record obtained from the dog asphyxiated by a method simulating typical hanging. *ECG*, electrocardiogram; *EEG*, electroencephalogram; *BP*, blood pressure; *ITP*, intrathoracic pressure; arrow (\uparrow); the starting point of the experiment

expiratory component. The respiratory movements increased rapidly to the maximum after 1 or 2 respiratory movements were maintained for 1–1.5 min and ceased after a respiratory movement with prolonged inspiratory phase. The cessation of the respiratory movement (the initial apnoea) lasted for 0.5–1 min before the terminal respirations occurred. These consisted of a number of respiratory movements with sharp inspiratory character, which occurred sporadically lasted for about 2–3 min, and disappeared just before the circulatory breakdown (Fig. 1). In a few animals, the dyspnoea stage consisted of only 2 or 3 large respiratory movements with the prolonged inspiratory character (Fig. 2). The whole time of the course of respiration and circulation in typical hanging was 4–6 min.

The BP increased precipitously after ligation to the maximum of one and a half times the original level which was maintained for about 1–1.5 min till the end of the dyspnoea stage and then decreased gradually. The circulatory breakdown occurred with the end of the terminal respirations. The heart rate increased after 2 or 3 respiratory movements and remained regular until circulatory breakdown (Figs. 1, 2).

The EEG disappeared approximately 1.5–2 min after ligation or a short time after the end of the dyspnoea stage. Convulsive waves appeared during the terminal respiration (Figs. 1, 2).

The analysis of the ECG complexes showed as follows. No distinguishable changes occurred during the dyspnoea stage. During the period of initial apnoea, ST segment elevation and tall upright T waves were seen. The voltage of T waves increased and reached approximately the same level as the R waves at the beginning of the stage of terminal respirations and then decreased gradually. However, the voltage of R waves did not decrease. The P waves did not disappear until the circulatory breakdown (Fig. 3).

Discussion

In the course of respiration during obstructive asphyxia in dogs, the dyspnoea stage immediately followed the occlusion of the trachea, and lasted for 2–3 min. Then

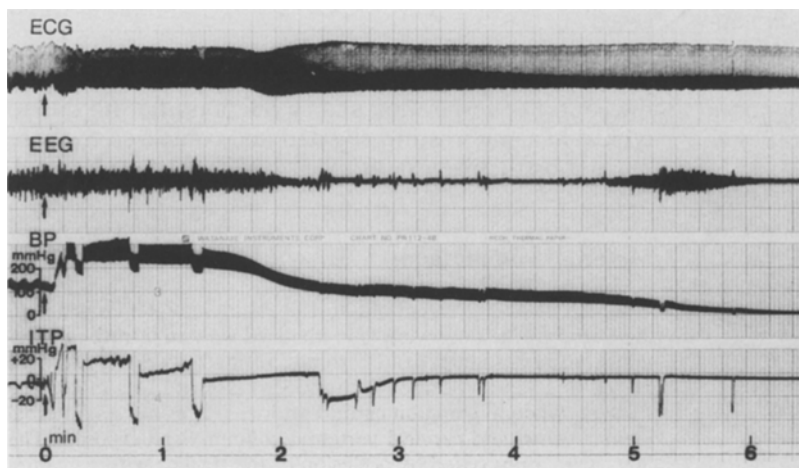


Fig. 2. The dyspnoea stage shows only 3 large respiratory movements with prolonged inspiratory character

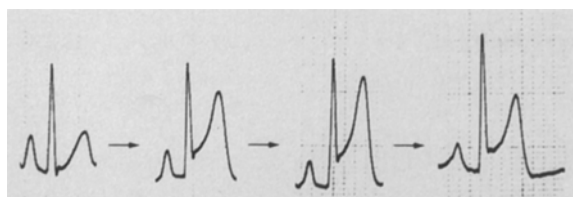


Fig. 3. Typical changes of ECG complexes obtained from the dog asphyxiated by a method simulating typical hanging

the initial apnoea of 1–2 min duration followed. The terminal respirations occurred after the initial apnoea and lasted for 1–2 min [4]. The course of obstructive asphyxia with ligature of the bilateral common carotid arteries, veins and nerves were almost identical to the course of simple obstruction [7]. However, the pathophysiology of respiration and hemodynamics during the agonal period of death due to typical hanging has not been investigated. This was due to difficulties in simulating the typical hanging while monitoring pathophysiological data in the experimental animal.

