Medical Studies on "Choking" in Judo, with Special Reference to Electro-encephalographic Investigation

K. Suzuki

(Orthopedic Surgery, Nippon Medical School)

Forword

Judo, our national sport has become very popular throughout Europe and America, particularly after World War II. Just at the time when judo had begun to be popularized abroad, a problem had arisen in Japan among those interested in judo, i. e. just how dangerous is the "choking" technique? Hitherto, it was practiced under the popular notion and without scientific basis, that it was a dangerous technique. However, as judo gradually developed into an international sport, it became necessary to decide whether or not to include "choking" among the regular techniques. As Dr. H. Sato and Mr. M. Morikawa had been conducting scientific investigations on "choking", the late Prof. Saito made an agreement with the Kodokan authorities to undertake scientific researches on "choking". The Kodokan authorities had requested to elucidate the following problems.

1. Is there any danger to life?
2. What is the mechanism which causes unconsciousness?
3. Does it have any injurious effect on the body?

The author of the present paper by request of the late Prof. Saito, undertook to conduct experiments on the above, the results of which form the substance of this paper.

There are several ways of performing the "choking" technique in judo, but the main point in all the methods is the same, i. e. the application of strong pressure on the neck, thereby causing unconsciousness. Now, since an application of strong pressure on the neck is the vital point in this technique, three systems of the body, namely the circulatory, the nervous, and the respiratory may be considered to have a relation to the resulting unconscious state. Consequently, the following causative factors occur to one's mind, i. e. a disturbance in the cerebral circulation due to pressure on the blood vessels in the neck, reflex action initiated by pressure on the nerves in the neck, and asphyxia due to closure of the trachea. Shibusawa (1) states, "in judo there is a technique in which one strangles his opponent's neck. This very often results in unconsciousness on the part of the one being strangled. This unconsciousness does not appear to be caused by asphyxia, the stimulation of the carotid sinus being the chief cause. There are individuals very susceptible to this technique, and the unconsciousness may be considered as a state of shock resulting from carotid sinus reflex". However, it is difficult to accept the belief that the carotid sinus reflex is the main cause of unconsciousness resulting from "choking" in judo. For example, during an epileptic seizure, inspiration of low oxygen air, or injection into the vertebral artery, a

similar condition of unconsciousness results, the cause of which cannot be traced to carotid sinus reflex.

In the present study an attempt was made to elucidate the causative factor or factors of the state of unconsciousness resulting from "choking" in judo, while giving consideration to similar conditions as the ones mentioned above.

Experimental Methods

1. "Choking" by the Katajyuji technique. In this method, pressure is mainly applied to the sides of the neck and not to the trachea.

2. "Choking" by means of wrapping a manschette around the neck. In this method pressure is applied to the whole circumferance of the neck. The experimental subject was placed in a supine position on an electrically insulated bed in the electro-encephalogram recording room. The "choker" squated astride over the subject's abdomen, and thus performed the "choking". Recordings were made by a Yokogawa type oscillograph, and a "Noken" type three stage amplifier. The method used was the fronto-occipital lead with silver disc electrodes of 1.0 cm diameter.

Experimental Subjects

Nine young men (over 7th grade) of the Kodokan, and one of our colleagues Morikawa

Table 1. Experimental subjects and particulars of experiment.

Method of "choking"	No.	Exp. subject	Unconscious	E. E. G.	Pupil	Reflex	Pulse	Bl. pres.
(1) Katajuji-shime	1	S. IV	+	○	○	○	○	○
	2	A. III	−	○	○	○	○	
	3	〃	−	○	○	○	○	
	4	〃	−	○	○	○	○	
	5	W. V	+	○	○	○	○	
	6	K. V	+	○	○		○	
(2) Choking by means of manschette	7	M.	+	○		○		
	8	〃	+	○				
	9	S. IV	−	○		○		
	10	O. IV	+	○		○		
	11	M. II	±	○		○		
	12	〃	±	○	○	○		
	13	M. III	−	○		○		
	14	K. V	+	○			ECG	

○ items investigated. (+) complete unconsciousness. (±) incomplete unconsciousness. (−) failure to attain unconsciousness.

(without experience in judo) were selected as experimental subjects. Of these, four were "choked" with method (1), and six with method (2). One of the subjects was "choked" three times with method (1), and two were "choked" twice with method (2), making a total of 14 experiments.

The items selected for experiment are seen in Table 1.

Results

A. Summary of Observation

a) The differences in the two methods of "choking". The symptoms elicited by "choking" with method (2) were mostly that of irritation of the trachea. Until the subject fell unconscious he seemed suffocated and coughed continuously. After awakening the subject complained of an unpleasant feeling in the larynx, and started coughing again. These symptoms were not formed when "choking" was done with method (1), from which it is inferred that with the latter method unconsciousness may be occassioned without complete closure of the trachea.

The time required for the subject to fall unconscious when "choked" with method (2) was approximately twice that required for method (1). This is because of the comparatively long time required for the air pressure in the manschette to rise to the requisite level. The above were the only differences observed in the course of "choking" done with the two methods.

Table 2. Time required to fall unconscious and regain consciousness.

Method of "choking"	No.	Unconscious	Time required to fall unconscious	Time required to regain consciousness	
(1) Katajuji-shime	1	+	8″		Resuscitat. process
	2	−			
	3	−			
	4	−			
	5	+	14″	7″	Natural awakening
	6	+	9″	12″	
(2) "Choking" by means of manschette	7	+	12. 5″	16″	
	8	+	13. 5″	18″	
	9	−			
	10	+	15″	10. 5″	Natural awakening
	11	±			
	12	±	(40″)	(7. 5″)	
	13	−			
	14	+	37″	13″	Natural awakening

b) Time required for the subject to fall unconscious and awaken. In Exp. 1, tonic dilatation of the pupils was observed when the subject lost consciousness, and thereafter the appearance of this condition was fixed as the criterion for the moment of falling unconscious. However, in Exps. 2, 3 and 4 (same subject), when as soon as dilatation of the pupils were noted the pressure to the neck was loosened, the pupils soon contracted and the subject recovered consciousness. In Exps. 7, 8, 9, 10, 11, 13, and 14 the pupils were not examined, and the relaxation of the muscles of the body was chosen as the criterion. The criterion adopted for the moment of awakening was the moment the subject opened his eyes. However, these criteria cannot be said to be very accurate as the subject was sometimes in a state of incomplete unconsciousness. Table 2 shows the time required for the subjects to fall unconscious and awaken.

When method (1) was used the average time required for the subjects to fall unconscious was 10.3 secs.; with method (2) it was 19.25 secs. The time required from the moment the subject fell unconscious to the moment of awakening with method (1) was 9.5 secs., and with method (2) it was 14.4 secs.

From the above it is to be noted that when an adequate pressure is applied to the proper place, unconsciousness may be occassioned very quickly, and when the pressure is relaxed directly after falling unconscious recovery is also prompt.

c) The following were observed in the eyes of the subjects during "choking". As pressure was applied there appeared spasmodic contractions of the eyelids, and gradual dilatation of the pupils with occassional contractions. The direction of sight gradually turned upward. When the subject fell unconscious the eyelids were closed tight in most cases, but in a few the eyelids were slightly open. The pupils became tonically dilated (the moment of falling unconscious). The eyeballs turned upward. As the subject's condition approached awakening, the tonic contractions of the eyelids became relaxed, the pupils gradually contracted, and the direction of sight returned to normal. When the pupils returned to normal size the subject suddenly opened his eyes and looked around. The moment the subject's pupils returned to normal size and opened his eyes was fixed as the moment of awakening, however complete recovery of consciousness came a little later.

d) Convulsions. As soon as pressure was applied on the subject's neck, the latter's face became flushed and exhibited a congested appearance. Directly before the subject fell unconscious the muscles of the body became rigid, the body became slightly opisthotonic, and the extremities began to vibrate rhythmically. The next moment the subject became limp and fell unconscious. This was directly followed by clonic convulsions covering the entire body. Seven to eighteen seconds after falling unconscious, the subject opened his eyes and awoke.

There was one case (Exp. 10) in which a recurrence of clonic convulsions was observed once after awakening. On the other hand in a few subjects clonic convulsions were not observed even during the unconscious stage, however in most cases the convulsions were observed. In some cases the convulsions were quite violent.

The convulsions were similar to the ones observed during electric shock, or epileptic seizure, but much shorter in duration. Because of this short duration no cyanosis in the face, coma, frothing from the mouth, or urinary incontinence were observed.

e) Respiration. When "choked" the subjects respiration became repressed and irregular.

Sometimes the subject emited a clucking sound and spasmodically inspired. As the subject became unconscious respiration appeared to stop temporarily, but when he was in convulsions irregular respiratory movement was observed. Some subjects at the inception of each convulsive seizure were seen to inspire, and when the convulsions subsided were seen to expire.

After awakening, respiration increased for some time.

According to Morikawa's X-ray observation on the movement of the diaphragm during the unconscious stage, respiratory movement appeared to be accelerated, and two minutes after awakening, returned to normal. This acceleration in the movement of the diaphragm might have been due to convulsions, however it implies an ingress and egress of air in the lungs to some extent. The increase in respiration after awakening is no doubt a compensatory action. After awakening unlike after an epileptic seizure there was no snoring. Also, there was no increase in the depth of respiration as after violent excercise.

B. Consciousness

The problem of "consciousness" is a very difficult one. According to Hayashi (2) the objective criterion of being conscious is the ability to reply. Araki (3) (4) states that the state of being unconscious is when the subject is unable to perform the articles classified by Schiller (1952), especially when the subject does not respond to sensory stimuli. He differentiates coma from semi-coma, however the criterion of differentiation is an empirical one, and gives reflex action as the main point. In the present experiment the duration of the state of unconsciousness was very short, consequently it was difficult to differentiate the various stages of unconsciousness. Here it was divided into two stages, loss of consciousness and dullness of consciousness.

