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STATISTICAL ANALYSIS OF THE HUMAN STRANGULATION EXPERIMENTS: COMPARISON TO +Gz-INDUCED LOSS OF CONSCIOUSNESS

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In 1943, Rossen, Kabat, and Anderson (RKA), published the results of their investigations involving acute arrest of the cerebral circulation in man. Their studies on the effects of brief periods of cerebral ischemia have become a classic reference in a wide variety of scientific publications including those related to acceleration induced loss of consciousness (G-LOC). An accurate quantitative kinetic comparison of the RKA data and data from G-LOC could provide information concerning the possible similarities and/or differences in the mechanisms of the two phenomena. To this end, we attempted to relate the RKA work with current G-LOC theory by re-analyzing the RKA data in such a way that quantitative comparisons could be accomplished. The induction time for RKA loss of consciousness (LOC) was 6.4 to 6.9 s and found to be similar to both the equivalent parameter in G-LOC research of 8.8 s, and the time at maximum +Gz prior to LOC of 7.3 s. The RKA time from LOC to recovery was 5.9 to 6.4 s and shorter than the equivalent G-LOC parameter of 23.7 s. However, the predicted minimum G-LOC recovery time is 6.8 s. Further, the loss of consciousness syndrome encompassing numerous psychophysiologic symptoms are essentially equivalent for both types of

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ABSTRACT

In 1943, Rossen, Kabat, and Anderson (RKA), published the results of their investigations involving acute arrest of the cerebral circulation in man. Their studies on the effects of brief periods of cerebral ischemia have become a classic reference in a wide variety of scientific publications including those related to acceleration induced loss of consciousness (G-LOC). An accurate quantitative kinetic comparison of the RKA data and data from G-LOC could provide information concerning the possible similarities and/or differences in the mechanisms of the two phenomena. To this end, we attempted to relate the RKA work with current G-LOC theory by re-analyzing the RKA data in such a way that quantitative comparisons could be accomplished. The induction time for RKA loss of consciousness (LOC) was 6.4 to 6.9 s and found to be similar to both the equivalent parameter in G-LOC research of 8.8 s, and the time at maximum +Gz prior to LOC of 7.3 s. The RKA time from LOC to recovery was 5.9 to 6.4 s and shorter than the equivalent G-LOC parameter of 23.7 s. However, the predicted minimum G-LOC recovery time is 6.8 s. Further, the loss of consciousness syndrome encompassing numerous psychophysiologic symptoms are essentially equivalent for both types of insults. The results seem to suggest an analogous mechanism for LOC induction and recovery caused by strangulation (RKA) and +Gz stress.

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INTRODUCTION

In the past, research on the effects of positive acceleration (+Gz) in man was focused on the physiology of the cardiovascular system. During that time, acceleration induced loss of consciousness (G-LOC) was acknowledged, yet, its significance was overlooked. Recently, the understanding of the neurophysiology of G-LOC has become paramount in aeromedical research not only because of the danger G-LOC imposes on fighter aviation but because of the wealth of information a G-LOC episode imparts. Indeed, loss of consciousness and the psychologic phenomena associated with unconsciousness are not only a fighter aviation medicine problem but also represent research avenues that complement other scientific and therapeutic endeavors in clinical medicine.

In 1943, Rossen, Kabat, and Anderson (RKA), published the results of their valuable investigations involving acute arrest of the cerebral circulation in man (16). Rossen et al described the symptoms that occur as a result of abrupt interruption of central nervous system (CNS) perfusion. Their studies on the effects of brief periods of CNS ischemia have become a classic reference in a wide variety of scientific publications including those related to G-LOC. The RKA study was accomplished using a cervical pressure cuff known as the Kabat-Rossen-Anderson (kra) apparatus. The cuff inflated to 600 mmHg within one-eight of a second and deflated "within a fraction of a second." When placed around the lower portion of the human neck, it caused occlusion of the major vessels to the CNS (strangulation) and subsequent loss of consciousness (LOC). On the other hand, G-LOC results from reduced blood flow to the CNS as blood is displaced towards the abdomen and the extremities while the subject is under +Gz stress. The kinetics of G-LOC, its recovery, and the psychophysiologic phenomena resulting from this event have been identified (5,6,10,27,28); specifically those associated with rapid onset (>=1.0 G/s) +Gz profiles. Comparison of G-LOC and the episodes of unconsciousness described in the RKA paper provide an opportunity to examine human unconsciousness induced by different techniques. The RKA study is a valuable source of quantitative kinetic LOC data in healthy humans, and ideal for comparison with recently published G-LOC data. An accurate quantitative kinetic comparison of the RKA-LOC data and the data from G-LOC could provide information concerning the possible similarities and/or differences in the mechanisms of the two phenomena. To this end, we attempted to relate the RKA work to current G-LOC theory (21,24) by re-analyzing the RKA data in such a way that statistical comparisons could be

accomplished (T-test). The data under consideration were limited to mean, standard deviation (S.D.), range (R), and number of subjects (N).

METHODS

To utilize the results presented by RKA for comparison with current G-LOC parameters, we attempted to more thoroughly quantify the authors' data presented in section III of their paper. The RKA experiments were reported to have been conducted using 11 schizophrenic patients and 126 apparently normal male volunteers ranging from 17 to 31 years of age. The data of interest included: RKA Figure 1 entitled "Distribution curve for time to fixation of the eyes during acute arrest of cerebral circulation in 111 normal young men" (p 520). This bar-graph described time from occlusion of the cervical vessels to eye fixation; The RKA Table entitled "Consistency of Time to Fixation of Eyes on Repeated Tests on Different Days" (p 521). This table was associated with RKA Figure 1 and described the time to fixation of the eyes of repeated trials ("raw data") in 74 subjects; and RKA Figure 2 entitled "Distribution curve for time of recovery of the light-buzzer response following arrest of circulation to the brain in 28 normal young men" (p 522). The figure presented the average times from eye fixation to recovery of consciousness (repeated tests).

