Cerebral Artery Blood Flow and Oxygenation in the Frontal Lobe Region in Response to a Judo Chokehold (*Shimewaza*)

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Abstract

Chokeholds (*shimewaza*) are allowed only in judo competitions. They are extremely dangerous and require a high level of technical mastery. However, only a few studies have examined the physiological effects of chokeholds since the Kodokan Judo Institute published the first results in 1958. In the current study, we measured middle cerebral artery blood flow during a chokehold in male members of a university judo club who all had an extensive judo background. Furthermore, we investigated the level of cerebral oxygenation and the blood volume in the frontal lobe of the cerebrum. Middle cerebral artery blood flow was completely cut off when a competitor tapped out just prior to losing consciousness (*ochi*) from a chokehold. When competitors tapped out due to a chokehold, their heart rates were approximately 10 beats/min higher, and their blood pressure increased by about 10–20 mmHg. Oxygenation of the frontal lobe was temporarily decreased prior to tapping out due to a chokehold and tended to slowly return to its previous level after the chokehold was released. In the midst of a chokehold, blood volume initially decreased in half but returned to its previous resting level after release of the chokehold. These findings suggest that the application of a chokehold is extremely dangerous in terms of the hemodynamics of cerebral blood flow even if the individual being choked is barely conscious and able to tap out.

Keywords: Judo chokehold; Cerebral; Blood flow; Oxygenation; Tap out; Passes out

Introduction

Chokeholds (*shimewaza*) are the only traditional grappling techniques still officially permitted in judo. The allowance of chokeholds makes judo unique among the all of the international and national official combative sports. Chokeholds are a set of techniques with which a competitor uses his forearm, wrist, or judo uniform to constrict the common carotid arteries of his opponent. Therefore, judo chokeholds are extremely dangerous techniques. A chokehold is applied until an opponent “passes out (*ochi* = asphyxia)” or “taps out (*maitta* = give up)” by tapping either the body of an opponent or the judo mats (*tatami* mats). Individuals subjected to a chokehold often lose consciousness. Ascertaining the physiological changes when a competitor loses consciousness is crucial to better understanding the technique and to preventing accidents during judo practice and competitions.

In 1958, researchers specifically chosen by the Kodokan Judo Institute reported “studies in general on choking” in which the physiological responses were monitored by using both X-rays of the heart and electroencephalograms (EEGs) to examine the mechanism of chokeholds from a medical perspective [1-3]. Loss of consciousness as a result of a judo chokehold was reported to be due to a combination of oxygen deficit in the cerebrum and the carotid sinus reflex [1-3]. Moreover, the aforementioned studies revealed that the cardiac silhouette shrinks in terms

of both its transverse diameter and area as the chokehold continues. The cardiac silhouette is smallest when the opponent loses consciousness, and it returns to its normal size 10 sec after the opponent regains consciousness [2]. Thus, cardiac volume gradually decreases in the midst of a chokehold, and venous return and cardiac output presumably decrease as well. EEGs with waveforms similar to a very brief epileptic seizure were also noted after a chokehold. In addition to problems with consciousness and seizures, signs of sympathetic nerve excitation were also apparent. These changes were presumably due to the effects of tachycardia and hypertension on the carotid sinus reflex [3].

Rodriguez *et al.* [4] used EEGs and $^{133}\text{Xenon}$ inhalation to examine regional cerebral blood flow in 10 judo practitioners before and after they lost consciousness due to a judo chokehold (*Kata-juji-jime*). They reported that typical delta waves of 3 Hz were produced primarily in the anterior regions of the brain around 10 sec after a chokehold was applied. Afterwards, the EEG gradually returned to normal. During a chokehold, the regional cerebral blood flow (CBF) decreased in all but one subject. The regional CBF decreased slightly in 4 subjects and markedly in 2 subjects [4]. Raschka *et al.* [5] used Doppler ultrasound to examine changes in the CBF due to judo cross-choke techniques (*Juji-jime*). They noted a significant decrease in end-diastolic blood flow in the middle cerebral artery.

Jöbsis [6] first used near-infrared spectroscopy to monitor the oxygen supply to the cerebrum. Later, Chance *et al.* [7-9] and Hamaoka *et al.* [10] used near-infrared continuous-wave spectroscopy (NIRcws) as a novel non-invasive technique to examine changes in oxygenation and in hemodynamics in skeletal muscle during human exercise. Although numerous studies of CBF have been conducted [11-14], none have used NIRcws to examine the level of cerebral oxygenation and blood volume to regions of the cerebrum in response to a judo chokehold.