When the subject's loss of conciousness was complete the state corresponded to the absence of all the articles described by Schiller (5), i. e. (1) no response to stimuli, (2) no response to sensory stimuli, (3) no concentration, (4) lack of discernment, (5) lack of memory, (6) inability to converse or communicate, (7) lack of voluntary movement, (8) lack of "consciousness" from the point of view of psychoanalysis.

At the inception of "choking" the subject's consciousness became vague, and according to Morikawa's own experience one felt as though a black curtain were drawn, and the next moment he was completely blacked out. As one awoke he felt as though a black smoke screen were swept away and gradually regained consciousness.

As the subject awoke after being "choked" he seemed to recognize the surrounding objects, but at first did not understand why he was there lying on a bed. The condition resembled the awakening after concussion of the brain, however the subject soon realized that he had been "choked". Unlike after awakening from the loss of consciousness caused by concussion of the brain, no subject asked questions like, "where am I", or "what happened". The stage of retrograde amnesia lasted only for a very short period.

All those who have experience in losing consciousness after being "choked" in judo say that the feeling is not bad at all, but quite good. Some say that they were in a dream. One subject explained that he was flying through space in his dream, while another said that he was viewing a beautiful scenery. It is interesting to note that not one had an unpleasant dream. There were some who did not remember whether he had a dream or not, but said that the feeling was quite good, and jokingly asked to be "choked" again. When

the subject once lost consciousness there was no further disturbance of consciousness. There was no condition similar to the secondary sleep described by Fuchs which often follows awakening after concussion of the brain. However, the subjects in the present study after awkening were put in a dimly lit room and told to remain quiet, and consequently after about 15 mins. many began to feel drowsy.

During an incomplete loss of consciousness, typical convulsive seizures were not observed, however the consciousness became vague, and response to stimuli became dull. Sometimes the light reflex disappeared, and presumably there were moments of momentary loss of consciousness, which recovered soon after loosening the pressure on the neck.

In cases where the subject did not completely lose consciousness he remembered that he had been "choked".

C. Pulse

In 1949, at a meeting of a group of orthopedic surgeons of the Nippon Medical School, Sato reported that during the unconscious stage resulting from "choking" the pulse rate was increased, while the present author stated that he had observed bradycardia. Sato's statement was based on electro-cardiographic observation, while the latter's was based on taking radial pulse at the wrist. Subsequently, the present author has also made electro-cardiographic observations endorsing Sato's report. Immediately prior to falling unconscious, the pulse became thready and rapid, finally it became imperceptible due to the fine contractions of the muscles. When the subject became completely unconscious it was difficult to feel the pulse due to convulsions. As awakeing approached, the tension became good, the pulse full and regular. Three minutes after awakening, the pulse returned to normal.

When "choking" was done by method (2) a few seconds prior to falling unconscious the height of T decreased and remained low during the unconscious stage. R also, generally was low except in a few cases in which it was high. As awakening neared, R gradually returned to normal, but T remained low until more than 10 secs. after awakening.

Because of the feeble pulse and the convulsions during the unconscious stage, it was difficult to make accurate observations of the radial pulse. There were two forms of recovery process of the pulse to normal. In certain individuals the pulse from tachycardia gradually returned to normal, whilst in others there was a period of bradycardia before returning to normal. This discrepancy was most probably caused by the different conditions in the internal pressure of the carotid sinus, as a result of the different ways of "choking". In two cases, comparatively accurate measurements were obtained as shown in Fig. 1. The R-R interval as shown in Fig. 1 was shortened a few seconds prior to and during the unconscious stage.

D. Blood Pressure

Blood pressure measurement was made in Exp. 1, however only the systolic pressure was obtained immediately prior to the unconscious stage. At rest the systolic pressure was 130 mmHg, the diastolic pressure 60 mmHg, and immediately prior to the unconscious stage, the systolic pressure registered 180 mmHg.

Aside from the present experiment, the common carotid arteries on both sides were pressed hard in three individuals (they did not lose consciousness), and their blood pressures

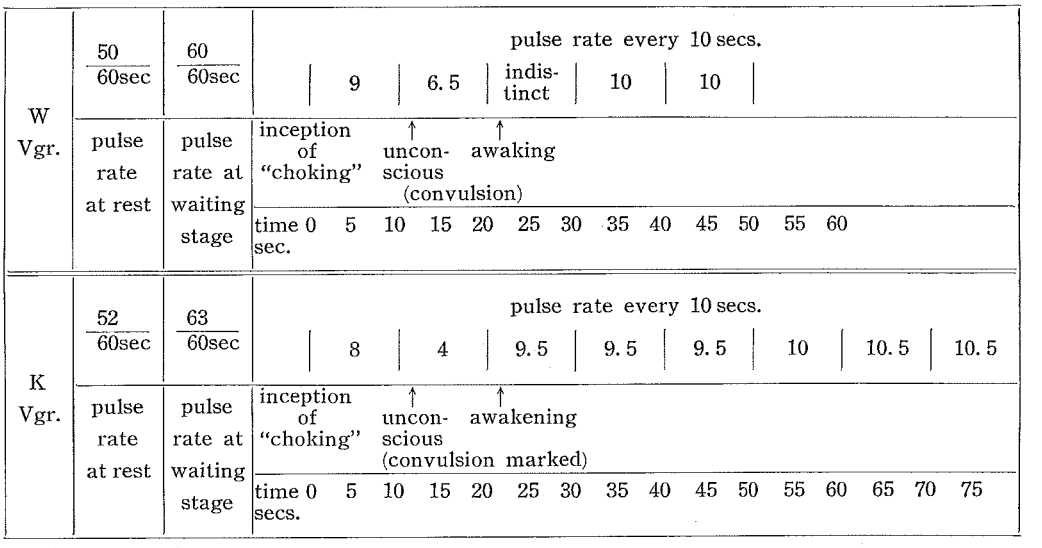


Fig. 1 Pulse rate during Katajuji-shime

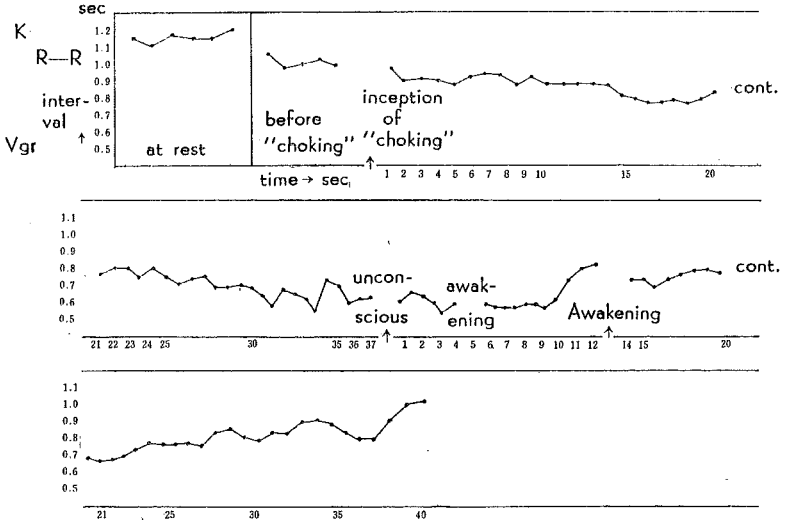


Fig. 2 R-R interval (electro-cardiogr.) during "choking" by means of manschette.

examined with the following results.

From the above it is clear that "choking" causes a rise in systolic pressure.

	At rest mmHg	During application of pressure mmHg
1	129～ 96	180～110
2	122～ 56	156～ 60
3	125～ 70	150～ 72

E. Reflex

After "choking" with method (1), the patellar reflex and foot clonus were examined, and after "choking" with method (2) the patellar reflex was examined. In one individual, during the unconscious stage (method (1)), a marked increase in patellar reflex was observed, and also in another individual (method (2)), a positive Babinsky was elicited.

Light reaction of the pupil was examined in all the subjects "choked" with method (1), and in only one subject "choked" with method (2). When the subjects lost consciousness completely there was temporary disappearance of the light reflex. As the subjects approached awakening the dilated pupils gradually contracted and observation of the light reflex became difficult.

F. Cerebrospinal Fluid

As it was difficult to make examinations of the cerebrospinal fluid during "choking", four subjects were selected and while in a sitting position, their common carotid arteries on both sides were pressed until pulsation stopped on the distal side, after which examinations were made on the cerebrospinal fluid. At the moment pulsation stopped distal to the point of pressure, the face became flushed, the veins on the forehead distended, and the eyes closed. The subjects did not lose consciousness but apparently were on the verge of losing consciousness. The results obtained are shown in Table 3. As will be seen from the

Table 3 Cerebro-spinal fluid pressure on application of pressure on neck.

Exp. subject	sex	age	condition of experimental subject	at rest (mm)	pressure on nech (mm)	pressure on jugular vein (mm)
Ki	♂	36	sciatica (right)	410	870	550
I	♂	23	lumbago	430	830	600
S	♂	29	acquired epilepsy (after meningitis)	400	850	490
K	♂	26	acquired epilepsy (after head injury)	420	740	500

Table, those with a previous history of cerebral lesion showed lower cerebrospinal fluid pressure than those without it. In all the above subjects when strong pressure was applied to the common carotid arteries, the cerebrospinal fluid pressure rose conspicuously.

