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# NON-LINEAR ASCENT PROFILES REDUCE THE RISK OF DECOMPRESSION ILLNESS AFTER DEEP NO-STOP DIVES

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The experiments reported herein were conducted according to the principles set forth in the current edition of the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Animal Resources, National Research Council.

This technical report has been reviewed by the NMRI scientific and public affairs staff and is approved for publication. It is releasable to the National Technical Information Service where it will be available to the general public, including foreign nations.

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The influence of no-stop ascent profile shape on decompression illness (DCI) risk after deep air and heliox dives was investigated using a swine model of neurological DCI. Following a simulated dive to 200 fsw for 24 min bottom time, while breathing air, pigs were decompressed over 10 min at either a linear 20 fsw/min, or on a non-linear fast-deep/slow-shallow profile. In the linear group, there were 11 cases of neurological DCI including 1 death and 8 cases with severe features, compared to 5 neurological DCI cases (1 severe) in the fast/slow group. 13/20 of the linear group versus 6/20 had moderate or severe skin DCI affecting >20% skin surface area. A similar study, but of paired, randomized, investigator-blind, sequential design was performed with pigs breathing 80/20% heliox. Pigs dived to 250 fsw for 8 min 50 s, then decompressed at either a linear 30 fsw/min rate, or on a fast/slow profile. Neurological DCI occurred significantly ($p = 0.024$) more frequently in the linear group (16/20; 1 death and 11 severe) than in the fast/slow group (8/20; 3 severe). Moderate or severe skin DCI affected 16 of the linear group compared to 3 of the fast/slow group ($p = 0.0002$). The study findings suggest that, for deep no-stop diving, a non-linear fast-deep/slow-shallow ascent profile is safer than a linear rate of ascent, irrespective of breathing gas. This finding has the potential to reduce the risk of both military and civil diving operations.

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The experiments reported herein were conducted according to the principles set forth in the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Animal Resources, National Research Council, DDH, Publ. No. (NIH) 85-23.

## INTRODUCTION

We do not know the optimum decompression profile to maximize safety for a diver returning to the surface after a no-stop dive. However, on theoretical grounds the currently used "linear" ascent profiles (1) may be less than optimum: Bubble volume is inversely proportional to the absolute pressure (Boyles Law) and if a plot of pressure against bubble volume is made the resulting curve is hyperbolic, not linear. Consequently, as it is known that all but the most trivial dives cause bubble formation in the divers' body during decompression (2), it seems reasonable to hypothesize that a diver might minimize the growth of bubbles in his/her body, and thus be safer, by following a hyperbolic decompression profile to the surface. This study sought to investigate this issue by adapting an established porcine model of DCI (4,5) to compare the incidence and severity of neurological DCI after linear and non-linear decompression from an otherwise identical pressure exposure.

## METHODS

The study was conducted in 2 parts; the first a comparison of ascent profile shape on DCI risk from a deep air dive, the second from a deep heliox dive. Subjects, animal husbandry and predive preparation were the same for both parts of the study.

### *Subjects and Animal Husbandry*

Juvenile, male, neutered, pure-bred, Yorkshire swine from a closed breeding colony (weight range 16-20 kg on delivery) were received as numbered littermates. On receipt, pigs were examined by a veterinarian and an adjustable canine chest harness (Coastal Pet Products, Alliance, OH) was fitted to each animal to facilitate handling. All pigs were housed singly, indoors, in different runs. Water was freely available in each run, and minimum daily diet consisted of 2% by body weight of Purina Hog Finisher No. 50, to maintain a gradual weight increase with growth.

For this study, pigs were in-house for about one week before diving. After an initial 24 h to adjust to their new surroundings, pigs were introduced to the laboratory environment. Each weekday, they were transported from the animal care facility to the laboratory in plastic transport kennels (Vari-Kennel, R.C. Steele, Brockport, NY). All pigs were then habituated to the general laboratory conditions and to the 4 x 4 ft. enclosure where they would be observed for signs of DCI after diving. Each pig was also familiarized with the compression chamber and with the noise of flowing gas experienced during a dive.