The RKA time periods were measured within 0.5 s with a stop-watch. Arrest of the CNS circulation lasted 5.0 to 10.0 s (p 513). The greatest number of subjects showed fixation of the eyes after 5.0 to 6.0 s of cerebral anoxia (pp 514, 524). LOC occurred 0.5 to 1.0 s after fixation of the eyes (pp 514, 521, 528) where the average time to LOC was 6.8 s (pp 527, 528). In the schizophrenic patients (supine position), arrest of circulation for periods as long as 100 s was accomplished (p 516).

To compare RKA results with G-LOC kinetic measurements (20,22), descriptive statistics of the RKA data had to be calculated. Three G-LOC variables were considered: **G-LOC induction time (LOCINDTI)** defined as the time from the onset of acceleration to the time the subject is observed to lose consciousness. For comparison purposes, it is necessary to combine the RKA time to eye fixation (RKALOC) with the RKA time from eye fixation to the onset of LOC (Xi = 0.5 to 1 s). Hence, the following relationship:

$$LOCINDTI = RKALOC + Xi$$

LOCINDTI encompasses the total +Gz acceleration period prior

to unconsciousness (time from +1Gz to G-LOC) and therefore is G onset rate dependent. Hence, an additional variable, the **total time at maximum +Gz (TAG)** prior to G-LOC, was considered.

The third G-LOC parameter considered was **G-LOC total incapacitation time (TOTINCAP)** defined as the time from loss of consciousness to response to a visual/auditory task. This time is equivalent to the RKA time of recovery of the light-buzzer response (RKAREC). However, the starting point for RKAREC was eye fixation, whereas for TOTINCAP is loss of consciousness. Again, in the RKA study, the time from eye fixation to loss of consciousness was Xi = 0.5 to 1 s. Hence, the following relationship:

$$TOTINCAP = RKAREC - Xi$$

Other events associated with loss of consciousness recovery were described in the qualitative portion of the authors' paper and shall be discussed later in relation to G-LOC phenomena.

RESULTS

Induction of unconsciousness RKA Figure 1 presented the time from the application of cervical cuff pressure to fixation of the eyes in 111 subjects. This figure is re-produced in Figure 1 where the estimated times are shown by each bar. The analysis of this figure required the following assumptions (see "derivation of the statistics" below): 1) N=111 subjects; 2) there was only one trial per subject; and 3) the time to fixation of the eyes presented in the x-axis was fixed at a discrete value. Under these assumptions, the resulting RKA time to fixation of the eyes (RKALOC) was: mean= 5.97 s, S.D.= 1.35 s, R= 6 s, N=111. For comparison purposes, LOCINDTI= RKALOC + Xi = 6.47 to 6.97 s (S.D. = 1.35 s)

The analysis of the information presented in the table associated with this figure was considered as a separate set of data because it described actual data on 74 subjects. The repeated trials were averaged per subject (Table I). Figure 2 is a graphical presentation of these computed averages. The resulting RKALOC mean time was similar to the mean time of Figure B where: mean= 5.91 s, S.D.= 1.39 s, and R= 7 s. For comparison purposes, LOCINDTI = 5.91 + Xi = 6.41 to 6.91 s (S.D. = 1.39 s).

Derivation of the statistics: The data given above were based on careful analysis of RKA Figure 1 and the associated table. In arriving at the statistics described above, we encountered

the following challenges: 1) When determining the number of subjects that exhibited each particular time to fixation of the eyes, we were unable to obtain a total of 111 subjects except by compromising our accuracy; 2) It was not clear if the data presented in the figure comprised repeated trials where the number of repeated trials was 1 to 4 (inferred from the associated table). Hence, we could not establish if all repetitions were used (an average of these trials) or if any particular trial was considered (or was not considered) in producing the bar-graph. This question was especially difficult to solve when considering the authors had stated 85 subjects had undergone repeated trials, 74 subject data were shown in the table, 111 subject-data were shown in the figure, and 126 subjects were used in the study; and 3) we could not ascertain if the time to fixation of the eyes shown in the figure was presented as time intervals where periods under 4 s and over 10 s were not included or if the times were fixed, in which case, the table would not necessarily correlate with the figure. Given these limitations, the data presented in the table (N=74) were used for the statistical comparisons with current G-LOC parameters.

Recovery of consciousness RKA Figure 2 presents the time from eye fixation to recovery of consciousness (RKAREC) in 28 subjects. This figure is re-produced in Figure 3 where the estimated times are shown by each bar. The mean RKAREC was 6.89 s, S.D.= 2.51 s, R= 8 s, N=28. For comparison purposes, the TOTINCAP was RKAREC - Xi = 5.89 to 6.39 s (S.D. = 2.51 s)

Derivation of the statistics: There was no avenue to relate the RKAREC resulting from a specific RKALOC episode given the data within the manuscript: 1) the number of trials accomplished per subject was not specified; 2) the subjects from RKA Figure 1 (RKALOC) which were presented (if any) in RKA Figure 2 (RKAREC) were not specified; 3) every other time interval was not included in the figure; and 4) only 28 subject data were presented and therefore available for comparison analysis.

DISCUSSION

In order to accurately compare the RKA results with current G-LOC research, it is necessary to examine the onset and offset rate of the "vehicles of unconsciousness" under discussion (kra apparatus and +Gz acceleration stress).