Experimental subject S (Tab. 3) had undergone trepanation ten years previously, and had a small window-like boneless area in the cranium which normally formed a depression in the scalp. From a few days prior to an epileptic seizure to the onset of the seizure, this depression had invariably disappeared. This disappearance of the depression was also observed upon pressure to the neck, which showed that intracranial pressure had become elevated.

G. Other Examinations

The observations listed in Table 4 were made between one hour and thirty minutes prior to "choking" with method (1) and also thirty minutes after awakening, but no significant changes were observed.

H. Electro-encephalography

When method (1) was used for "choking" electro-encephalographic recordings were made at rest, inception of "choking", unconscious stage, and after awakening. When method (2) was used the recordings were made at rest, waiting period, inception of "choking", uncon-

Table 4

	Exp. subject	Sh. IV gr.	A. III gr.	W. V gr.	K. V gr.
before expesiment	blood pressure	130~60	110~70	125~65	115~65
	pulse	55	59	50	52
	vital capacity	4100	5720	5600	4500
	dorsal muscle strength	150	200	168	180
after experiment	blood pressure				
	pulse	56	58	52	52
	vital capacity	3550	5730	5380	4650
	dorsal muscle strength	145	195	169	180
	heaviness of head, headache	(—)	(—)	(—)	(—)
	vertigo, vomiting	(—)	(—)	(—)	(—)

scious stage, and after awakening.

a. Electro-encephalographic Findings of Each Experiment.

(Exp. 1) Until 3.5 secs. after inception of "choking" recording was defective. Subsequent recordings until 5.3 secs. were also hindered due to electrical discharge from the muscles, but a slight decrease in amplitude was noted. From 5.7 secs. after inception of "choking" the amplitude increased, and 150~200 msec waves were seen with α waves. From 6.7 secs. a change in the form of the waves were noted, with the appearance of 75~100 μV. 200~230 msec waves. At 8.0 secs the subject fell unconscious with the appearance of 4 waves of 71~51 μV. 110~160 msec, followed by a 70 μV. 330 msec wave and one spike-like wave. From 1 sec. after the moment the subject fell unconscious to 1.5 secs., the recording was disturbed due to artifacts caused by the convulsions.

Thereafter, the recording became irregular due to the appearance of 3/sec~1/sec wave which superposed the α wave, and high voltage 150~200 msec wave. Also the resuscitating operation disturbed the electrodes and recording thereafter was not possible.

(Exp. 2) From the inception of "choking" to 8.5 secs., recording was disturbed by artifacts. From 8.5 secs., a low voltage wave appeard for one second, and from 9.5 secs. to 11 secs., the wave became similar to that at rest, however an oscillation of the base line was observed. Thereafter until 16 secs., artifacts again appeared. From then on for 4 secs. an irregular combination of α, δ and β waves were recorded. In the following 3 secs., the 330~600 msec wave was superposed by α or a wave having 1/2 its wave length. These gradually separated and became independent waves.

(Exps. 3, 4) Nearly identical to Exp. 2.

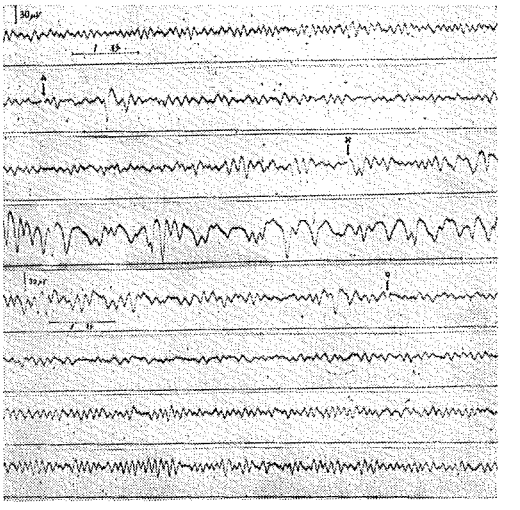
(Exp. 5) Certain parts of the recording were not complete, however 8 secs. after inception of "choking", the wave length increased, at 10 secs., waves of over 200~250 msec. 70~100 μV appeared, at 14 secs. the subject fell unconscious. Sixteen seconds after loosening pressure, artifacts appeared and recording was stopped; 1.5 mins. after awakening recording was resumed, and at that time although the α wave was predominant a few 10/sec. 80 μV

waves were interspersed. Even 2 mins. after awakening, δ waves superposed by α waves appeared.

In this subject as seen in Table 1, other examinations were simultaneously conducted, and consequently might have slightly disturbed the recording.

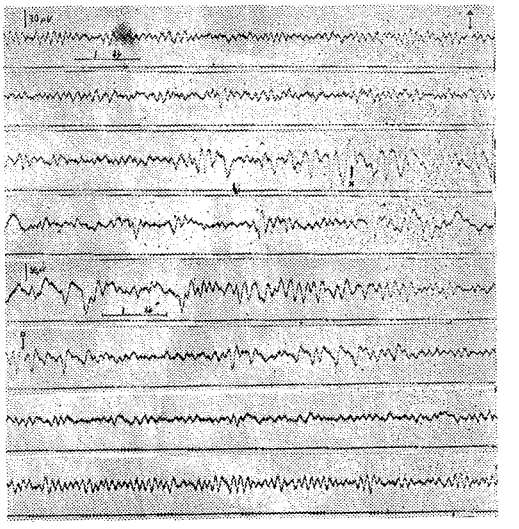
(Exp. 6) Aside from electro-encephalographic observations, the subject's pulse, and the pupil of one eye were observed. The α wave at rest had a frequency of 102 msec., with an amplitude of 5~20 μV (average 9.9 μV). Until 5 secs. after inception of "choking" artifacts due to the movement of the body appeard, however from 2 secs. after inception acute waves of 50 msec. 50μV appeared at 0.7~0.8 secs. intervals. In the following 1.5 secs. a superposition of α and β waves, and in the next 2 secs. a mixture of δ and α waves were observed, which were followed by 300 msec. 33 μV wave. The next moment the subject fell unconscious, after which for 2 secs. artifacts appeared, and in between 50 msec. 77μV waves were recorded. From 2 to 3 secs. after falling unconscious, a high voltage α and δ waves were recorded, after which for 2.5 secs. artifacts appeared, followed by 200 msec waves for another 2 secs. Again artifacts appeared for 2.5 secs., and at 17 secs. a trail, and 10/sec sharp waves were recorded, and at 21 secs. the subject awoke. For 20 secs. after awakening a mixture of high voltage wave of 150~200 msec and a small frequency α wave were recorded. One minute after awakening the continuity of the α wave was still low but from 1.5 mins. after awakening normal conditions were revived.

(Exp. 7) For 1 sec. after inception of "choking", β waves were predominant, and subsequently until 7secs., the frequency slightly became longer and 6/sec~7/sec waves were seen sporadically. Until 10 secs. the recording was irregular and the amplitude was on the decrease. For 1.5secs. after 10secs. the frequency became 110~160 msec, and the amplitude was 2~3 times that of the α wave at rest. For the next 0.5 sec. lower voltage waves were seen, and the subject fell unconscious. For 1.4 secs. after falling unconscious, waves with larger frequency and amplitude appeared followed for 1 sec. by δ waves, and then the wave length gradually increased. In the following 1 sec. waves with 3~6 times the amplitude of α waves at rest were seen, and subsequently until 9.5 secs. 4/secs~3 sec waves appeared. From 9.5 secs waves with larger frequency and amplitude than α waves appeared for 1.5 sec. From then on until awakening the voltage gradually decreased and α, β waves and intermediate slow waves were recorded. For 6 secs. after awakening β waves superposing α and β waves appeared, with occassional appearance of α waves with low amplitude, after which until 20.5 secs. α waves were predominant, however the amplitude was still low. Subsequently for 1.5 secs. intermediate slow waves appeared. Normal waves were recorded 1.5 mins. after awakening, however intermediate slow waves were still intermingled.



E. E. G of Exp. 7

(Exp. 8) For 1 sec. after inception of "choking" β waves were predominant and superposed α waves, and until 1.5 secs. the amplitude was lower than at rest. Subsequently until 7.8 secs. the frequency and amplitude showed a tendency toward the increase, and intermediate slow waves, α and β waves appeared irregularly.



E. E. G of Exp. 8

From 7.6 secs. to 9.3 secs followed waves with increased frequency and amplitude than α waves, and thereafter for 1.7 secs. the voltage become low. From 11.3 secs. the amplitude suddenly increased with a frequency similar to α waves, but two waves with amplitudes 3.5 times that of α waves suddenly made their appearance. At the moment the subject fell unconscious, four waves of 5/sec. 47μV were recorded. This was followed by 455 msec. 50 μV and 255 msec. 47μV waves with superposed α waves. A few moments later high voltage δ waves appeared, but were hindered by muscle electrical discharge. 2.5 secs. to 8.5 secs. after the subject lost consciousness waves with low voltage were predominant, and were irregular. Low voltage fast waves, intermediate slow waves, an occassional high voltage fast waves, and artifacts were recorded. From then on until 12.7 secs. large slow waves appeared, followed by waves of slightly high amplitude, but predominantly intermediate slow waves with 2-3 times the amplitude. At the time of awakening for 1.5 secs. the voltage became low. For 2 secs. after awakening low voltage α and β waves were recorded, and from 3.4 to 5.2 secs δ waves were predominant. From 6 secs. after awakening, low voltage α waves were predominant, but occassionaly superposed waves. From 1 sec. to 20 secs. after awakening there was a tendency for the voltage to become lower than at rest, but α waves were predominant with occassional appearance of β waves. Four minutes after awakening β wave were seen only rarely; the frequency became lower and the amplitude higher, and α wave became spiked. Seven minutes after awakening the recordings were nearly indentical as at 4 mins.