All pigs were trained to run on a modified laboratory treadmill (Marquette Electronics, Milwaukee, WI). Training was made easier if a novice pig first observed an experienced pig running on the treadmill. The best time for training was in the morning, before feeding, as gastro-colic reflex effects were reduced, and feeding immediately after the treadmill session induced a Pavlovian response to training. After 3 - 4 sessions, most pigs ran easily on a 5% incline at a speed of 4 mph for 5 min. More prolonged or strenuous training was avoided because physical conditioning had been found to reduce the risk of neurological DCI (4).

*Predive Preparation*

Procedures were carried out with pigs passively restrained in a Panepinto sling (Charles River, Wilmington, MA), which cradled the animals' body while the legs hung down through holes. The slings were mounted on wheeled carts, which permitted easy movement around the laboratory.

On the afternoon before their dive, pigs were anesthetized (IM ketamine, 400 mg; xylazine, 20 mg) and venous catheterization of an ear vein was performed. This enabled both venous blood sampling predive and rapid venous access if a pig developed DCI after diving. We used customized, 18-inch long, polyurethane catheters (Braintree Scientific Inc. Braintree, MA), with 0.040" external / 0.025" internal diameter and an integral luer hub. After a cut-down onto the ear vein, the sterilized catheter was advanced 10 - 12 in into the central thoracic veins, then lightly tied into the vessel. The cut-down incision was sutured closed and

an injection port (Interlink System, Baxter, Deerfield, IL) was fitted to the catheter. Pigs were then given IV chloramphenicol, 500 mg, to reduce the risk of infection, and then the injection port was heparinized to maintain catheter patency overnight. The catheter was then firmly secured to the dorsum of the pig's ear using 2-inch woven, surgical, adhesive tape (National Patent Partnership, Dayville, CT). This type of tape proved the most reliable in confounding the pigs' attempts to remove it.

Before diving, each pig fasted overnight but had access to water *ad libitum*. In the morning, the pig was weighed, then placed in a Panepinto sling. The IV catheter was untaped and predive blood samples were drawn. Next, to prevent the catheter tubing from causing bubble nucleation during decompression (4,6), the catheter tip was withdrawn from the central veins into a peripheral vein (an indelible mark 2 - 3 in. from the catheter tip, made prior to sterilization, was helpful here). The catheter was then re-taped to the ear. Handled gently, pigs tolerated these predive procedures with no or minimal complaint, and sedation or anesthesia was unnecessary.

*Dive Procedures*

All pigs were dived once only, as described below. The compression chamber was a 66 x 30 in. cylinder (Bethlehem Corporation, Baltimore, MD). Each pig was dived while unrestrained in a transport kennel. The compression profile was controlled automatically by a computerized unit (Digital Control Programmer, Honeywell Corp., Phoenix, AZ), responding to a pressure transducer in the chamber (Smart Transmitter, 900 Series, Honeywell Corp.), and driving automated valves that control compression and exhaust (SVF, Santa Anna, CA). The decompression profile was guided manually to follow a preprogrammed track displayed by the computerized unit. This displayed a real-time, plus or minus display calibrated as fsw off track, and an accuracy of within ± 2 fsw from track was consistently achieved during the decompression.

Air dives

A total of 40 pigs performed an air dive (see Figure 1). The air dives were to 200 feet of seawater (fsw) (612.6 kPa). Compression took 5 min: 2 min at 20 fsw/min (61 kPa/min) to 40 fsw, 1 min at 40 fsw/min (122 kPa/min) to 80 fsw, then 2 min at 60 fsw/min (183 kPa/min) to chamber bottom. The initial compression rate was slow to allow pigs to clear their ears. Time from leaving surface to starting decompression was 24 min (time at chamber bottom was 19 min). Total decompression time was 10 min - the linear group decompressing at 20 fsw/min; the fast/slow group at 60 fsw/min to 110 fsw then 12.9 fsw/min to surface.

The first 10 pigs in each of the linear and fast/slow groups were intended as a pilot study and pigs were not randomized, nor was the principal investigator blinded to the dive profile at the time of diagnosis. The last 20 pigs were dived as 10 matched litter pairs. One pig of each pair dived the linear profile and the other pig of the pair dived the fast/slow profile. The first pig of each pair to dive was randomized to dive either profile by coin toss. To avoid possible diagnostic bias, the Principal Investigator (PI) was not present during the randomization or the dive and was admitted to the laboratory only on surfacing. Diagnosis of neurological DCI and severity of skin DCI was therefore made without knowledge of the preceding dive profile.