Therefore, in the current study we used Doppler ultrasound to examine the CBF in the middle cerebral artery during application of a judo chokehold. Specifically, this study focused on whether CBF is maintained at the minimum flow when a judo competitor “tapped out” at the limit of consciousness, i.e., just prior to losing consciousness. In addition, this study used NIRcws to examine changes in blood volume and in resultant oxygenation of the frontal lobe of the cerebrum during the period from application of a chokehold to the recovery phase after its release.

Methods

Subjects

This study was conducted with the approval of the ethics committee of the Faculty of Medicine, University of Tsukuba in Japan. Subjects were 4 male judo practitioners who were members of a university judo club. These individuals were extremely healthy and practiced judo almost every day for approximately 3 hrs. All subjects had practiced judo for a number of years and were highly proficient and experienced. Table 1 lists the subjects’ physical characteristics as well as their age,

judo background, and rank (*dan*). Fully informed consent was obtained from subjects who agreed to participate in this study.

Chokehold protocol

The chokehold was performed using judo choke techniques (*Kata-juji-jime*), which constrict the common carotid arteries. Subjects assumed a resting position on the floor (tatami mats), and the chokehold was applied by a professional judo teacher (Figure 1). In the present study, the chokehold was applied until the subject made a sign of “tapped out” which indicated the limit of consciousness. The chokehold was not continued until the subject lost consciousness (*ochi* = asphyxia); that is, when the subject tapped out, the chokehold was immediately released. The point of this study was to examine the state of CBF and the relationship between cerebral oxygenation and blood volume at the limits of cerebral activity, i.e., at the limits of consciousness. This study took the safety of the participants fully into account.

Measurement of CBF, heart activity, blood pressure, and oxygen saturation

hiCBF was measured in the middle cerebral artery using a Doppler ultrasound imaging system (SSD-5500, Hitachi Aloka, Japan). Electrocardiograms (ECGs) were also recorded using a Fukuda Cardio Base ECG recorder (FCP-4270, Fukuda Denshi, Japan). Systolic and diastolic blood pressure was monitored with an automated sphygmomanometer (STBT-780, Japan Colling Co. Japan). In addition, oxygen saturation was recorded using a pulse oximeter (Hasimo Rad-5, Fukuda Dens Japan).

Measurement of both blood volume and oxygenation in the cerebral frontal lobe region with NIRcws

A near-infrared single-distance continuous-wave photometer (HEO-100, Omron Inc., Japan) was used to non-invasively monitor cerebral oxygenation (gauged using oxy-hemoglobin/myoglobin, oxy-Hb+Mb) and blood volume (gauged using deoxy-hemoglobin/myoglobin, deoxy-Hb+Mb). The logical underpinnings and basic principles of near-infrared spectroscopy have been described in detail by Chance *et al.* [7-9]. In brief, near-infrared spectroscopy is performed with probes and a unit that is controlled via a computer. The probes consist of a light source and an optical detector. A pair of 2-wavelength LEDs is used as the light source. The LEDs have maximum wavelengths of 760 and 840 nm. Oxyhemoglobin (gauged using Hb/Mb) and total hemoglobin were calculated using the following formulae [15]:

$$\text{Doxy-Hb+Mb} = 0.66\text{DOD}_{760} + 0.80\text{DOD}_{840}$$

$$\text{Total-Hb+Mb} = 0.14\text{DOD}_{760} + 0.41\text{DOD}_{840}$$

ΔOD_{760} and ΔOD_{840} indicate changes in the optical densities (OD) at 760 and 840 nm, respectively. Oxy-Hb+Mb and $\Delta\text{Total-Hb+Mb}$ indicate changes in the concentrations of oxyhemoglobin/myoglobin (Hb+Mb) and total Hb+Mb, respectively. The light source was located 3.0 cm from the measurement site.

Statistical analysis

Data are presented as the mean $\pm$ standard deviation. In addition, Pearson’s method was used to examine statistically significant differences with $p < 0.05$.

Table 1: Ages, length of judo careers, judo rank, and physical characteristics of the subjects.