(Exp. 9) Until 4.8 secs. after inception of "choking" recording was hindered by artifacts, thereafter until 13.3 secs., although the voltage was rather low, waves of various frequencies were recorded, after which δ waves appeared. Until 22-31.7 secs. both frequency and amplitude were increased. After 31.7 secs α waves superposed δ waves. In this experimet the subject did not lose consciousness, and "choking" was intervened at 38.7 secs. Thereafter for 3.4 secs artifacts appeared, after which until 8.8 secs. intermediate slow waves were predominant, although a few δ and α waves appeared. From then on for 3 secs. artifacts appeared. Thirteen seconds after releasing pressure, aside from the occassional appearance of β and δ waves, the recording was nearly back to normal.

(Exp. 10) Until 6 secs. after inception of "choking," the amplitude was low, and fast waves were seen intermingled, after which there was a tendency for the frequency to increase, and from 10 secs. δ waves appeared. For 1 sec. after 12.5 secs. low voltage waves were

seen, and from 13.5 secs. five 5/sec. 20-30 μV waves appeared for 1.2 secs. This was followed by a disturbance of the base line, and meanwhile the subject lost consciousness. From then on for 5 secs. there was a disturbance of the base line. Meanwhile a short wave of 30-50 msec appeared. For the next 3 secs. a low voltage low frequency slow wave appeared. Thereafter until awakening 2-3 intermediate slow waves either supperposed by α waves or independently appeared. After awakening, for 3 secs. the voltage was low, and intermediate slow waves superposed by β waves, or β waves alone appeared.

From 3 secs., a slightly high voltage β wave appeared with an occasional appearance of intermediate slow waves superposed by β waves. From 1.5 mins. after awakening α waves with a slightly low amplitude were predominant with an occassional appearance of intermediate slow waves, and intermediate fast waves, and 4 mins. after which although the amplitude was low α waves were predominant, and an occassional appearance of intermediate fast waves were noted. Seven minutes thereafter, fast waves very often appeared.

(Exp. 11) 5.6 secs. after inception of "choking" a marked change in the form of the waves appeared and 270 msec. 55 μV waves followed by a rather low slow wave for 3 secs.. From 9.0 secs. to 14.5 secs. high voltage wave with a maximum of 64 μV with varying frequencies of from 100-325 msec were recorded. From 14.5 secs. to 23.7 secs. various waves with high voltage were seen, after which until 26.9 secs. δ waves appeared. Thereafter, the voltage gradually lowered with an occassional appearance of β waves. At 33.5 secs. "choking" was intervened, after which for 10 secs. the recording was nearly indentical with that at rest, and from then on until 30 secs. recording became irregular, δ waves being predominant with occassional appearance of large slow waves. These large slow waves might have been artifacts.

(Exp. 12) Until 2.5 secs. after inception of "choking", and also from 3.7 secs. to 4.2 secs. artifacts appeared. From 11.5 secs. to 14.5 secs. the amplitude became slightly reduced, and β waves appeared with intermediate slow waves.

Thereafter until 19 secs. recording was nearly identical to that at rest, and for 1 sec. after 19 secs. the volage became low. and β waves were seen. From 21.5 secs. to 24.2 secs, high voltage slow waves were predominant, after which until 37.1 secs, recording became irregular, and 250-310 msec. ca 50 μV large slow waves appeared sporadically, also various other waves were recorded. From 37.1 secs, artifacts appeared with a disturbance of the base line. At 40.2 secs. "choking" was intervened. At 38 secs. a spiked wave of 80 μV was recorded. Five seconds after releasing pressure artifacts appeared, and from 5 secs. to 5.8 secs. waves appeared followed by high voltage intermediate slow waves. Thereafter for 0.9 secs. low voltage β waves were also seen, and 0.8 secs. after which the subject awoke. Recording thereafter was disturbed due to movement of the electrodes.

(Exp. 13) This subject also did not lose consciousness completely, and the amplitude lowered, and β waves appeared. From then on until 19.5 secs., aside from an occassional appearance of 140-170 msec intermediate slow waves, no marked changes were observed. From 19.5 secs., changes in the form of the waves were recorded, and from 31 secs., δ waves of 150-300 msec were predominant with an occasional appearance of low voltage α waves. A few fast waves were also seen. Thereafter until releasing pressure, a low amplitude with a low frequency α wave and β wave were predominant. 4.3 secs. before releasing pressure, there were a few high amplitude waves although in general voltage was low. Directly after

releasing pressure an increase both in amplitude and frequency was observed, and α waves were predominant. 8.4 secs. after releasing pressure, the amplitude gradually decreased, and from 12.5 secs. to 17.4 secs., the voltage became low and δ waves appeared. Thereafter, for 3 secs normal α waves were seen after which followed low voltage waves, normal α waves, and again low voltage waves. 2.5 secs. after releasing pressure the average amplitude reached its minimum. Meanwhile, although β waves were numerous δ waves also appeared often, making the average frequency rather low. One minute after releasing pressure normal findings were observed.

b. Analysis of Electro-encephalographic Findings.

I. An analysis according to Motokawa's method of the electro-encephalographic findings of Exps. 6, 7, 8, 9, 10, 11, 12, and 13 are given in Tab. 5. As seen in the Table the subjects were divided into three groups: i) those that lost consciousness completely, ii) those that lost consciousness incompletely, iii) those that did not lose consciousness.

i) Those that lost consciousness completely. a) During the act of "choking." Toward the end of this period, slow waves were recorded, and as a result in spite of the appearance of waves with high frequency and low amplitude earlier, the average frequency was low. The amplitude at the inception of "choking" became low, but just prior to losing consciousness, the frequency was elongated and the amplitude increased, consequently the average amplitudes in Exps. 6, 7 and 8 were high, and that of Exp. 10 low. The rates of appearance and continuity of α wave were in all experiments low.

b) During the unconscious period. The average frequency was further increased at this period, however toward awakening the frequency tended to return to normal. The amplitude at the begining of this period was markedly increased, consequently the average amplitude was also high. The rate of appearance of α wave was further lowered, and in Exp. 10, it was 1/2 that of normal. The continuity of α wave in Exp. 7 and 8 was slightly better than during the "choking" period, but lower than normal. Toward the end of this period α waves were seen successively, making the continuity better than during the previous period.

c) Directly after awakening. The average frequency in Exp. 6 was higher than at rest, whilst in others slightly lower. The average amplitude in Exp. 6 was very high, in Exp. 10 slightly high, and in Exps. 7 and 8 slightly lower than at rest. The rate of appearance of α wave in Exp. 6 was low, in others it was slightly low. The rate of succession in Exps. 6 and 10 were low, in Exps. 7 and 8 slightly low.

d) One minute after awakening. The waves appeared nearly identical to those at rest, however the average frequency in Exps. 7 and 8 was comparatively low, in Exp. 10 slightly low, and in Exp. 6 nearly identical to that at rest. The average amplitude in Exp. 6 was very high, in Exps. 7, 8 and 10 slightly low. The rate of appearance of α wave in Exp. 6 was good, in Exps. 7, 8 and 10 low.

e) Four minutes after awakening. The average frequency in Exps. 7 and 8 were rather high, and in Exp. 10 nearly identical. The average amplitude in Exps. 7 and 8 was slightly high, in Exp. 10 slightly low, however the rates of appearance of α waves in all the experiments were identical to that at rest, showing that approximately 4 mins. after

Table 5

Exp. No.	Period during experiment	Aver. frequency (msec)	Aver. amplitude (μV)	Rate of appearance of α wave	Continuity of α wave
6	at rest	102.0	9.9	77	7.0
	latter part of "choking"	137.3	12.7	26	2.0
	unconscious stage	not clear			
	directly after awakening	96.7	26.3	22	1.8
	from 1 min. after awakening	107.1	21.1	74	4.7
	〃 1.5 mins. 〃	102.2	14.3	83	7.0
av. 7, 8	at rest	99.8	9.7	96	9.5
	during "choking"	107.8	11.6	87	6.1
	unconscious stage	137.4	18.2	77	7.7
	directly after awakening	106.6	8.5	84	8.6
	from 1.5 mins. after awakening	105.1	9.4	90	9.3
	〃 4 mins. 〃	96.1	12.3	96	6.7
	〃 7 mins. 〃	94.6	12.9	97	11.2
	〃 12 mins. 〃	96.2	11.6	97	10.1
9	at rest	92.2	8.8	91	8.5
	early part of "choking"	105.6	6.8	75	5.4
	latter 〃	176.6	18.2	42	2.9
	from 3 secs. after releasing pressure	130.9	10.5	69	4.4
	〃 25 secs. 〃	116.6	6.0	79	5.2
	〃 50 secs. 〃	91.9	7.5	68	8.3
	〃 4 mins. 〃	89.8	7.2	91	9.6
10	at rest	91.0	9.5	93	10.8
	during "choking"	123.8	8.3	74	5.8
	unconscios stage	172.2	25.8	46	2.3
	directly after awakening	98.4	11.1	84	4.1
	from 1 min. after awakening	93.3	8.3	88	7.8
	〃 4 mins. 〃	91.2	7.8	93	23.6
	〃 7 mins. 〃	88.5	7.8	92	9.6
av. 11, 12	at rest	96.5	7.8	98	35.0
	early part of "choking"	106.0	18.2	74	4.1
	latter 〃	101.5	12.3	72	3.7
	directly after releasing pressure	102.9	9.3	80	5.5
	from 15 secs. after releasing pressure	140.0	15.3	64	3.2
13	at rest	104.7	13.9	98	15.8
	early part of "choking"	103.8	11.4	85	7.7
	middle 〃	117.2	12.6	69	7.6
	latter 〃	83.2	9.5	64	4.3
	directly after releasing pressure	103.7	9.5	87	5.6
	from 2.5 secs. after releasing pressure	105.6	7.3	73	5.2
	〃 1.5 mins. 〃	104.7	16.4	97	10.4

awakening the waves were nearly normal.