Heliox dives

A total of 40 pigs performed a dive breathing 80/20% heliox (see Figure 2). The dive was to 250 fsw (765.8 kPa) for 8 min 50 s. Compression took 5 min 50 s; 2 min at 20 fsw/min (61 kPa/min) to 40 fsw; 1 min at 40 fsw/min (122 kPa/min) to 80 fsw; then 2 min 50 s at 60 fsw/min (183 kPa/min) to 250 fsw. Time at chamber bottom was 3 min, followed by decompressions lasting 8 min 20 s at either a linear rate of 30 fsw/min, or on a fast/slow profile: 80 fsw/min to 130 fsw; 30 fsw/min to 60 fsw then 13.3 fsw/min to surface.

Several technical aspects of the heliox dives were different from the air series. Pigs were dived inside a transport kennel specially modified to be gastight, other than through inlet

and outlet valves. Heliox (80/20%) was flushed into the kennel at flow rates up to 10 ℓ/min for 15 min before diving. We were unable to measure the atmospheric contents of the kennel directly, but assumed that the nitrogen content would be minimal at the commencement of the dive. The chamber was then pressurized with air while maintaining a positive flow of heliox into the kennel. This was intended to ensure that the pigs breathed heliox throughout the dive, while achieving considerable economy in the use of heliox.

All heliox dives used matched litter pairs randomized by coin toss to either the linear or fast/slow decompression. The P.I. was blinded to the profile dived by individual pigs as described above for the later air dives.

*Postdive*

On surfacing, all pigs were transferred from the chamber into the laboratory observation pen where they were closely observed. Behavioral features and the development of pruritus (denoted by scratching), skin DCI, or constitutional signs such as lethargy were noted but not treated. If neurological signs such as limb weakness, paralysis, or marked ataxia developed, pigs were placed in a Panepinto sling and observed for up to 1 h postdive. If any signs of distress were observed, pigs were sedated by diazepam, 5-10 mg IV, and observation was continued until 1 hour postdive whereupon a final assessment of skin DCI was made. At this point, affected pigs were first anesthetized by IV injection of ketamine (400 mg in 4 ml) and xylazine (20 mg in 1 ml) via the ear vein catheter, then euthanized by bolus IV injection of 30-50 ml of 4-molar potassium chloride solution.

When pigs failed to develop subjective neurological signs after 1 h of observation, the pigs ran on the treadmill and their gait was assessed. Pigs with no discernable gait abnormality were categorized as "no neurological DCI". These pigs took no further part in the protocol.

*Grading Severity of Neurological and Skin DCI*

The above procedures allowed the severity of neurological DCI to be crudely graded

into 5 categories (below) of which (d) and (e) were considered "severe":

a) Functionally normal

b) Able to run on the treadmill but detectable gait abnormality (lame/ataxic)

c) Can stand but weakness of one or more limbs

d) Unable to stand due to fore- or hind leg paresis/ataxia

e) Dead

The severity of skin DCI was also subjectively estimated as the proportion of skin surface area affected:

Severe - > 50% skin surface area affected

Moderate - 20 - 50% affected

Mild - < 20% affected

None - No visible skin DCI

*Statistical Methods*

Statistical comparison was by $\chi^2$ analysis of discrete variables in 2 x 2 contingency tables, taking $p = 0.05$ as the threshold of significance.

There is no accepted way of combining statistical analysis of both the incidence *and* severity of DCI, and statistical analysis of *incidence* for the air dive series is not strictly valid due to the combination of the non-randomized, unpaired pilot study (20 pigs) and the paired, randomized pigs from the later part of the study (20 pigs). However, valid statistical comparison of *severity* for both neurological and skin DCI was performed retrospectively. Nonetheless, the study results from the air series are believed be clear from simple observation (7) of both incidence and severity data and it was considered inappropriate use of animals to expend an additional 20 or 40 pigs on the air series merely to satisfy a statistical design criterion for incidence alone.