	Age (years)	Career (years)	Judo rank (dan)	Height (cm)	Body mass (kg)	Body mass index
Subject 1	20	11	3	162.0	62.0	23.9
Subject 2	20	10	3	167.5	65.0	23.0
Subject 3	20	9	3	170.5	73.5	25.2
Subject 4	20	12	4	175.0	87.5	29.6
Mean	20.5	10.5	3.3	168.8	72.0	25.4
(SD)	(0.6)	(1.3)	(0.5)	(5.5)	(5.5)	(2.4)

Figure 1: Application of a judo chokehold (Kata-juji-jime).

Results

Blood flow in the middle cerebral artery in response to a chokehold

Doppler ultrasound was used to measure the middle cerebral artery blood flow in response to a chokehold. Results of the chokehold and recovery process for subject T. S. are shown in Figures 2-8: Figure 2 is at rest; Figure 3 is at the start of the chokehold; Figure 4 is 5 sec after the chokehold began; Figure 5 is when the subject tapped out; and Figures 6-8 are 2 sec, 15 sec, and 30 sec after the chokehold was released. As these figures show, the chokehold promptly resulted in restriction of the middle cerebral artery blood flow. Blood flow decreased markedly after 5 sec, and it was completely cut off at the point the when the subject tapped out. Immediately after the chokehold was released, the blood flow increased markedly in the recovery phase. After 15 and 30 sec, blood flow tended to return to its previous resting level. This trend was observed in all of the subjects (data not shown). The mean peak flow at rest was 82.8 cm/sec. Figure 9 shows the changes in peak flow from the start of the chokehold to the point of tapping out and then after the chokehold release. Mean values for the peak flow at each time point are shown. The peak flow disappeared when the subjects tapped out, and then the peak flow was substantially increased at 2 and 5 sec after the chokehold was released. The time from the start of the chokehold to when the subjects tapped out differed somewhat depending on the subject but was approximately 7–15 sec, with a mean of 10.75 sec (Table 2). When the subjects tapped out, the mean O_2 saturation was 89.8% (Table 3).

Figure 2: Peak blood flow in the middle cerebral artery at rest.

Figure 3: Peak blood flow in the middle cerebral artery at the onset of a chokehold.

Figure 4: Peak blood flow in the middle cerebral artery at 5 sec after the onset of a chokehold.

Figure 5: Peak blood flow in the middle cerebral artery at the point of tapping out from a chokehold.

Figure 8: Peak blood flow at 30 sec after the release of a chokehold.

Figure 6: Peak blood flow at 2 sec after the release of a chokehold.

Figure 7: Peak blood flow at 15 sec after the release of a chokehold.

Figure 9: Peak blood flow in the middle cerebral artery during the application of a chokehold and in the recovery phase. The mean $\pm$ SD ($n = 4$) is shown.

Figure 10: Blood pressure and heart rate during a chokehold and in the recovery phase. The mean $\pm$ SD ($n = 4$) is shown.

Blood pressure and heart rate during a chokehold

Changes in both heart rate and blood pressure when a chokehold was applied are shown in Figure 10. Since the subjects were current judo athletes who practiced approximately 3 hours per day, their physical health was reflected in their mean heart rate in a resting supine position at about 43 beats/min, which increased to about 50 beats/min when the subjects tapped out. One minute after the chokehold was released, the heart rate returned to about 45 beat/min. The mean blood pressure in a resting supine position was 122/60 mmHg. When the subjects

Table 2: Time of the chokehold from onset to tapping out.

Subject	1	2	3	4	Mean	SD
Time(sec)	7	12	9	15	10.8	3.5

Table 3: Oxygen saturation at the point of tapping out of a chokehold.

Subject	1	2	3	4	Mean	SD
Oxygen saturation (%)	88	90	92	89	89.8	1.7

tapped out, the mean blood pressure rose to 137/62 mmHg. One minute after the chokehold was released, the blood pressure returned to the resting level of 120/50 mmHg.

The effect of a chokehold on the blood volume and on oxygenation in the frontal lobe region of the cerebrum using NIRcws

Changes in oxygenation and in blood volume due to a chokehold applied to subject Y. I. are shown in Figure 11, and similar changes in subject T. S. are shown in Figure 12. These figures indicate that oxygenation in the brain decreased abruptly when the chokehold was applied and reached its lowest level when the subject tapped out. When the chokehold was released, however, oxygenation gradually increased and plateaued at a level about half of that at rest. Oxygenation then tended to slowly return to its previous level. Thus, the return of oxygen levels to the resting level took some time. When subjects tapped out, the mean OD was -0.022 (Table 4). The blood volume similarly decreased abruptly when the chokehold began. Nevertheless, although the time before tapping out varied among the subjects, the blood volume returned to its resting level in about half the time that the chokehold took to apply. In other words, oxygenation in the brain temporarily decreases until an individual reaches the point of tapping out. By contrast, blood volume returns to its resting level as the chokehold is approximately 50-60% complete, i.e., prior to the individual tapping out.