ii) Those that lost consciousness only incompletely. From the averages of Exps. 11 and

12, a) at the inception of "choking" the findings were identical to that of those that lost consciousness completely, however as "choking" was continued further, toward the latter part, the average frequency became lower and average amplitude higher than at rest, although slightly less than at the beginning. However, the rate of appearance of α wave was low. This might have been due to the appearance of β waves toward the latter part.

b) Immediately after "choking" was released, the finding approached those at rest more than during any other period.

c) Fifteen minutes after "choking" was relaxed, the findings became very similar to that at the unconscious stage.

iii) Those that did not lose consciousness. In Exp. 9, at the inception of "choking", the findings were similar to the "choking" stage of those that lost consciousness completely, and the findings during the latter part of the "choking" stage were identical to those of the unconscious stage of those that lost consciousness completely. Three seconds after releasing pressure, the findings were still very similar to the ones during the unconscious stage. 25 secs. after releasing pressure, the findings were very similar to those during the awakening period of Exps. 7 and 8. 50 secs. after releasing pressure, the average amplitude was slightly low, and the rate of appearance of α waves low. Four minutes after pressure was released, the average amplitude became low and the average frequency became high, however the rates of appearance and continuity of α waves were good.

In Exp. 13, toward the middle part of the "choking" period, the findings were similar to those during the "choking" period of those that lost consciousness completely, but toward the latter part the findings showed characteristic features, i. e. the averages of the amplitude and frequency were smaller than at rest, and the rates of appearance and continuity of α waves were low. From this it may be inferred that many α waves also appeared. When the "choking" was relaxed, although the average amplitude was low the average frequency was nearly normal. The rates of appearance and continuity of α wave were low, consequently it may be inferred that intermediate slow waves with comparatively low amplitude, and β waves also appeared. One minute after "choking" was relaxed, findings returned to normal.

II. The histograms of the amplitude and frequency of Exps. 7, 8, 9, and 10 are shown in Figs. 3, 4, 5, 6, 7, 8, 9 and 10.

(Exps. 7, 8) The maximum of the frequency distribution during the resting period was 100 msec with 80-100 msec waves being predominant. As regards amplitude 7-8 μV was the maximum.

During the "choking" period the maximum of the frequency was 110 msec with 70-120 msec waves being predominant. 130-170 msec waves were also very often seen. 40 msec β waves gradually began to appear. The frequency while shifting toward the increase also tended toward the decrease making the distribution curve low. The maximum of the amplitude was around 5-10 μV, with the appearance of 5-12 μV being most often, however reaching 43-44 μV.

During the unconscious period, the maximum frequency was 110 msec, and 70-130 msec waves being predominant. The curve was low reaching 380 msec. The maximum amplitude was 9-12 μV, tending toward the increase, and reaching 61-62 μV, resulting in a low curve. Directly after awakening, the 100-120 msec waves were numerous with 110 msec being the maximum, and reaching 310 msec. The maximum amplitude was 7-8 V with 3-10 μV waves