Onset rate The kra apparatus occludes the major vessels to the CNS within 0.125 seconds whereas the onset rate of current G-LOC data (rapid onset rate exposures) obtained in the laboratory (human centrifuge) is 2.8 to 7.6 G/s to peak +Gz

levels ranging from +7-9 Gz. The induction time to unconsciousness is similar in both experimental situations: RKA= 6.4 to 6.9 s and +Gz= 8.8 s (28); However, onset rate (0.07-3.69 G/s) in the acceleration environment affects LOCINDTI where LOCINDTI exhibits an inverse relationship with onset rate and the predicted minimum LOCINDTI is 7.26 s (26). This value is not significantly different from the RKA data of 6.4 to 6.9 s (p = .18 and .57 respectively). Onset rates greater than 1.25 G/s do not seem to cause any further marked reduction of LOCINDTI. This finding suggests a similar mechanism of unconsciousness for both kra strangulation and G-LOC.

Some of the available literature suggest that +Gz level does not play a role on the time required at +Gz to induce unconsciousness. Cochran et al (3) found that TAG prior to G-LOC was 7.5s (S.D. = 1.5 s) for +Gz levels ranging from below 3.5 to 7.0. The time to attain maximum +Gz during these exposures ranged from 3.9 to 6.3 s. Whinnery et al (28) found TAG to be 7.29s (S.D. = 3.01 s) for peak +Gz levels ranging from 7 to 9 (mean = +8.5 Gz S.D. = 0.72). These values were not significantly different from Rossen et al data. Franks et al (7) found that ear opacity decreased to a minimum 4 to 6 seconds after peak +Gz was reached and was not quantitatively related to the amount of G applied. Suffice it to say that a serious reduction of blood flow to the brain will probably cause unconsciousness within 7 seconds.

It is difficult to define "serious reduction" both qualitatively and quantitatively; the kra apparatus essentially occludes the vessels to the CNS ("trapping" the blood in the head), whereas +Gz acceleration actively reduces blood flow to the CNS as the subject is accelerated. It is unclear exactly how much blood (i.e. oxygen) is actually available to the tissues during these two types of insults, or exactly how a lack of blood flow actually causes unconsciousness. Henry et al (11) found that consciousness was lost when mean cerebral blood pressure fell below 25 mmHg and that a mechanism which compensates for the fall in cerebral arterial pressure induced by +Gz was evident. He further stated that significant deep channels which can remain patent in spite of subatmospheric pressures are available for the return of blood to the brain. Howard (12) explained the development of markedly sub-atmospheric pressures in the jugular veins at high +Gz levels ensures that the fall in arterial pressure is counter-balanced by the formation of a syphon so that "blood is sucked through the brain...adequate perfusion is accordingly preserved at levels of acceleration greater than would be predicted by hydrostatic theory alone, and consciousness is maintained until collapse of the jugular veins breaks the syphon". Krutz et al (14) found that the onset of zero forward blood flow in the temporal artery coincided with a reduction in mean arterial pressure to 20 mmHg and that this reduction occurred 4-9 s prior to blackout. Wood et al (29) found that the average latent period after the

onset exposures to accelerations greater than +3Gz was 6.8 s (3.5-9 s) suggesting that G-LOC is caused by a sudden acute stoppage or near stoppage of cerebral blood flow as that caused in Rossen et al subjects. Glaister (8) found that there is less blood in the brain during +Gz and this reduction is proportional to the reduction in HbO_2. Further, the disappearance of the blood and the eventual level achieved is proportional to the +Gz level achieved. Burton (2) has noted that G-LOC is not a problem related to blood oxygen content per se but rather a problem of getting blood to the appropriate places in the body. Sandler et al (17) found that a minimum of 6 seconds of total brain blood flow cessation was necessary before black-out occurred and that cessation of flow correlated consistently with loss of peripheral vision. Further, it has been demonstrated with lower body negative pressure studies (9) that subjects exhibiting syncopal symptoms show a reduction of cerebral perfusion pressure suggesting a reduction in cerebral blood flow. Transcranial doppler methodology has also been utilized to determine cerebral blood flow in the +Gz environment (15,18) where cerebral blood flow velocity was calculated to be reduced by up to 58% during certain +Gz profiles.

The question of blood availability to the brain has been addressed in terms of the mechanism of G-LOC (21,24). This mechanism is based on the observation of symptoms resulting from G-LOC and the time sequence of these symptoms. In essence, when blood flow to the CNS is reduced by +Gz stress, ischemia/anoxia occurs in a top to bottom, watershed pattern based on the CNS circulatory system. To ensure maximum survival, the neurons optimize energy conservation by minimizing extracellular activity. This local inhibition reduces electrical output to other neurons and neuronal metabolic expenditure. When a critical mass of locally inhibited neurons is attained, the inhibitory reticular formation becomes disinhibited and gains control of the CNS through induction of global inhibition. The onset of this global inhibition is loss of consciousness, an active mechanism to protect the integrity of the CNS. The process above is termed the functional buffer period or loss of consciousness induction time (LOCINDTI). As blood flow returns, the neuronal inhibition is reduced and segments of the CNS become sequentially re-activated. The CNS regains function beginning with the primitive system, and progressing toward the cortical system. The proposed mechanism is basically a concerted effort to protect the CNS from injury.

Offset rate The kra apparatus deflates within 1 second after unconsciousness occurs, whereas, subjects experiencing G-LOC in the centrifuge are currently decelerated within 2-8 seconds depending on the acceleration profile and +Gz level the subject achieved prior to G-LOC. Clearly, the resulting total incapacitation R.K.A = 5.9 to 6.4 s; G-LOC = 23.7 s is

affected by this offset rate. The longer the time to restore adequate CNS perfusion, the longer the period of incapacitation. It is important to note, especially when considering G-LOC recovery, that under the conditions of the RKA study, the time to regain consciousness (TOTINCAP) was 5.9 to 6.4 seconds when the insult (vessel occlusion) was abolished within 1 second. G-LOC research suggests that offset rates greater than 2.0 G/s offer very little reduction of the absolute incapacitation period resulting in a predicted minimum absolute incapacitation period of 6.83 s (19). Unfortunately, we were not able to quantify the exact period of absolute incapacitation (subject is clearly unconscious), from the Rossen et al data, or more specifically, the period of relative incapacitation (confusion, apathy, temporary paralysis). Therefore, the effect of offset rate and time of unconsciousness was not available for comparison with G-LOC research findings.