The vertebral arteries are not obstructed by compression of the horizontal part of the neck because they ascend through the foraminae in the transverse processes of the cervical vertebrae [2]. We could obstruct both vertebral arteries by exposing the vertebral column and ligating it with the rope running horizontally just below the bilateral transverse processes. Using this method, the respiratory movement, ECG, BP and EEG in a condition simulating typical hanging were registered, and thus the pathophysiology during the agonal period of death due to typical hanging was first investigated.

The course of respiration and circulation of typical hanging was different from that of obstructive asphyxia in several respects. The dyspnoea stage and the initial apnoea stage lasted for only 1–1.5 min and 0.5–1 min, respectively, and the stage of terminal respirations was maintained for 2–3 min. In typical hanging, the course of respiration was characterized by shorter stages of dyspnoea and initial apnoea, and a longer stage of terminal respiration when compared to that of obstructive asphyxia. However, the fatal time of the course of respiration and circulation in typical hanging was not different from that of obstructive asphyxia. The respiratory rate during the dyspnoea stage was much lower than that in obstructive asphyxia. The BP increased rapidly with the increase of heart rate by one and a half times the original level and was maintained for 1–1.5 min, while in the obstructive asphyxia, it increased gradually without an increase in heart rate and was maintained for 3–4 min. The EEG lasted only 1.5–2 min, and the time was one half of that in obstructive asphyxia. This suggested that in typical hanging, brain damage occurs more rapidly

than in obstructive asphyxia. These characteristics in the course of respiration and circulation in typical hanging seem to be due to complete and rapid blocking of arterial blood supply to the respiratory and circulatory center of the brain by ligature of the bilateral common carotid and vertebral arteries.

The analysis of ECG complexes showed ST segment elevation and tall upright T waves at the period of dyspnoea. These changes have been reported to occur in the course of different forms of asphyxia [8, 9], and they might be due to the direct effect of oxygen deficiency on the heart muscle. But the sequence of P-QRS-T waves were maintained until circulatory breakdown. This suggests that the heart muscle adapted to oxygen deficiency. In typical hanging, the oxygen level in blood might be somewhat higher than in obstructive asphyxia, because in typical hanging, the blood oxygen cannot be consumed by the brain after ligature of trachea. However, the circulatory breakdown in the present experiment occurred in almost the same time as that in the obstructive asphyxia.

From the findings of the present experiments, it is suggested that in typical hanging, the dyspnoea stage lasts for only half as long as in obstructive asphyxia, and the EEG disappears more rapidly than in obstructive asphyxia, although the total time of the course of respiration and circulation is not different. Therefore, it is considered that brain damage in typical hanging occurs more rapidly and easily than in obstructive asphyxia.

References

1. Polson CJ, Gee DJ, Knight B (1985) The essentials of forensic medicine, 4th edn. Pergamon Press, Oxford, pp 357–388
2. Brinkmann B, Koops E, Wischhusen F, Kleiber M (1981) Halskompression und arterielle Obstruktion. *Z Rechtsmed* 87:59–73
3. Brinkmann B, Püschel K, Bause H-W, Doehn M (1981) Zur Pathophysiologie der Atmung und des Kreislaufs bei Tod durch obstruktive Asphyxie. *Z Rechtsmed* 87:103–116
4. Suzuki T, Ikeda N, Umetsu K, Kashimura S (1986) Zum Ablauf der Atmung bei Tod durch obstruktive Asphyxie. *Z Rechtsmed* 96:105–109
5. Suzuki T, Ikeda N, Umetsu K, Kashimura S (1985) Swimming and loss of consciousness. *Z Rechtsmed* 94:121–126
6. Ikeda N, Takahashi H, Umetsu K, Suzuki T (1989) The course of respiration and circulation in death by carbon dioxide poisoning. *Forensic Sci Int* 41:93–99
7. Suzuki T (1987) The course of respiration and circulation in death due to obstructive asphyxia and drowning. *Jpn J Leg Med* 41:522–535
8. Ikeda N, Takahashi H, Umetsu K, Suzuki T (1990) The course of respiration and circulation in death due to plastic bag suffocation. *Yamagata Med J* 8:15–20
9. Ikeda N, Takahashi H, Umetsu K, Suzuki T (1990) The course of respiration and circulation in toluene-sniffing. *Forensic Sci Int* 44:151–158