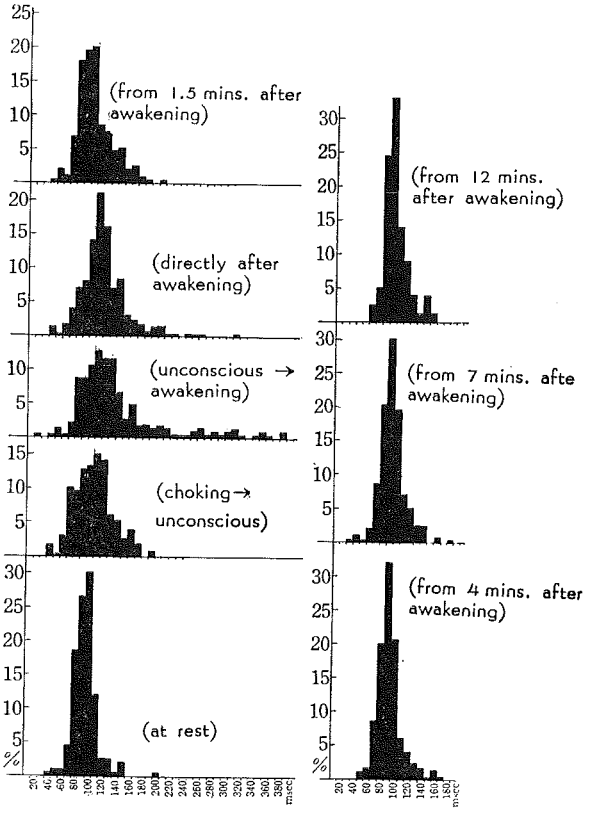


Fig. 3 Frequency Histogram of Exps. 7, 8

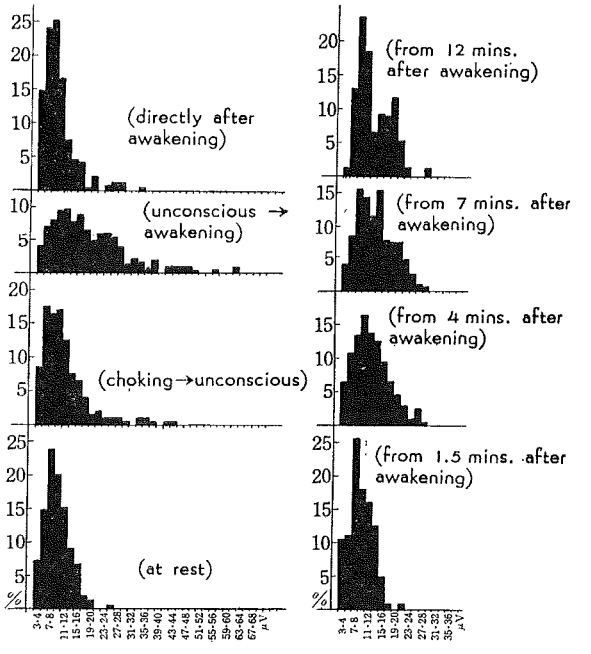


Fig. 4 Amplitude Histogram of Exps. 7, 8

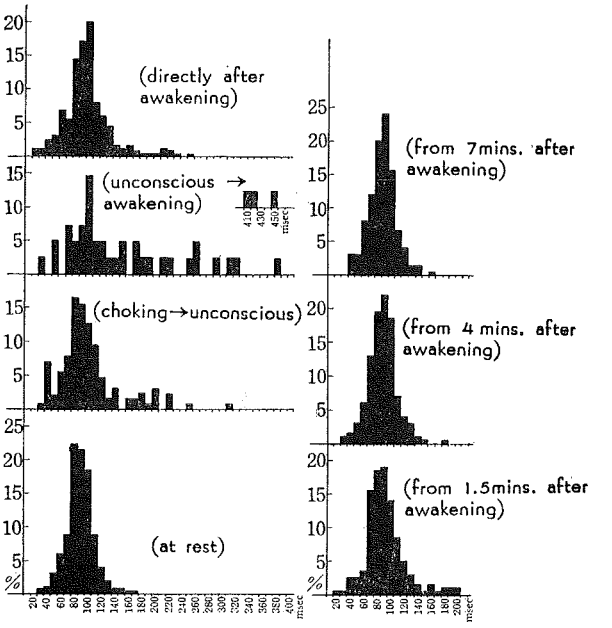


Fig. 5 Frequency Histogram of Exp. 10

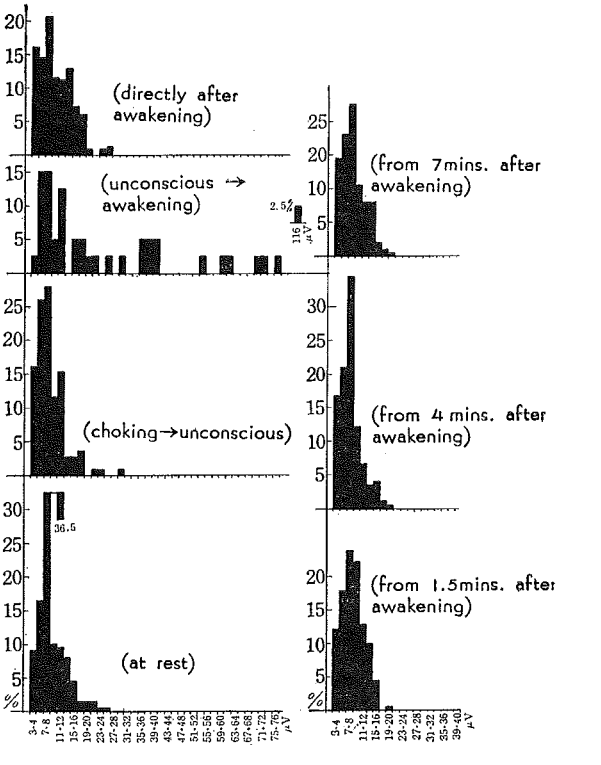


Fig. 6 Amplitude Histogram of Exp. 10

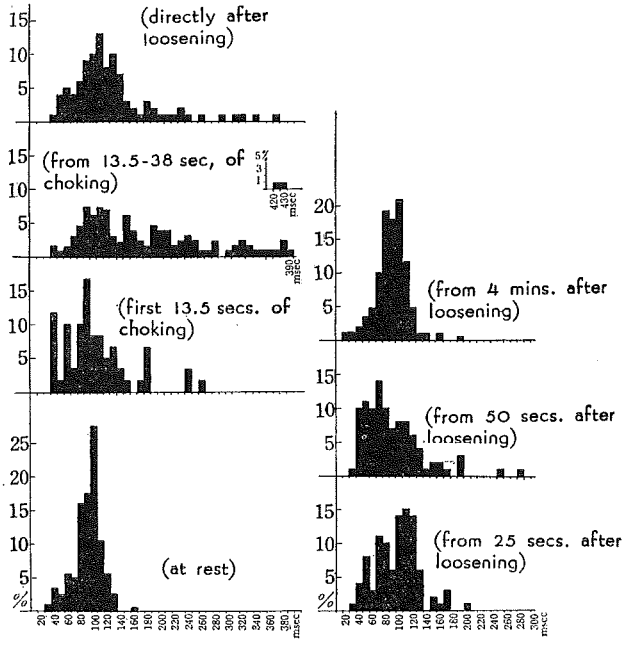


Fig. 7 Frequency Histogram of Exp. 9

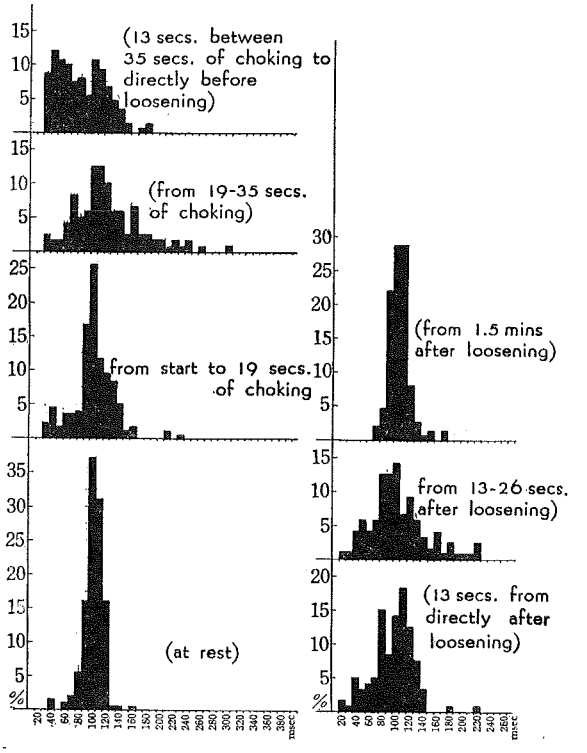


Fig. 9 Frequency Histogram of Exp. 13

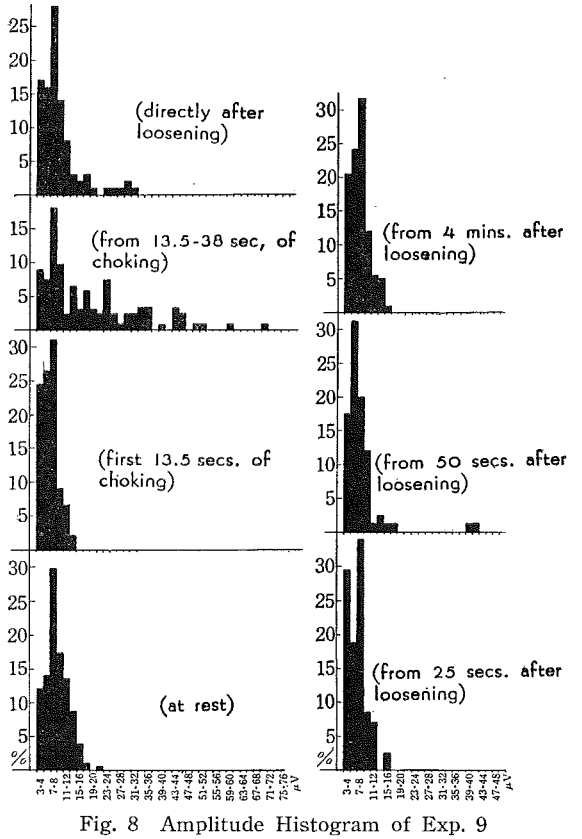


Fig. 8 Amplitude Histogram of Exp. 9

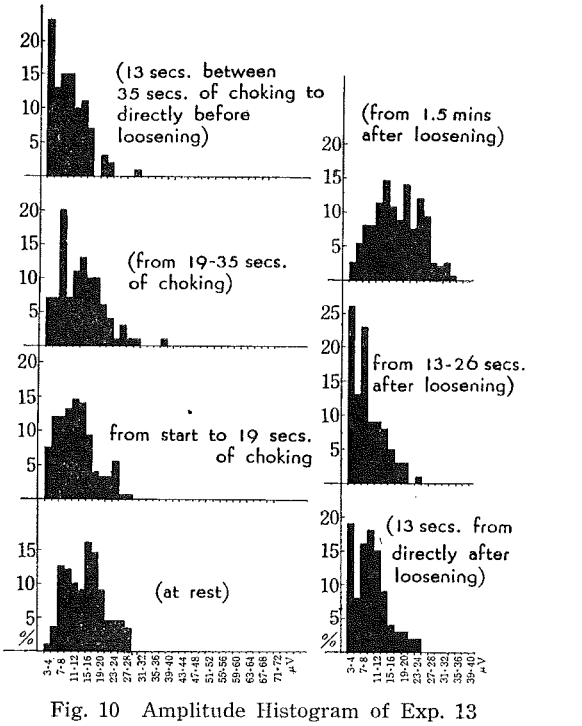


Fig. 10 Amplitude Histogram of Exp. 13

being the most numerous, and with an occassional appearance of 23-28 αV waves.

One minute after awakening the maximum frequency was 100 msec with 80-100 msec waves being numerous, and resembled the findings at rest. However, the appearance of 80 msec waves were more frequent than at rest, also 100 msec waves appeared less than at rest. The amplitude distribution was nearly identical to that at rest.

Four minutes after awakening the frequency distribution was very similar to that at rest, however the maximum was 90 msec. The amplitude after this period tended toward the increase.

(Exp. 10) Due to the small number of waves that appeared during the unconscious period the distributions of frequency and amplitude were irregular. The findings were somewhat similar to those of Exps. 7 and 8. However, the maximum frequency at rest was 80 msec and that 1.5 mins. after awakening was 90 msec, also the amplitude 4 mins. after awakening was nearer normal than 1.5 mins. after awakening.

(Exp. 9, did not lose consciousness) From the point of view of frequency and amplitude distribution the findings were somewhat similar to those of Exps. 7, 9 and 10, however the recovery time was somewhat slower.

(Exp. 13, did not lose consciousness) The distribution was somewhat different to that of Exp. 9. During the middle stage, i. e. from 19 secs. to 35 secs., the frequency distribution was somewhat similar to the unconscious stage of Exps. 7 and 8. From 13 secs. to 26 secs. after releasing pressure, the distribution was somewhat similar to that of Exp. 7, 8 and 10 during the awakening stage. From 1.5 mins. after releasing pressure, the distributions of frequency and amplitude neared that of the resting period.

The characteristic points in the amplitude distribution in Exps. 9 and 13 were, the appearance of two peaks at 3-4 μV and 7-8 μV, in the former from 25 to 26 secs., in the latter from 15 to 26 s$\frac{e}{o}$s..

c. Summary of Electro-encephalographic Findings.

The lead employed in this experiment was Berger's fronto-occipital method. With this lead the waves are said to appear very prominently, but in this experiment the amplitude was lower than that reported by others. A filter circuit was used, thereby eliminating waves with frequencies lower than 30/sec, and higher than 3/sec, and since large waves have distortion the action current of the muscles are eliminated. Taking into consideration the disturbances due to convulsions during the unconscious stage, and the opening and closure of the eyelids, the electro-encephalographic findings may be summarized as follows.

Changes in the 1 st stage. There was a period of from 1-3 secs. during the "choking" period marked by an appearance of low voltage waves prior to the appearance of high voltage slow waves. The frequency was short, but with an occasional appearance of slow waves. In those "choked" with method (1), this change was rather vague, and following a disturbance of the base line changes in the 2 nd stage made their appearance. In those subjects that did not lose consciousness whether "choked" by method (1) or (2), low voltage waves appeared, but were not very clear. The predominance of 3-6 μV in the amplitude histogram and 40 msec in the frequency histogram were all due to changes in the 1 st stage.

Changes in the 2nd stage. Following the 1st stage there was a stage marked by the

appearance of slow waves whose amplitude was not so high as that of the waves in the 3rd stage, and whose frequency was not so low as that of the waves in the 4th stage. Depending on the subject the amplitude was comparatively high in some, whilst in others it was not so high. Although the frequency differed greatly, the findings were those of the pre-unconscious stage.

Changes in the 3rd stage. Directly after losing consciousness there was a stage marked by the appearance of high amplitude slow waves. The frequency was higher, and the amplitude higher than the waves in the following stage. In Exp. 11 in which the subject did not lose consciousness completely, this change was seen for quite a long time.

Changes in the 4th stage. The frenquency was very low. This stage was marked by the appearance of slow waves, whose wave pattern was slightly rounded. The amplitude was lower than that of the waves in the 3rd stage.

Changes in the 5th stage. This is the stage nearing the awakening period, and marked by the appearance of α, β and δ waves making the pattern rather irregular. The amplitude and frequency gradually became similar to those at rest.

From the 3rd stage to the 5th stage, the subjects were unconscious, and the average frequency low and the average amplitude high, with minimal appearance of α waves. However, the occassional appearance in the histogram of waves with high frequencies, and also waves with amplitudes lower than 6 μV was due to changes in the 5th stage.