Figure 4 describes the comparison of the loss of consciousness incapacitation times during G-LOC and kra apparatus induced strangulation. Howard (13) discussed an interesting experiment by Beckman et al (1) where the induction time to "unconsciousness" was found to be approximately 4 s once the subject had reached +3-5 Gz. These +Gz levels were assumed to be the point at which cerebral circulation failed (based on oxygen saturation at ear level). This time was found to be significantly smaller than the one found by RKA. This difference was explained based on the trapping of blood in the head caused by strangulation whereas "in the centrifuge experiments there is an active draining of blood from the brain, and the oxygen reserve is thereby diminished". Indeed, this active draining of blood from the brain begins when +Gz begins. Hence, the time to loss of consciousness found by Beckman et al of 4 seconds is a rather conservative figure since its measurement was based on +4Gz as a base G level (load in G being 3 G). The reasoning behind this base +Gz level was the assumption that cerebral circulation is stopped by an accelerative stress of 3-5 G. The Beckman et al study is probably not ideal for comparison with RKA data since Beckman's subjects did not experience unconsciousness as defined by the G-LOC literature or RKA data: The electroencephalogram did not show any significant changes; of the 11 subjects used for the study, those subjects who experienced confusion and those who experienced unconsciousness per se were not specified; and no loss of motor control or convulsive movements were evident in Beckman's subjects. The task utilized to herald loss of consciousness in Beckman's study was a continuous response to light and buzzer signals until the subjects were told to stop. Unfortunately, this task required the subjects be holding the button/switch (not-specified) throughout the experiment. An occasional result of loss of consciousness is the subject continuing to hold whatever instrument or apparatus he/she is holding rather than releasing it upon unconsciousness (4). This end-point discrepancy (acknowledged by Beckman) might be

the reason why it was suggested there was a significant difference between the time-course of the unconsciousness produced by acceleration and that which occurs when the cerebral circulation is arrested by other methods. Whether loss of consciousness is induced by +Gz or strangulation, the mechanism seems to be similar in both insults: Protective mechanisms are activated when some threshold is reached. This threshold is based on oxygen (and/other energy substrates) consumption or availability. It is reasonable to believe that a unique "consortium" such as the human body will protect itself prior to any imminent lack of energy/support occurring rather than wait until no further sustenance is available.

Symptoms Loss of consciousness is not the only symptom resulting from these studies. It is for this reason the entire symptom complex has been described as the G-LOC syndrome (23,25). A summary of the symptoms common to both methods of arrest of the cerebral circulation sufficient to induce unconsciousness is in table III.

The relationship of the subject's state of mind prior to each trial was addressed in Rossen et al paper where recovery from unconsciousness (RKAREC) varied from subject to subject and trial to trial. This may be the reason why the authors were not able to determine a relationship (if any) between RKALOC (resistance to anoxia) and RKAREC. Also, as stated by the authors, "The time to fixation of the eyes, as a measure of resistance of the cerebral neurons to anoxia, might be expected to correlate with the time of recovery." However, only if that time of recovery is measured from the onset of unconsciousness to return of consciousness (end of absolute incapacitation) can this comparison be accomplished. The recovery times that Rossen and co-workers measured (RKAREC) appear to be the summation of both absolute (unconscious/"asleep") and relative incapacitation (period of confusion, eyes open, apathy, until subject responds to the buzzer). Relative incapacitation is highly influenced by subject characteristics (mainly psychological as RKA stated). Hence, this correlation was difficult to ascertain given the data available for consideration.

The psychologic sequelae of G-LOC are very similar to those described in Rossen et al paper, and continues to be quantified in G-LOC research. These phenomena and their effect on loss of consciousness recovery times are of special significance when recovery methods (interaction with the subject / aircraft recovery) are considered. Rossen et al found that psychologic factors play an important role in recovery from unconsciousness. Psychophysiologic reaction to the loss of consciousness episode itself and its possible consequences (dream, transient paralysis) defines, to a large degree, the period of relative incapacitation (5,6).

Figure 5 describes the sequence of events during loss of consciousness and recovery for the schizophrenic patients

whose CNS insult was considerably greater (up to 100s of altered perfusion). This duration of ischemic insult, although shown to be completely safe by the RKA experiments, far exceeds the ischemic insult duration of the acceleration experiments. These experiments do illustrate the effect of prolonged CNS ischemia on the kinetics of convulsive activity.

Since the time for inducing unconsciousness by rapid occlusion of the cervical vessels supplying the CNS (strangulation) and by preventing blood from reaching the CNS (+Gz stress) are very similar, it appears that a similar mechanism for the alteration of normal CNS function supporting consciousness exists for these two techniques. This strongly supports an ischemic etiology for +Gz induced unconsciousness and recovery. In addition, it appears that there is little if any advantage for preserving or recovering consciousness gained by having residual blood trapped within the CNS (as would be present in strangulation only). As the techniques involved in centrifuge studies more closely approach the techniques of RKA, the kinetics of the induced unconsciousness and recovery appear to approach very similar values. These findings have significant implications regarding the precise mechanism by which consciousness is lost and subsequently regained. The comparison illustrates the exquisite sensitivity of the CNS to lack of an adequately oxygenated supply of blood. It also emphasizes the importance of an accurate description of not only the technique to induce unconsciousness/restore consciousness but also the measurement parameters used to describe the quantitative symptomatologic kinetics of this insult. Unfortunately, the valuable G-LOC research done in the past (published literature) described the event in a qualitative manner. To accurately describe the G-LOC syndrome, it is imperative to (a) define the G-LOC syndrome; (b) provide a detailed description of the +Gz environment, the measurements, and measurement techniques used to describe the event; and c) perform accurate statistical analysis of these data. Current research efforts attempt to accomplish this task.