Discussion

Chokeholds in judo are traditional martial arts techniques with a long history in Japan. These techniques can directly affect human life and death, and among the modern international and

Figure 11: Oxygenation and blood volume by NIR_{cws} in the frontal lobe during the application of a chokehold on subject Y.I.

Figure 12: Oxygenation and blood volume by NIR_{cws} at the frontal lobe during the application of a chokehold on subject T.S.

Table 4: Optical density values at the point of tapping out of a chokehold.

Subject	1	2	3	4	Mean	SD
OD	-0.020	-0.024	-0.024	-0.020	-0.022	0.002

official national sports chokeholds are allowed only in judo. There are numerous types of chokeholds, among which *Kata-juji-jime* is typical. This technique involves holding on tightly to the collar of the opponent's judo jacket and compressing both common carotid arteries with substantial pressure from the outside. This constricting technique cuts off blood flow without causing pain. In judo practice and matches, the chokehold is usually applied until the opponent taps out or loses consciousness. One hypothesis in this study was that a minimum blood flow would be maintained, despite a massive decrease in the CBF as a way for the body to preserve life, even when a chokehold brought a subject to the brink of either losing of consciousness or stupor. That is, the blood flow would presumably be maintained, albeit at a minimal level, so that an individual could tap out before completely losing consciousness. In anatomical terms, the right and left vertebral arteries arise from the subclavian arteries deep in the neck and join to form the basilar artery that connects to the arterial circle of the cerebrum. These arteries were assumed to preserve some blood flow even if the common carotid arteries were constricted. The common carotid arteries bifurcate into the internal carotid arteries, which in turn branch into the middle cerebral artery. As indicated by the hemodynamics of the middle cerebral artery and peak flow, blood flow abruptly decreased 5 sec after a chokehold began. Notably, blood flow in the common carotid arteries was cut off just prior to the subject ultimately losing consciousness;

that is, the CBF has already stopped when an individual remains barely conscious when tapping out. In previous studies of chokeholds, Ikai *et al.* [1] reported that the time prior to an individual completely losing consciousness was 10–12 sec, while Suzuki *et al.* [3] reported that it was 8–14 sec. One study that involved applying a chokehold until the individual passed out according to a sphygmomanometer cuff reported that the time prior to loss of consciousness was 12–15 sec. [3]. The current study did not involve applying a chokehold until the subject completely lost consciousness, and the chokehold was released at the same time the individual tapped out. The time from when the chokehold began until the subject tapped out was 7–15 sec (mean time of approximately 11 sec) when the subject was in a supine position and not resisting, which is consistent with that in the aforementioned studies. Thus, the CBF is completely cut off when an individual taps out due to a chokehold, which is almost at the same time in which they lose consciousness. If there is even a slight delay in the time from when an individual taps out until the chokehold is released, the individual could be brought close to death.

The current study used Doppler ultrasound. Ultrasonic waves were verified as being correctly directed at the blood vessel being imaged. Typically, the blood flow in ultrasound images is calculated as a steady state assuming that blood vessels are a perfect cylinder. Like the current study, a study by Raschka *et al.* [5] used Doppler imaging and cited a peak flow of 85.3 cm/sec. Similarly, the current study noted a peak flow at rest of 82.8 cm/sec. However, a look at the flow during a chokehold indicates that blood vessels deform considerably as a result of strong external pressure and ultimately close completely. Thus, the peak flow at different times does not accurately reflect flow at certain levels because of the changes in the shape of the blood vessels. In addition, various other factors may considerably affect blood flow, such as differences in externally applied pressure, the time prior to a tap out, the thickness and elasticity of a subject's vascular walls, and the size of the vascular lumen. The peak flow at 2 and 5 sec after a chokehold was released was about 20% greater than that at the resting level, because large quantities of blood had pooled due to occlusion of the common carotid arteries. Soon after the chokehold was released, the vascular diameter increased substantially from the diameter at rest, producing a large contractile force in response. Moreover, blood vessels are likely to expand due to the increases in the arterial CO₂ concentration [16]. Thus, the shape of the blood vessels differed widely at rest, during a chokehold, and after a chokehold was released. Evaluating the peak flow with the similar changes in vascular morphology is currently impossible, but the peak flow data from this study are still vital to determining blood flow in the middle cerebral artery during a chokehold.