Changes in the 6th stage. This stage follows directly after awakening, and the wave pattern was rather irregular with the appearance of intermediate slow waves. The average frequency was still rather low and the average amplitude sometimes was lower than that at rest. In the amplitude histogram the high rate of appearance of waves of over 6 μV may be noted. The rate of appearance of α waves was low.

One minute after awakening. When "choking" was done with method (1), the findings returned to normal, however when it was done with method (2), although it appeared as though normal condition had been revived, the frequency histogram showed a few intermediate slow waves. The average amplitude was sometimes higher than at rest and at other times lower, although in the histogram the form was nearly that at rest. The rate of appearance of α waves was still rather low.

Four minutes after awakening. Normal findings were recorded, however the average amplitude was irregular.

d. Examination of the Electro-encephalogram

The changes appearing at the inception of "choking" may be considered due to biological artifacts. When "choked" with method (2), this change was sometimes not recorded and marked by a regular appearance of α waves, as a result the artifacts may be due to the movement around the neck caused by the "choking" method of method (1).

In regard to the diminution in the amplitude of the α waves, and the appearance of fast waves during the 1st stage, it is to be noted that Loomis, Harvey and Hobart (7) reported that in the early stage during sleep, although α waves still appear the amplitude gradually decreases. Adrean and Yamagiwa (8) also reported that during light sleep, the amplitude of α wave decreases, however the inhibition of α wave is not restricted to the

early stages of sleep, but as Berger and others have pointed out mental effort also inhibits α wave. Motokawa (9) summarizes the studies made by various workers on the relation of cerebral circulation and brain wave as follows:

slight congestion → slight anaemia → intermediate anaemia → cessation of circulation
(increase in frequency, decrease in amplitude) (slight change in frequency, increase in amplitude) (decrease in frequency, increase in amplitude) (no brain wave)

Furthermore, an excess of CO_2 in the blood will also influence electro-encephalographic findings and produces fast waves. Also, it is stated that the decrease in average amplitude and frequency is a manifestation of excitation of the cerebral cortex. During the 1st stage, the subject is aware that he is being "choked", and it is assumed that much mental activity takes place. On the other hand, from the point of view of the difference in elasticity between the arteries and the veins, there results a difference in time of closure between the two vessels. From the flushed appearance of the face this may be assumed to occur. Motokawa also states that when there is a circulatory disturbance in the brain, there occurs a marked manifestation of psycho-electric phenomenon, so when the subject is being "choked", the principle factor in the brain wave is mental excitement. As will be described later, in the experiment with rabbits, the electro-encephalogram at the time of closure of the jugular vein was very similar to the changes in the 1st stage. Hence, it may be assumed that changes in the 1st stage are due to mental excitement and congestion of cerebral vessels.

The similarity of the findings at this stage to those during a CO_2 excess condition, and also during the early stages of sleep may be interpreted thus: due to external conditions respiration becomes diminished and irregular, accompanied by muscular activity causing an increase in CO_2, and due to internal conditions, because of mental excitement, CO_2 is further increased. It is also reported (9) that during sleep, there is an increased blood flow in the brain and an increase in CO_2 tension in the cerebral arteries.

2nd stage. Jasper states that when there is an increase in the excitation of the cerebrum the frequency decreases, and conversely when there is a decrease in the excitation an increase in frequency results. Motokawa states that this may be applied to the condition of sleep. Although the condition of stupor→unconsciousness in "choking" is not exactly the same as that of sleep, Jasper's theory may be applied here, as in those subjects who did not lose consciousness, the changes following those of the 2nd stage became similar to the electro-encephalogram during sleep. Yeager and Walsch reported that when one of the carotid arteries was ligated during an operation, δ waves appeared and when the ligature was abolished the electro-encephalogram became normal. This condition is very similar to that of "choking" in which a decrease in oxygen occurs in the cerebrum.

3rd stage. It is simple when one considers the appearance of a not very low frequency, but high amplitude wave as an interference. However, in Robert Cohn's book (11), he describes a patient with a haematoma in the cornu occipitale of the right cerebral ventricle, whose electro-encephalogram was somewhat similar to the one during this stage. Also a similar electro-encephalogram was taken from a patient with convulsions as a sequelae to an encephalitis of unknown origin. Similar recordings were taken from patients with sequelae from a head injury. From the above it may be assumed that the changes at this stage are subcortical in origin. Similar electro-encephalographic findings are seen in epileptic patients. Since the changes in the 3rd stage sometimes appeared before the subject lost consciousness,

and when the subject was in a rigid condition, it is assumed that there is a relation to the onset of convulsions. At this stage. both the carotid arteries and the jugular veins are blocked, however the vertebral arteries are not. In this respect it is interesting to compare with Sano's experiment in which he induced convulsions in epileptics by intra-arterial injection into the vertebrals.

4th stage. The low frequency and high amplitude waves which appear during this stage also appear physiologically during sleep (Gibbs' F period); experimentally during hypoglycaemia, hypoxaemia, and deep anaesthesia; pathologically during epilepsy, brain tumor, abcess and haematoma. Robert S. Schwab (4) states that during stupor and unconsciousness, slow waves appear, and that anoxia is necessary for the state of unconsciousness. Naka (22) reports that during extreme lack of oxygen, frequency and amplitude both increase. The electro-encephalographic manifestations during this stage is assumed to be due to metobolic disturbances in the brain resulting from cerebral circulatory disturbances.

5th stage. At this stage cerebral circulation has already revived. Although during the near awakening stage of a physiological sleep, the abnormal metabolism gradually and smoothly returns to normal, in the case of "choking", when the pressure is released and cerebral blood flow revived, it is hard to say that the brain cells during this short period smoothly revive normal metabolism. The electro-encephalographic recordings during the awakening stage from a normal sleep recovers normal pattern regularly, however after being "chocked" the recordings recover normal pattern very irregularly.

6th stage. This stage corresponds to the stage directly after awakening, and is manifested by the appearance of α waves, although irregular. Furthermore, the appearance of intermediate slow waves makes this stage somewhat different from the awakening stage of a physiological sleep.

In Shimatake's (23) experiments, the subjects were made to inspire pure nitrogen and also low oxygen air. The electro-encephalographic recordings taken were very similar to those of the present author's, and especially those taken during the inspiration of low oxygen air were almost identical with the present work. The only difference was that in Shimatake's experiment the time required for the subjects to lose conciousness and fall into convulsions was longer.

From the above it may be assumed that the unconsciousness caused by "choking" is due to a disturbance in the cerebral circulation resulting in lack of oxygen in the brain.

Experiment with Rabbits

Electro-encephalograms of 7 rabbits were taken under the following conditions.

1) Application of pressure upon the carotid arteries with the fingers.
2) Strangulation by means of a manschette.
   (The following manipulations were done after exposure of the trachea and the blood vessels through an incission in the skin)
3) Application of pressure upon the trachea alone.
4) Closure of the jugular veins.
5) Closure of the common carotid arteries.
6) Closure of the jugular veins followed by closure of the common carotid arteries.

From the outward appearance those that showed rigidity were those of Exp. 2, and in none of the above was there any manifestation of clonic convulsions.

The normal electro-encephalographic findings of rabbits differ from that of normal humans, however in Exp. 4 the changes observed were similar to the 1st stage of "choking". In Exp. 4 the changes observed after closure of the common carotids were somewhar similar to those of the 2nd stage of "choking". The changes observed in Exp. 6 were the nearest to that of "choking", i. e. during closure of the veins, changes similar to the 1st stage were observed, and at the commencement of the closure of the arteries (10 secs. after closure of the veins) changes similar to the 2nd stage were observed. For 0.7-1.3 secs. following 12 secs. after closure of the arteries, the changes were somewhat similar to the 3rd stage, and subsequently for 2.0-4.0 secs changes were similar to the 4th stage, thereafter low voltage waves appeared.

Summary and Discussion

Popularly it is believed that loss of consciousness resulting from strangling the neck is due to asphyxia caused by closure of the trachea. Even at the Kodokan the strangling technique is called "choking" among the foreigners. However, the present paper shows that this loss of consciousness is not the result of closure of the trachea for the following reasons.

1) The differences in results obtained by method (1) and method (2). When the subject is "choked" by method (2) irritation of the trachea results indicating that the trachea is also pressed, whereas with method (1) no such symptoms are evident, however with both methods the subjects lose consciousness.

2) The time required for the subject to lose consciousness is very short. For example when an individual is completely submerged under water it takes longer for him to lose consciousness. Also the revival time in "choking" is very quick. Sato (24) closed the trachea of dogs and caused convulsions, however the time factor and the form of convulsions were different from the results in the present paper.

3) In the experiments with rabbits, the electro-encephalographic findings showed a similarity to "choking" when both the jugular veins and the carotid arteries were closed, but no similarity were observed when only the trachea was closed.

It is also of interest to know whether the loss of consciousness is the result of stimulation of the vagus nerve. If the vagus nerve were stimulated, there must be evidence of inhibition of the heart, however during "choking", symptoms similar to stimulation of the sympathetic nerves were observed, evidenced by tachycardia, hypertension and mydriasis. Waller, Czermak and Hering described the bradycardia resulting from pressure upon the carotid region as being caused by stimulation of the vagus nerve as a result of pressure on the nerve. Later Erben contradicted this theory and explained the phenomenon as being caused by the stimulation of the vagus nerve as a result of circulatory disturbance in the brain due to pressure upon the carotid arteries. Saito (25) after studying the results of many researchers and of his own explains the bradycardia and hypotension caused by the application of pressure upon the carotid region as being due to carotid sinus reflex, and further states that depending on the locality where the pressure is being applied, either bradycardia

or tachycardia results. It is natural that the role of the carotid sinus during "choking" cannot be overlooked. Experiments of ligature of the common carotid artery have been carried out by many. Siciliano (26) concludes that the tachycardia and hypertension resulting from ligation of the common carotid artery are due to a reflex action initiated by the abrupt drop in blood pressure within the common carotids. Saito attributes this drop in pressure within the common carotids as a decrease in pressure within the carotid sinus. During "choking", also the common carotids are closed causing a drop in pressure within the carotid sinus, and reflexly causes a rise in blood pressure, and tachycardia. Saito reports that of the 72 subjects very sensitive to pressure upon the carotid region, 9 lost consciousness and went into convulsions when pressure was applied. However, those who lose consciousness as a result of application of pressure upon the carotid sinus are only special cases, and those who lose consciousness after being "choked" are not always those individuals highly sensitve to pressure upon the carotid region. The loss of consciousness and convulsions in the above mentioned low oxygen air inspiration experiments of Shimatake, and Sano's intra-vertebral arterial injection experiments cannot be explained by carotid sinus reflex.