SUMMARY

The excellent, pioneering work of Rossen, Kabat and Anderson on arrest of the cerebral circulation was examined in an effort to more accurately relate acceleration induced loss of consciousness (G-LOC) with loss of consciousness induced by strangulation. Table II comparatively summarizes the parameters analyzed. The induction time for RKA loss of consciousness to occur was 6.4 to 6.9 s (S.D. = 1.4 s and was found to be similar to both the equivalent parameter (LOCINDTI) in G-LOC research of 7.26 s (S.D. = 2.7 s), and the time at maximum +Gz prior to unconsciousness (TAG) 7.3 s (S.D.

= 3 s). The time from loss of consciousness to recovery (RKAREC) was 5.9 to 6.4 s (S.D. = 2.5 s) and found to be significantly shorter than the equivalent parameter (TOTINCAP) in G-LOC research of 23.7 s (S.D. = 9.9 s). This difference was partially attributed to the offset rate (rate of return of blood flow to the CNS) of the insult, where the kra apparatus was deflated within 1 second, and typical +Gz profile offset rates encompass 2-8 seconds. The predicted minimum +Gz stress recovery time has been found to approach 6.83 s. These induction and recovery times are very similar and given the differences in experiment techniques for measuring the parameters, there appears to be no compelling reason to suggest that the induction and recovery times are different for the two different methods of inducing unconsciousness in healthy humans. The additional symptoms of the RKA loss of consciousness syndrome are essentially the same as those for the G-LOC syndrome. The opportunity to examine a classic paper such as Rossen, Kabat and Anderson work has provided additional insight to the theory of unconsciousness. The wealth of data contained in their publication is an incentive to further elucidate the nuances of the loss of consciousness phenomenon.

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Table I. Mean Time (s) to Fixation of the Eyes in 74 RKA Subjects from 1-4 Trials (EYE1-EYE4) as Taken from the Data in the Original Table.

TRIAL	EYE1	EYE2	EYE3	EYE4
MEAN	5.7	6.0	5.7	6.2
S.D.	1.4	1.4	1.0	1.3
MIN	3.0	4.0	4.5	5.0
MAX	10	12	9.5	9.5
N	74	74	47	15

EYE1-EYE4: Four trials

Table II. Relationship of RKA (kra Apparatus Strangulation) and GLOC Parameters.

Legend: According to Rossen et al[9], Cochran et al[2], Houghton et al[6], and Whinnery et al[12,19,21]

ONRATE = onset rate
OFFRATE = offset rate
STRESS = type of insult
TAG = tune at +62
USAFSAM = School of aerospace medicine
NAD = Naval Air Development Center (NAWC-AD)

	kra		+Gz			
VARIABLE	Rossen et al	Cochran et al	Houghton et al	Whinnery et al		
ONRATE	.125 ( ) s, 126	3.9-6.3 s, 935	4 ( ) s, 8	predicted 1.25 G/s	2.3 ( ) s, 107	USAFSAM 2 s {3.8 G/s}, 90 NADC 2.5 s {3.1 G/s}, 17
OFFRATE	< 1.0 ( ) s, 126	_____	_____	predicted 2.0 G/s	7.5 ( ) s, 107	USAFSAM 8 s {.97 G/s} NADC 2.8 s {2.75 G/s}
STRESS	600 mmHg	<+3.5-7.0 Gz, 935	+6.1 (0.7) Gz, 8	+3.0-9.0 Gz	+7.0-9.0 Gz	+8.0-9.0 Gz
TAG	_____	7.5 (1.5) s, 935	_____	_____	7.3 (3) s, 107	_____
LOCINDTI	6.4-6.9 (1.4) s, 74	_____	9.2 (1.4) s, 8	predicted min 7.26 s	8.8 (2.69) s, 107	USAFSAM 10.22 (3.05) s NADC 8.7 (2.43) s
ABSINCAP	_____	_____	12.1 (5.04) s, 8	predicted 6.83 s	10.68 (3.36) s, 105	USAFSAM 10.47 (3) s NADC 7.59 (3.14) s
RELINCAP	_____	_____	11.6 ( ) s, 8	_____	13.06 (9.94) s, 101	USAFSAM 14.4 (10.05) s NADC 5.4 (3.38) s
TOTINCAP	5.9-6.4 (2.5) s, 28		23.7 ( ) s, 8	_____	23.68 (9.96) s, 103	USAFSAM 25.04 (10.13) s

Table III. Common Symptoms for Both Arrest of the Cerebral Circulation Methods.

COMMON TO BOTH METHODS		NOT COMMON
VISUAL:	* eye fixation * constriction of visual fields * turning up of eyes	* blurred vision * scotomas
MOTOR:	* anoxic convulsions * tingling of extremities * tonic/clonic convulsions * convulsions during blood flow return * LOC= head/body slump * transient paralysis/frozen hand * individual muscle twitching	* shooting pains (prior to LOC)
OTHER:	* no excess salivation * no tongue biting	
PSYCH:	* amnesia * confusion * foolish smile * excitation\euphoria * apathy * denial of LOC	
EEG:	* delta waves upon eye fixation/LOC	
ECG:	* slight drop in heart rate or no change	
LOC:	* induction time: 6-8 s * No correlation between recovery and incapacitation time	