Changes in both heart rate and blood pressure were examined from the start of a chokehold until a subject tapped out and after the chokehold was released. When the substantial external pressure of the chokehold was applied to the neck and the subject tapped out, the subject's heart rate increased by approximately 10–15 beats/min. At the same time, the subject's systolic blood pressure increased by around 10–20 mmHg, and

the subject's diastolic blood pressure increased 10 mmHg. After the chokehold was released, the subject's heart rate and blood pressure returned to their resting levels. These findings are generally in agreement with that of previous studies [1–3]. The cardiovascular response to a chokehold can be considered in terms of the mechanism of the carotid sinus baroreflex [17, 18]. *Kata-juji-jime* compresses and constricts the common carotid arteries, which substantially compresses the common carotid sinus at the root of the internal carotid artery at the same time. Therefore, occlusion of the common carotid artery increases both blood pressure and heart rate. However, when the walls of the carotid sinus stretch, baroreceptors in those walls sense the stretching and excite the cardiac branches of the vagus nerve in response. Simultaneously, the sympathetic activity on both the heart and blood vessels is inhibited. As a result, heart rate, cardiac output, peripheral resistance, and blood pressure decrease. Moreover, electrical stimulation of the carotid sinus has similarly demonstrated evidence of a carotid sinus baroreflex [17, 18]. This reflex decreases both blood flow and blood pressure, potentially causing a loss of consciousness, syncope, and cardiac arrest. Nevertheless, the heart rate and arterial blood pressure both increase just prior to either a tap out or loss of consciousness because of baroreceptors in the carotid sinus and aortic arch, which are nerves that regulate blood pressure. When blood pressure drops, inhibition of the cerebrovascular center in the medulla oblongata consequently decreases, and sympathetic nerve impulses increase, helping restore blood pressure to its previous level [18, 19]. Thus, the heart rate and blood pressure increase even though the carotid sinus baroreceptors are excited [18, 19]. In addition to this mechanism, inhibited blood flow excites chemoreceptors in the carotid body, causing an O₂ deficit (circulatory hypoxia = anoxia). Increased chemoreceptor activity causes a pressor reflex that is assumed to be associated with an elevated heart rate and elevated blood pressure [18, 19]. Furthermore, increases in the CO₂ levels in the blood are thought to bring about expansion of the blood vessels [16, 18, 19]. When Ganong [16] clamped the right and left common carotid arteries below the carotid sinus, the pressure in the sinus decreased, systemic blood pressure rose, and the heart rate increased. However, Ganong [16] described the effect as minimal, because although baroreceptors in the common carotid sinus and aortic arch act together, aortic arch baroreceptors probably still function normally when the common carotid arteries are clamped below the carotid sinus. Studies by Ikai *et al.* [1] and Suzuki *et al.* [3] supported the view that a chokehold increases both heart rate and blood pressure from the perspective of the carotid sinus baroreflex. Takagi and Nagasaka [20] stated that the pressure in the carotid sinus decreased as a result of compression of the common carotid arteries. In response, the heart rate, pulse pressure, and blood pressure in the brachial artery all increased.

No previous studies have used NIRcws to examine changes in brain oxygenation with a judo chokehold. The current study specifically examined both oxygenation and blood volume in the frontal lobe region of the brain. The blood volume was at its lowest level at midway from the start of the chokehold until the individual tapped out (around 5–6 sec after the chokehold