For the heliox series, a maximum of 60 pigs (30 pairs) was decided upon, based on the results from the air series. The design was sequential in order to minimize animal use:

For a positive result (i.e., the incidence of DCI with the linear ascent greater than with the fast/slow ascent), stopping criteria were that the excess of DCI cases in the linear group was $\geq 5$ after 10 or 20 pairs, and $\geq 7$ after 30 pairs. For a negative result, the stopping criteria were that the excess of DCI in the linear group was $\leq 2$ after 10, 20, or 30 pairs. This was a one-tailed design. Computer simulation indicated that, assuming a linear group DCI incidence of 0.5 and a fast/slow group incidence of 0.2, the probability of the study terminating without a decision was $< 0.05$, and the alpha value was $\leq 0.5$, with a power of about 0.6.

**RESULTS** (See Annex A and B for full data)

*Air Series* (See Table 1 for summary)

Neurological DCI

Of the pigs dived on the linear ascent profile, 11/20 (55%) were diagnosed as suffering from neurological DCI compared to 5/20 (25%) of the pigs decompressed on the fast/slow ascent.

Of the 11 affected pigs in the linear group, 1 died and 8 were unable to stand due to severe paresis of 1 or more limbs. The remaining 2 affected pigs had unequivocal mild limb weakness, which in 1 case resolved spontaneously within an hour. Of the 5 affected pigs in the fast/slow group, only 1 pig was unable to stand due to severe weakness, 1 had signs suggestive of bilateral sensory dysfunction in the hind legs but no weakness, and the remaining 3 could all stand, or run on the treadmill, but had gait disturbance due to lameness or mild ataxia. Application of chi-squared analysis to compare the functional severity of DCI between groups (9/20 vs. 1/20 severe cases) suggests a significant difference (Yates corrected $\chi^2 = 6.53$; $p = 0.01$).

Skin DCI

Of the 20 pigs in the linear group 13 developed skin DCI affecting 20% or more of their skin surface area, compared to 6/20 of the fast/slow group ($\chi^2 = 4.91$; $p = 0.027$). Only

2 of the linear group had no skin DCI compared to 6 in the fast/slow group.

Table 1 **Air series: Summary of neurological DCI incidence, functional severity, and skin DCI severity.**

| | Linear group (n=20) | Fast/slow group (n=20) | p value |
|---|---|---|---|
| Neuro DCI cases | 11 | 5 | N/A |
| Severe cases | 9 (1 death) | 1 | 0.01 |
| Skin DCI >20% | 13 | 6 | 0.027 |

*Heliox Series* (See Table 2 for summary)

Neurological DCI

Of the pigs dived on the linear ascent profile, 16/20 (80%) were diagnosed as suffering from neurological DCI compared to 8/20 (40%) of the pigs decompressed on the fast/slow ascent (Yates corrected $\chi^2 = 5.1$; $p = 0.023$).

Of the 16 affected pigs in the linear group, 1 died and 11 were unable to stand due to severe paresis of 1 or more limbs. The remaining 4 affected pigs were able to stand but had unequivocal gait disturbance due to lameness or ataxia. By comparison, of the 8 affected pigs in the fast/slow group, only 3 pigs had severe limb paresis preventing standing, 1 pig had signs suggestive of bilateral sensory dysfunction in the hind legs but no weakness, and 3 pigs were able to stand or treadmill but were unequivocally lame. The remaining affected pig had transient but unequivocal hind leg weakness that had resolved completely by 1 h postdive. The fast/slow group had significantly less severe disease (12/20 vs 3/20; Yates corrected $\chi^2 = 6.83$; $p = 0.009$).

Skin DCI

16/20 of pigs in the linear group developed skin DCI affecting 20% or more of their skin surface area, compared to 3/20 of the fast/slow group (Yates corrected $\chi^2 = 14.44$;

p = < 0.0002). Only 2 of the linear group had no skin DCI compared to 8 in the fast/slow group.