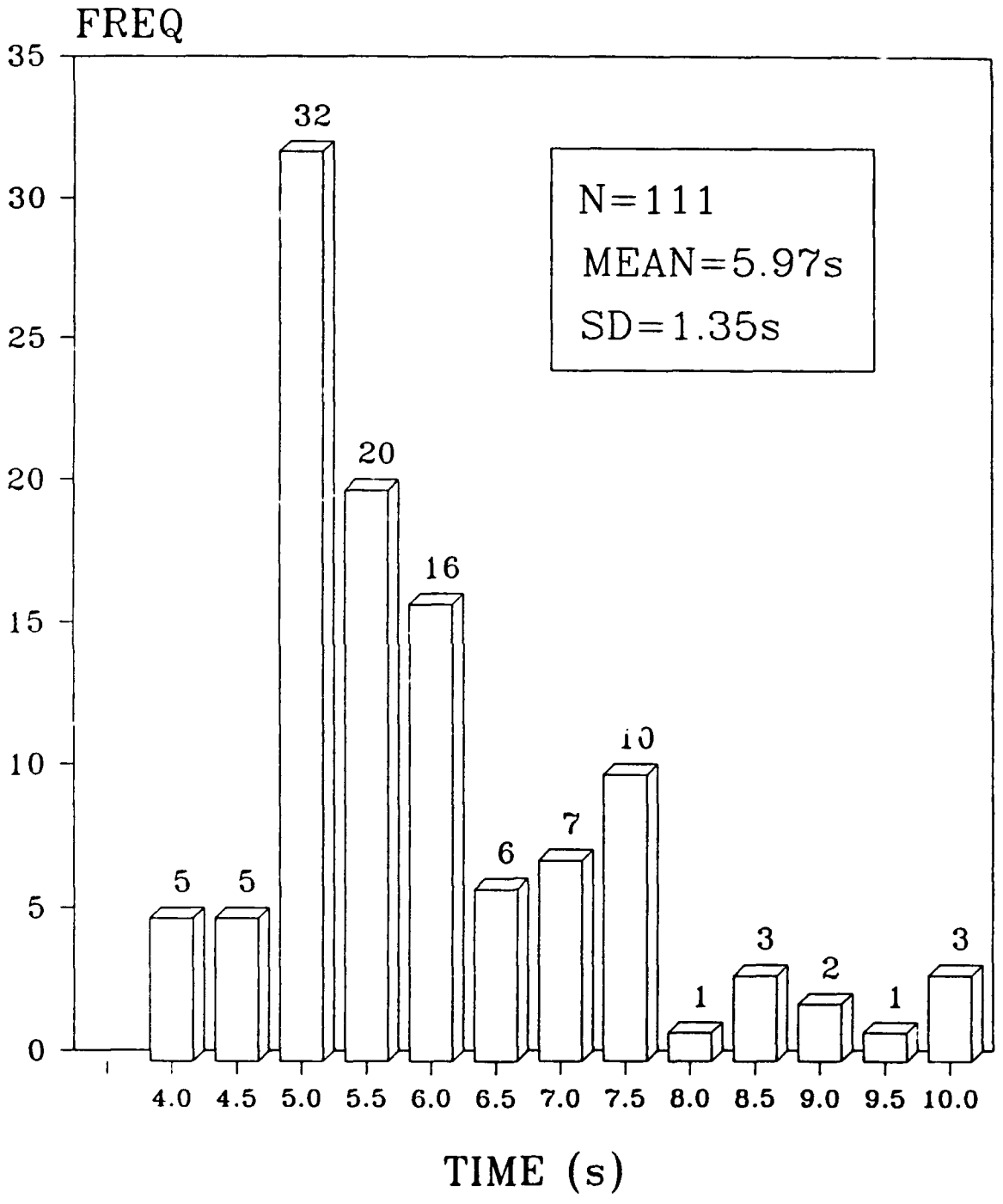

Figure 1. Reproduction of RKA Figure 1: Time to Fixation of the Eyes (RKALOC).