began). After release of the chokehold, the blood volume then gradually returned to its level at rest. This pattern was evident in all subjects. Oxygenation temporarily decreased up to the time of tap out, i.e. on the brink of consciousness. The mean OD at this point was -0.022 in the 4 subjects. About 5–6 sec after a chokehold is released, oxygenation plateaus at a level of about half of that at rest and does not quickly return to its resting level. Instead, the recovery of oxygenation in the brain is delayed even after blood flow in the middle cerebral artery has recovered. Therefore, oxygenation in the brain appears to take a relatively long time to return to its resting level, although there are individual differences in the time needed to recover. These findings can be used to better understand oxygenation in the brain resulting from a judo chokehold. Blood flow to the cerebrum reaches the frontal lobe through the common carotid artery, internal carotid artery, and middle cerebral artery. Therefore, a cutoff of blood flow in the common carotid artery due to a judo chokehold leads to a loss of cerebrum functions, such as the conscious, memory, and judgment [19]. Although blood volume quickly decreases when the chokehold begins, blood volume returns in about half of the time that elapsed from the start of the chokehold until the individual taps out. Therefore, in response to the crisis of reduced blood flow in the common carotid arteries that is cut off or restricted by the chokehold, residual blood volume from sites other than the frontal lobe of the cerebrum is thought to contribute blood to preserve life [19]. The circle in Willis has markedly less blood flow than that in the basilar artery. Furthermore, the basilar artery joins the posterior cerebral artery, and the combined blood flow circulates in the cerebellum, brain stem, and occipital lobe [19]. Therefore, it may be very difficult for the individual to function when the blood flow to the posterior cerebral artery is redirected to compensate for the reduced flow in a chokehold. By contrast, oxygenation temporarily decreases until an individual taps out, probably because brain neuronal cells continue to take up O_2 in the form of Oxy-Hb+Mb, and neural activity is maintained even while the blood supply is substantially restricted. However, oxygenation does not readily return to its resting level in the recovery phase after a chokehold is released. Therefore, once the functioning of brain cells has been maintained to the limit, Hb+Mb, O_2 binding ability in braincells, and recovery of consciousness are thought to be delayed even if the CBF is restored. Consequently, brain activity may take some time to recover. These phenomena can also be explained in terms of reactive O_2 species. Once a chokehold is released, large quantities of reactive O_2 species would be generated in the brain if oxygenation abruptly returned to its resting level, causing serious damage to brain cells and oxidation of blood vessels in the brain [21]. This phenomenon could cause brain dysfunction similar to that during reperfusion after ischemia following acute myocardial infarction. The body's defensive response is to inhibit or to avoid an abrupt generation of reactive O_2 species, which is presumably why oxygenation recovers at a slower rate.

The relationship between O_2 saturation and the OD for cerebral oxygenation during a tap out was also assessed. When a subject tapped out due to the chokehold in this study, the initial level of

O_2 saturation was the level just prior to loss of consciousness, i.e., asphyxia. A previous study [1] that measured O_2 saturation in the ear lobe reported that O_2 saturation was 82–85% when an individual lost consciousness. Assuming that O_2 saturation at rest is 98%, then a decline of 13–16% results in cerebral oxygen deprivation and may lead to a loss of consciousness. According to the present NIRCws results, the OD for oxygenation in the brain was -0.022 during a tap out. Assuming the individual completely loses consciousness, the OD would be approximately -0.030. In either event, examining cerebral oxygen deprivation in terms of the relationship between O_2 saturation and oxygenation (gauged using the OD) is a topic for future study.

The current results indicate that when a chokehold is applied, circulation is cut off, which is extremely dangerous in terms of the hemodynamics of CBF. This is true even if a competitor has sufficient brain activity to remain conscious enough to tap out. The brain is exquisitely sensitive to changes in oxygen dynamics, such that if blood flow to the brain is cut off, consciousness is lost within 10 seconds. In particular, sensitivity for hypoxia in the cerebral cortex is suggested to be higher than that in the brain stem. Moreover, even if brain functions are partly recovered after a hypoxic state, mental functions and intelligence do not return as quickly to their former state [16]. Although chokeholds are permitted by the rules during judo competitions, whether or not they should continue to be permitted in light of their physical effects relative to their benefit must be examined in the future. Being subjected to a chokehold reduces brain activity during growth and development in preadolescence, adolescence, and adulthood. Rendering an individual unconscious for a prolonged period could lead to accidental death, so such actions should probably be prohibited in principle. Assuming, however, that chokehold techniques are permitted in combative sports in the future and then the use of those techniques should only be permitted by adult athletes. During daily practices, coaches must provide adequate monitoring and supervision. In addition, frequently applying a chokehold to the same individual should be strictly prohibited. During a match, an athlete need not continue competing until his opponent taps out to indicate a clear winner and loser. Instead, the referee should declare victory when an athlete has assumed a position where a chokehold technique can be fully applied and that technique will be readily effective but before his opponent taps out due to the chokehold. This issue is important from medical, health, and educational perspectives. Moreover, the risks of chokeholds should be fully considered in future judo competitions overseas.

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