Table 2 **Heliox series: Summary of neurological DCI incidence, functional severity, and skin DCI severity.**

| | Linear group (n=20) | Fast/slow group (n=20) | p value |
|---|---|---|---|
| Neuro DCI cases | 16 | 8 | 0.023 |
| Severe cases | 12 (1 death) | 3 | 0.009 |
| Skin DCI >20% | 16 | 3 | 0.0002 |

**DISCUSSION**

Compared to pigs decompressed on a linear ascent profile, pigs decompressed in the same amount of time on the non-linear "fast/slow" ascent profile were significantly less likely to develop neurological DCI. In those pigs of the fast/slow group that did develop neurological DCI, the disease was likely to be less severe than in the linear group. As an alternative outcome measure, the severity of skin DCI was also significantly less in pigs ascending on the fast/slow profile. These observations were consistent for both air and heliox dives. The mechanism behind the observation is speculative, but one of the two following explanations seem possible.

*Possible Mechanism (a) - Continued On-gassing During the Deeper Phase of Decompression*

In the linear ascent group, a slow rate of ascent during the deep stage of decompression may allow unsaturated tissues to continue on-gassing, even though the ambient pressure is decreasing. Not only would this result in a greater overall tissue gas burden, but tissues would have relatively less time to eliminate the excess in the shallow stages of the decompression.

By contrast, the fast/slow ascent may minimize the time for continued on-gassing by an initial, fast depth reduction, and the lower gas burden that results has more time to be eliminated because of the slower rate of ascent nearer the surface. The combination of these factors may reduce the likelihood of bubble formation during the shallow phase of the decompression when the "driving force" for off-gassing of tissues is greatest, and the potential for volume expansion of any bubbles formed is maximal.

Central nervous system (CNS) tissue is generally considered to be among the "faster" tissues with respect to gas elimination half-time. If the above theory is the true mechanism, it is interesting to note that for the air dive, at the time of leaving bottom, a tissue with a 5-minute half-time would be 93-96% saturated (8). The fast/slow decompression reduced the time spent deeper than 110 fsw by only 3 min compared to the 20 fsw/min linear decompression. In 3 min, the calculated increase in saturation of a 5-minute tissue is only about 2% at most (8). It seems unlikely that this small difference could be responsible for the markedly different clinical outcomes of the linear and fast slow groups of pigs. This implies that either the spinal nervous tissue(s) responsible for DCI in the pigs has a half time considerably slower than 5 min, that the tissue in question does not conform to the theory on which the calculation of tissue saturation and half-time is based, or that DCI risk is not directly related to tissue half time.

*Possible Mechanism (b) - More Efficient Off-gassing During Decompression*

An alternative explanation may not involve marked differences in on-gassing: In a manner similar to that originally hypothesized by Haldane (2), the fast/slow decompression may allow more efficient off-gassing by the tissues responsible for neurological DCI because the fast initial phase of the ascent maximizes the driving pressure to eliminate the inert gas load. In Boycott and Haldane's original 1908 paper (2), the practical application of this concept was to use progressively longer staged decompression stops after an initial rapid ascent to about half the depth of the dive. In goats, they found that

this method of staged decompression was considerably safer than a linear decompression of the same overall duration (2, p. 364). In essence, we have applied the same Haldanian theory to the ascent from a *no-stop* dive and found a similar reduction in the risk of bends.

Arguable support for our observations being due to this Haldanian mechanism may be drawn from the empirical safety measure practiced by some sport divers of making a shallow "safety stop"on a dive normally performed with no stops. Vann recently reported that a 3-minute stop at 20 fsw after a 28-minute, nitrox dive to 101 fsw, significantly reduced the risk of venous gas emboli detected after the dive by precordial Doppler ultrasound (9). However, the "safety stop" ascent took 3 min longer than the direct ascent, so the ascent profiles are not directly comparable as with our linear and fast/slow ascents.