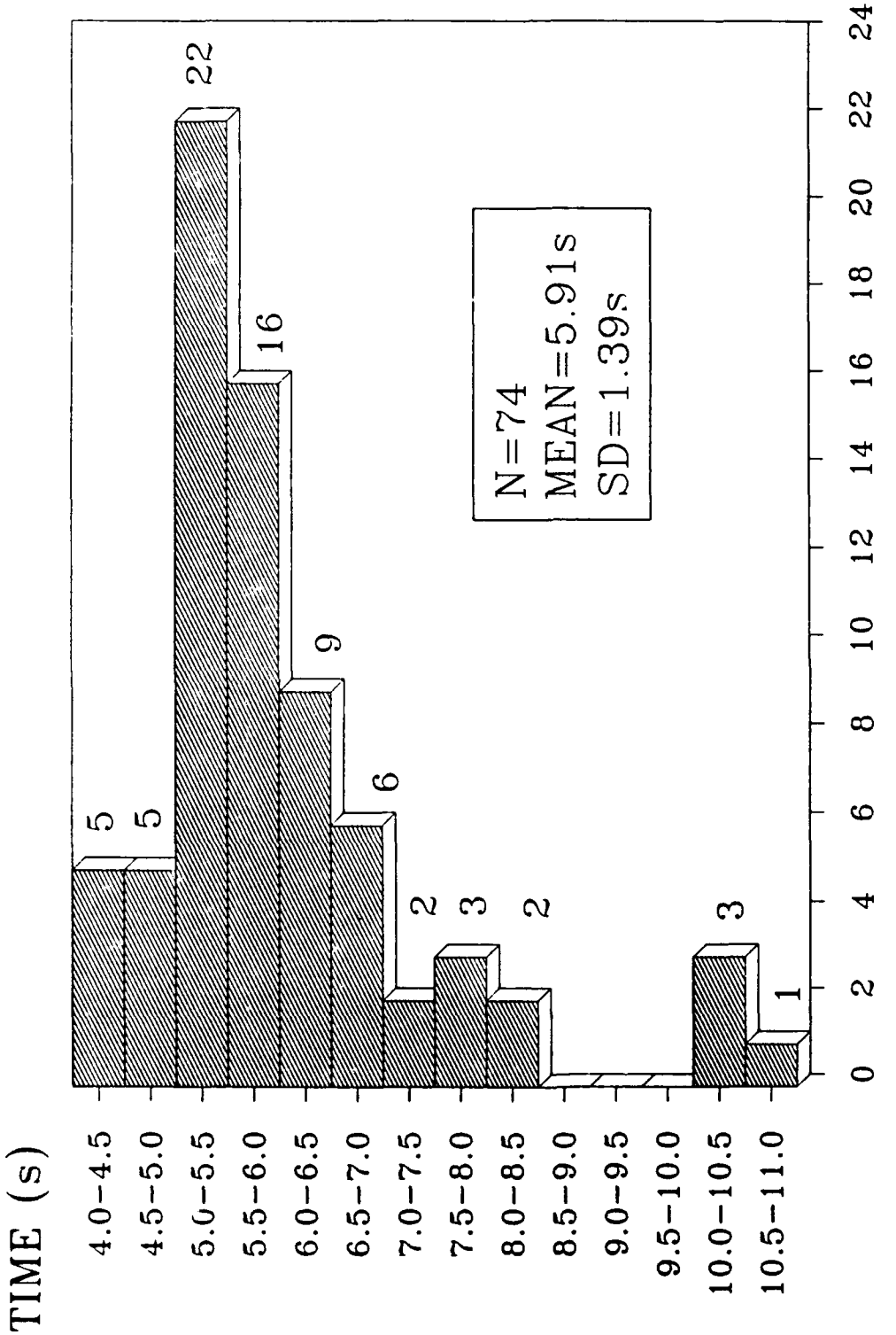

Figure 2. Summary of RKA Table Associated with RKA Figure 1: Time to Fixation of the Eyes (RKALOC).
Legend: Computed Average RKALOC of 1 to 4 Trials in 74 Subjects.

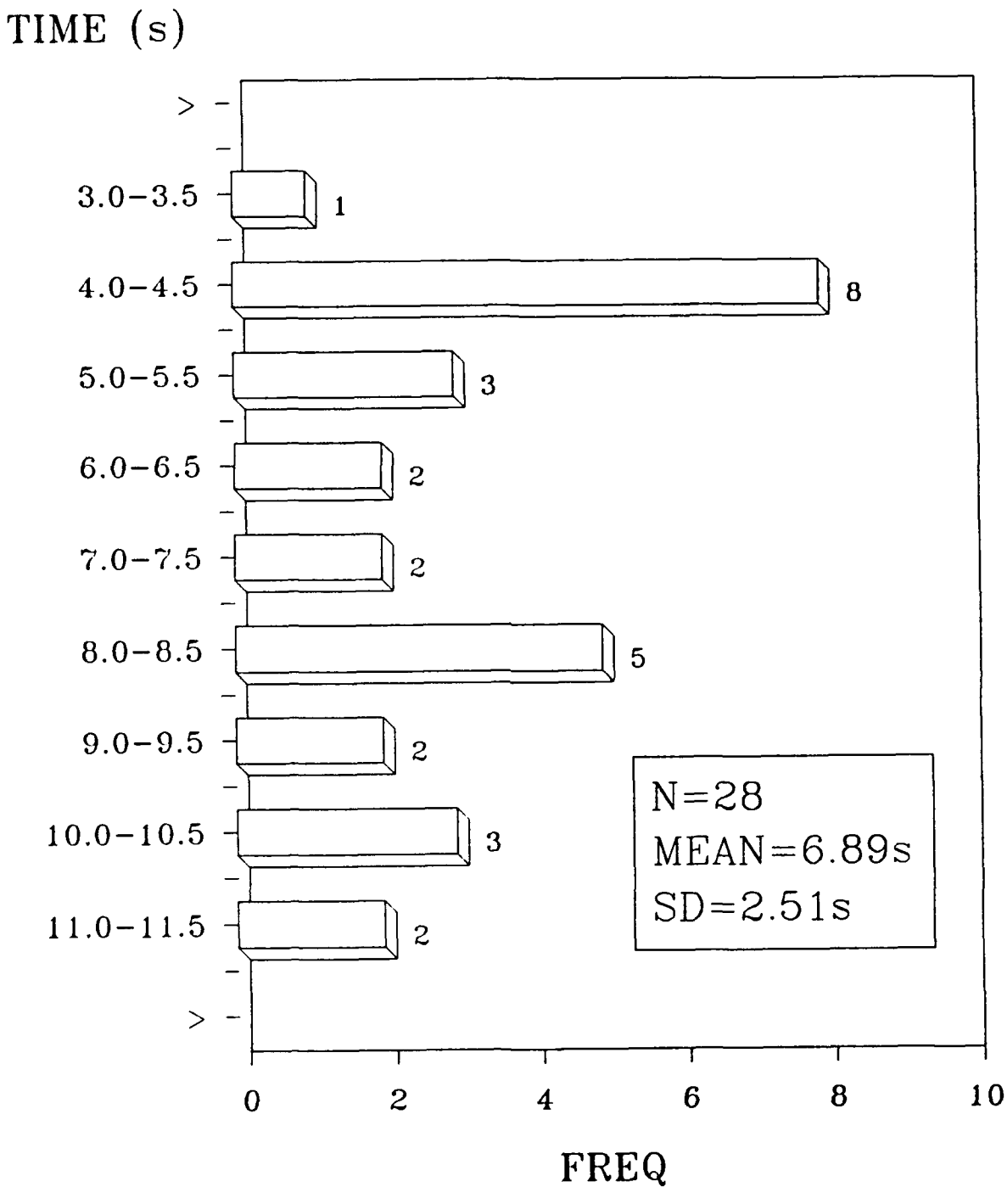

Figure 3. Reproduction of RKA Figure 2: Recovery of Consciousness (RKAREC).
Legend: 28 Subjects. Frequency Estimates are Shown by Each Bar.