Furthermore, Exp. 6 (rabbit experiment) in which the electro-encephalographic findings were very similar to "choking" cannot be fully explained as being caused by carotid sinus reflex.

The unconsciousness resulting from "choking" cannot be considered as being in a state of shock as the symptoms are quite different from primary shock.

The author wishes to introduce the following reasoning as being appropriate. The main point in "choking" is the resulting unconsciousness. There are various theories advanced as to the locality of the centre of consciousness in the brain and nothing definite has been decided so far. Recently, there is a tendency to attribute the subcortical region around the ventricles as being related to consciousness, however Hayashi(27) (15) maintains that the centre of consciousness is limited to certain areas of the cerebrum. The aim of the present experiment, however is not to elicit the centre of consciousness in the brain, but to investigate how unconsciousness is brought about during "choking". According to Matsuda's report (28), the oxygen consumption of the brain tissue is approximately ten times that of the average of other tissues in the body, testifying the sensitivity of the brain tissue to lack of oxygen. At the inception of "choking", the jugular veins are first closed blocking the outflow of blood from the brain, causing a distension in the cerebral veins and resulting in a rise in intracranial pressure. A moment later (because of difference in elasticity of the vessel walls) the carotid arteries are closed, but the vertebral arteries are still intact and continues sending blood to the brain, further conjesting the cerebral veins, and increasing intracranial pressure. This causes the cells in the brain to become edematous, and electro-encephalographically δ waves are recorded. The brain of man receives the greater part of its blood supply through the internal carotids, and although communication is established between the internal carotids and the vertebrals through the circle of Willis, the drainage area of the two vessels are fixed. Consequently, when the carotids are blocked, the blood supply from the vertebrals to the drainage area of the carotids are not sufficient, resulting in anoxia of the cortex which according to Hayashi (27) (15) is the seat of consciousness.

If on the other hand as previously mentioned the centre of consciousness is located in the subcortical nucleus around the ventricles the following explanation may be given. Ac-

cording to some observers, when the intracranial pressure reaches a certain limit there occurs a great change in cerebral circulation. In "choking" as mentioned above there occurs a rise in intracranial pressure (cerebro-spinal pressure over 800 mm H_2O). When it reaches a certain point, the cerebral vessels begin to contract, inhibiting blood flow from the vertebral arteries. The contraction of cerebral vessels, according to Uchimura's theory is a causative factor of epileptic convulsions. Also, contraction of the cerebral vessels will result in lack of oxygen supply to the brain cells, and consequently causes unconsciousness.

The explanation for mydriasis, and increase in tendon reflex, both signs of stimulation of the sympathetic nerve is not quite clear. Forbes and Wolf (30) state that the cerebral vessels are contracted by stimulation of the sympathetic nerve, and also as it is known that the higher center of the sympathetic nerve is in the optic thalamus a disturbance in the circulation of the vertebral arteries will cause a stimulation. Or possibly, as a result of the congestion of the cerebral veins the sympathetic nerve is stimulated. As regards increase in tendon reflex, it is thought to be of pyramidal origin.

Conclusion

The unconsciousness resulting from "choking" in judo is mainly due to lack of oxygen and metabolic disturbance created in the brain, as a result of disturbance of cerebral circulation.

The symptoms are very similar to a very short epileptic seizure.

Aside from loss of consciousness and convulsions, symptoms indicating stimulation of the sympathetic nerve also appear, of which tachycardia and hypertension may be attributed to carotid sinus reflex.

As to whether "choking" is dangerous, it may be considered dangerous, and there has been an instance in which death resulted from "choking", although his condition, most probably pathologic, when he was "choked" was not ascertained. However, it may be considered less dangerous than a knockout in boxing, and there is no necessity of completely excluding "choking" from judo, provided necessary precautions are taken.

As to whether any deleterious after effects remain after being "choked", according to our experience there were none.

I wish to thank Profs. Takagi and Saito for their kind supervision and advice, and also Assist. Profs. Mizutani and Ito for their kind cooperation and encouragement. Finally, I wish to thank Mr. Morikawa who kindly supervised the electroencephalography, and also the other members of the institute for their kind assistance.

Abstract of a report by M. Morikawa:

1. In order to study the relation between the pressure applied to the neck and falling unconscious, a study was made by wrapping a manschette around the neck at height III-VC.. After resting for some time air was pumped into the manschette, thus "choking" the subject. Results obtained are in Table 1.

As seen in Table 1 the three subjects of experiments 1-10 lost consciousness when the

Table 1 Relation of pressure applied to neck and resulting unconsciousness

Exp.	Subject	Body wgt. (kg)	Blood press. at rest	Pres. applied to neck (mmHg)	State of consciousness	Time required to fall unconsc.
1	M. M.	49.2	syst. diast. 120— 60	240	+	5
2	〃	〃	〃	260	+	6
3	〃	〃	〃	240	+	5～ 6
4	〃	〃	〃	※ 280	+	12～13
5	〃	〃	〃	250	+	5～ 6
6	〃	〃	〃	240	+	6～ 7
7	T. ○	60.0	120— 60	280	+	5
8	〃	〃	〃	260	+	5～ 6
9	〃	〃	〃	280	+	7～ 8
10		70.2	115— 74	260	+	15
11		75.3	134— 82	over 300	+	37
12		57.5	144—110	〃	±	40
13		58.2	120— 58	〃	±	over 40
14		78.2	130— 70	280	−	
15		47.8	143— 90	300	−	

※ Voluntarily increased tension of cervical muscles
+ complete loss of consciousness
± incomplete loss of consciousness
− did not lose consciousness

air pressure in the manschette was raised to 240-280 mm Hg. The pressure necessary to cause unconsciousness was approimately fixed in the same individual, however when the neck muscles were under tension the time and pressure required for the subject to lose consciousness had to be increased (Exp. 4). More pressure and time were required for grade holders to lose consciousness than non-grade holders.

2. Electrocardiogram.

Exp. 4, supine position, lead II.

Findings:

P: While unconscious it was slightly elevated.

R: While unconscious, and also after awakening it was low.

T: While unconscious it was markedly low. The height of the crest was 1/2 that of the normal. After awakening it was still markedly low.

PP: The application of pressure markedly shortened its length.

3. CO_2 measurement in blood of internal jugular vein.

Blood was drawn from two subjects while at rest, and during the unconscious stage. The results obtained are in Table 2.

Table 2 CO_2 content in internal jugular vein

Exp. subject	Duration of unconsciosness	CO_2% at rest	CO_2% while unconscious
M. M.	16 secs.	49.7	49.3
T. ○	12 secs.	50.1	49.9

References

1) Shibusawa: Clinical aspect of shock, Igaku-sosho, 13, 87.
2) Hayashi: Cerebral cortex, Seirigaku-koza, 10, II, 4.
3) Araki: Head injuries, Nippon Geka-zensho 10, 8.

4) Araki : Saishin Igaku, S, 11, 1265.
5) Schiller, F. : Consciousness reconsidered, Arch. Neurol. & Psychiat., 67, 171, 1952.
6) Motokawa : Electro-encephhalography, pp. 40 & 209.
7) Loomis, A. L., et al : J. Exp. Psychol., 21, 127-144, 1937.
8) Adrian, E. D., et al : The origin of the Berger rhythm, Brain, 58, 323-351, 1935.
9) Aizawa : Nojunkan, 34, 101.
10) Yeager, C. L., et al : J. Am. Med Assoc., 114, 1625.
11) Robert Cohn : Clinical electro-encephalography, 1949.
12) Izumi, et al : No to Shinkei, 8, 1, 395.
13) Nakada, et al : Geka, 2, 1. 3.
14) Sano : No to Shinkei, 2. 1, 28.
15) Hayashi : No to Shinkei, 2, 5, 24.
16) Uchimura : No to Shinkei, 7, 6, 313.
17) Katsura : Nisshin Igaku, 32, 638.
18) Katsura, et al ; No to Shinkei, 4, 6, 20.
19) Shiozuki, et al : Geka-gakkai-shi, 55, 3, 322.
20) Uchimura, et al : No to Shinkei, 3, 1, 9.
21) Robert Schwab: Electro-encephalograpy in clinical practice.
22) Naka : Koku-igaku, 1, 280.
23) Shimatake : Seishin-shinkei-shi, 53, 4, 169.
24) Sato : No to Shinkei, 3. 5, 280.
25) Saito : Keidomyakukyu oyobi Doshinkei.
26) Siciliano : cited by Saito (25).
27) Hayashi : Cerebral cortex, Seiri-gaku-koza, 10, II, 4, 9.
28) Matsuda : Nojunkan, Seiri-gaku, 8, II.
29) Aizawa : Nojunkan, 101.
30) Fobes, H. S. & Wolf, H. G. : Neurol. Psychiat., 19, 1057, 1928.