*Relevance of Findings to Human Diving*

To assess the implications of the study findings, the relevance of the pig model to human diving must be reviewed: The pig model employs a relatively severe dive to produce a high DCI rate, against which the effect of different interventions may be compared under controlled conditions. The manifestations of DCI produced by the model are similar to those seen in severe, early-onset human DCI (5). Because the mechanisms of DCI remain an area of debate, the model was designed to circumvent this problem by having direct assessment of function as the primary outcome parameter. The pig acts as a biological, mammalian "black box", from which differences in DCI outcome allow comparisons to be made between variables that may influence tissue gas kinetics. Although desirable, it is not necessary to understand the mechanisms of a beneficial intervention before determining its potential value as a protective measure in humans. The anatomy and physiology of humans and pigs are sufficiently similar (10, 11) that any qualitative differences between pig and human tissue gas kinetics highly unlikely. Thus, any assertion that the phenomenon demonstrated in the study would not apply to humans is difficult to sustain.

We cannot be sure that the reduction in risk demonstrated will apply to DCI manifestations other than nervous system DCI; however, CNS injury is the most feared and therapeutically challenging form of DCI. Furthermore, we have shown a similar beneficial effect of a fast/slow ascent on both CNS *and* skin manifestations, and it would seem implausible that the underlying mechanism of musculoskeletal DCI is so different that it would not benefit also.

In addition, it is uncertain whether the degree of risk reduction achieved by the fast/slow decompression is proportionate or absolute. For example, in both the air and heliox series, the observed risk was reduced by a factor of 2 or more. In the heliox series the risk reduction was from 80% to 40% (95% confidence limits for the relative risk of 2.0 are $1.12 < RR < 3.57$). Most human diving is performed with an absolute DCI risk of < 5%, and one possibility is that a fast/slow profile would merely reduce the relative risk proportionately by a factor of about 2 (i.e., from 5% to 2.5%). Alternatively, if the fast/slow profile, which we have shown can markedly reduce the risk of a high risk dive, achieves this by an absolute effect on gas exchange, then a dive currently with a linear ascent and 5% risk would probably become very safe indeed. Our study finding that DCI *severity* as well as incidence is significantly reduced by the fast/slow decompression would suggest that the process is not simply stochastic and that the latter, absolute effect may be more likely. If this is true, then by adopting a non-linear ascent profile it may be possible to increase the bottom time of a no-stop dive currently assessed at a 5% risk level, without exceeding that risk level. The bottom time increment possible before the risk would again approach the 5% level is currently unknown. These issues can only be resolved by further studies.

*Operational Relevance of Study Findings*

The phenomenon appears to apply to both air and heliox dives. For the heliox dives in particular, the linear 30 fsw/min rate of ascent was chosen to correspond to current U.S. Navy diving practice, and the depth was chosen to be in the range where an operational capability

is desired by the military underwater Explosive Ordnance Disposal (EOD) community. The results of the study strongly suggest that the risk of operational, deep, heliox bounce dives could be reduced by a factor of 2 or more by adopting a no-stop ascent profile that starts fast and slows toward the surface. This change in operational procedure could be implemented empirically and would carry little, if any, operational penalty: In practice, for added safety, divers could be advised to calculate their no-stop ascent times before the dive, based on the anticipated dive depth and the currently recommended 30 fsw/min linear ascent. They could then ascend fast (60 fsw/min) to about half the maximum dive depth, then markedly slow their rate of ascent so as to reach the surface no sooner than the total of their bottom time plus calculated ascent time.

The air dive ascent, at 20 fsw/min, is slower than in operational military practice. Operational application of the above procedure to an air dive with 30 fsw/min ascent is by extrapolation and would be premature before confirmation in further animal and human trials.

*Research Relevance of Study Findings*

The study findings have obvious research implications, particularly for risk-based decompression table development. It is desirable to be able to reconcile the observations with mathematical models of decompression, and to do this more data points are necessary. The issue of absolute versus relative risk reduction may be answered by repeating the studies in pigs using a linear dive profile with a lower baseline DCI rate. Plans to conduct these studies are being formulated. Confirmation of the phenomenon in man, breathing air (nitrox) and heliox should be given high priority in human experimental diving.

## RECOMMENDATIONS

1. In extreme operational circumstances, particularly for a high risk dive, breathing heliox, where in-water stops are undesirable, a fast-deep / slow-shallow ascent profile may be recommended empirically to reduce risk of neurological DCI in human divers.

2. Confirmation of the phenomenon in human, deep, no-stop dives, breathing air (nitrox) and heliox should be given high priority in human experimental diving, particularly that supporting the EOD diving community.