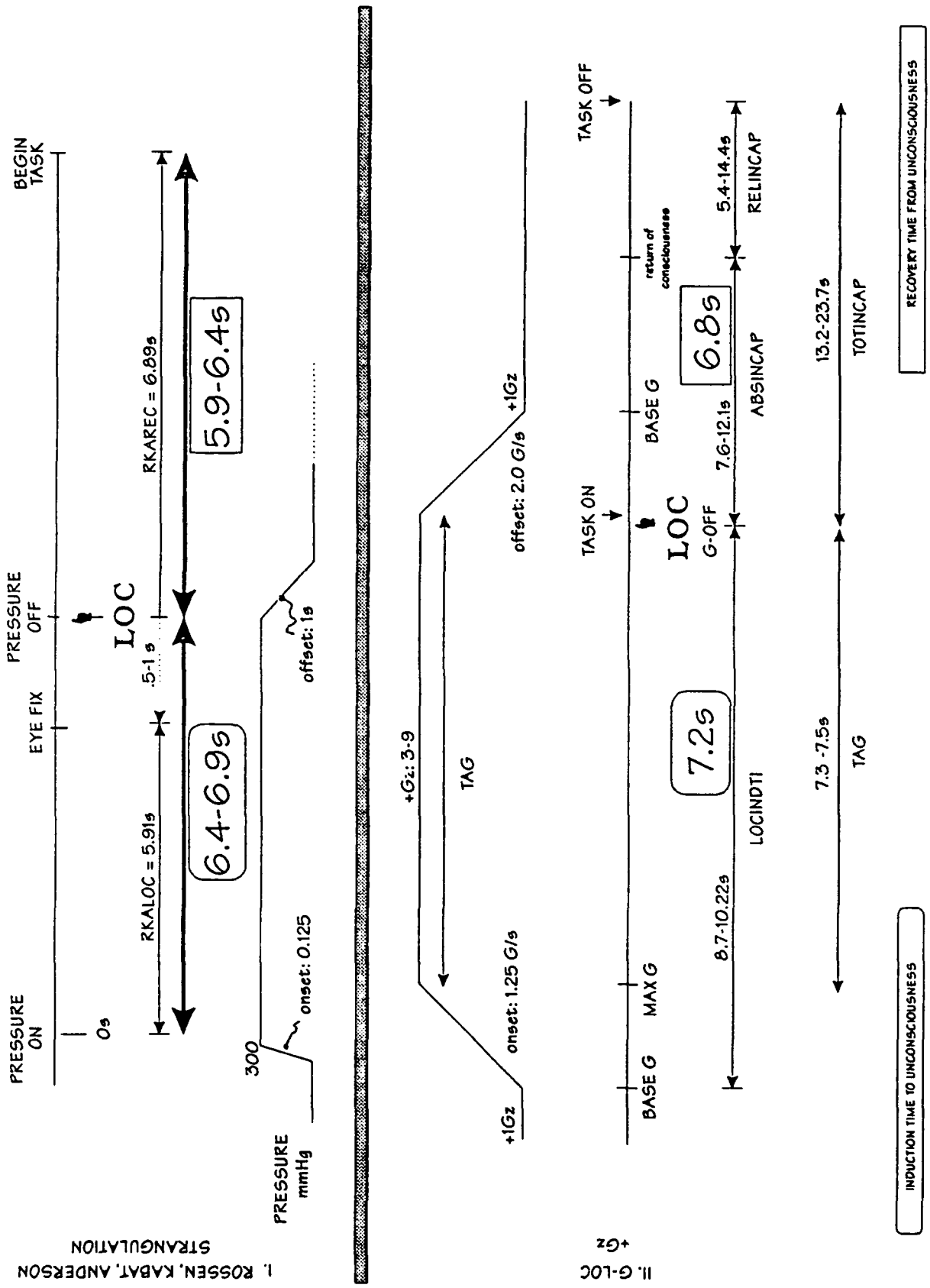

Figure 4. Comparison of RKA and G-LOC Parameters.

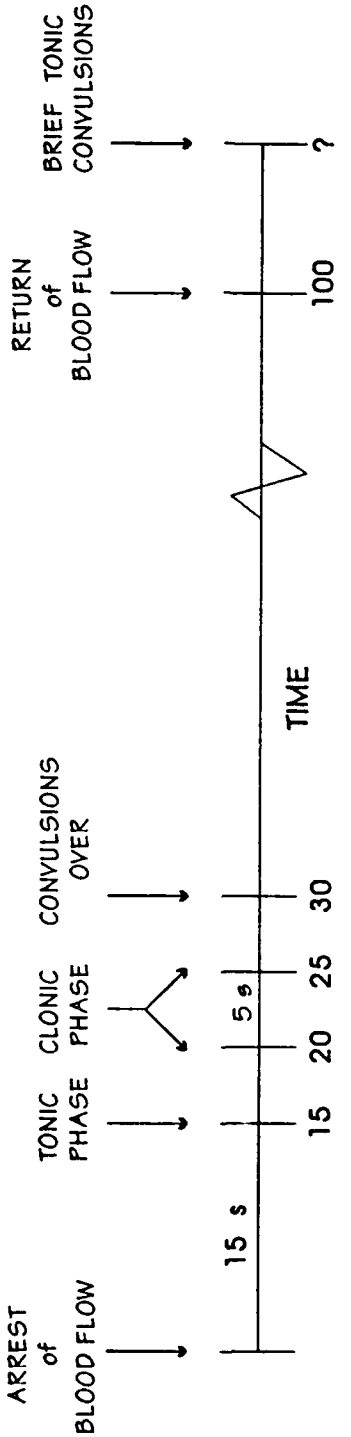

Figure 5. Patient Studies Sequence of Events.

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