3. The issue of whether the fast/slow ascent procedure confers *absolute* versus *relative* risk reduction should be resolved as rapidly as possible by further animal experiments using lower risk dive profiles.

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Fig. 1

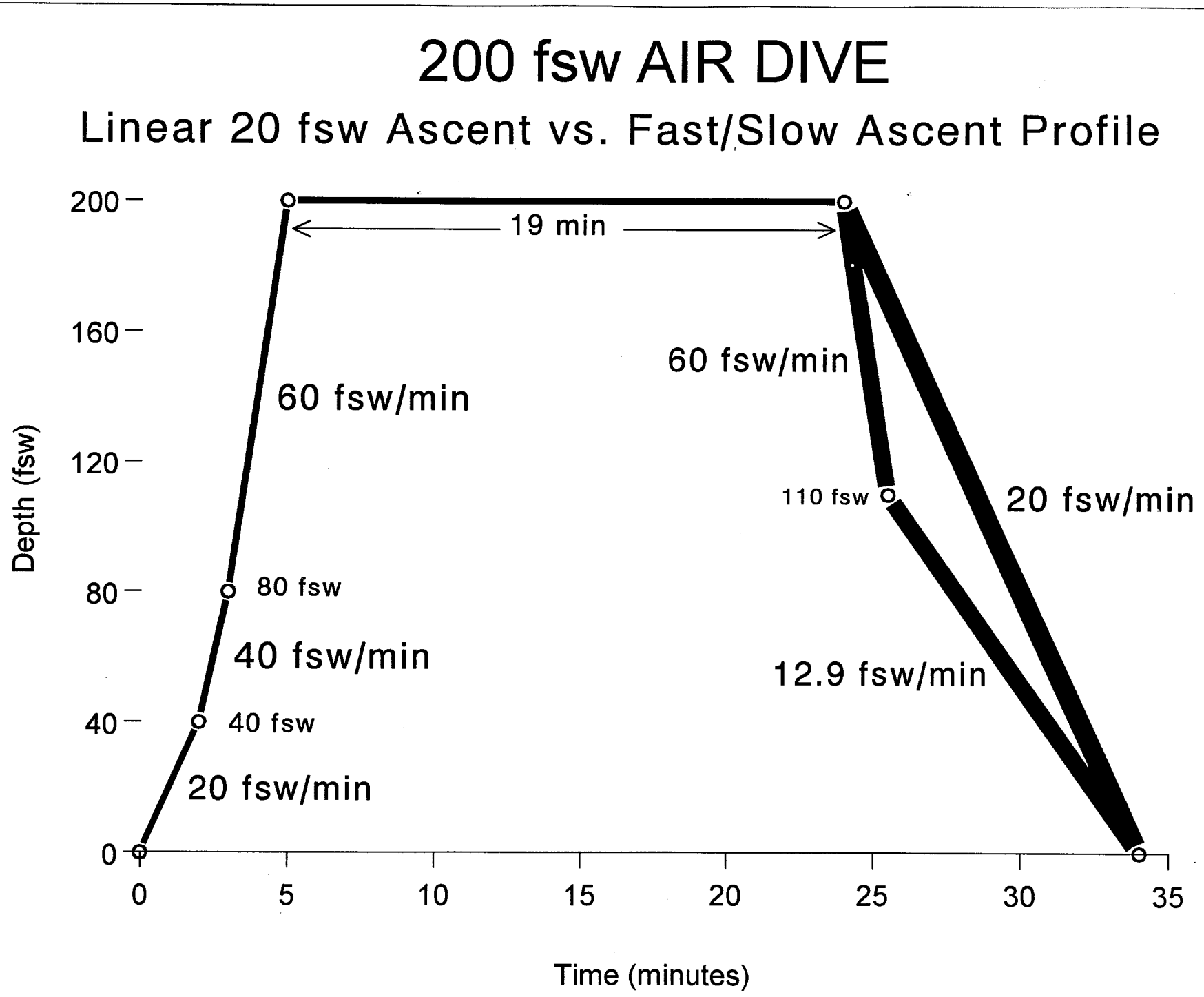
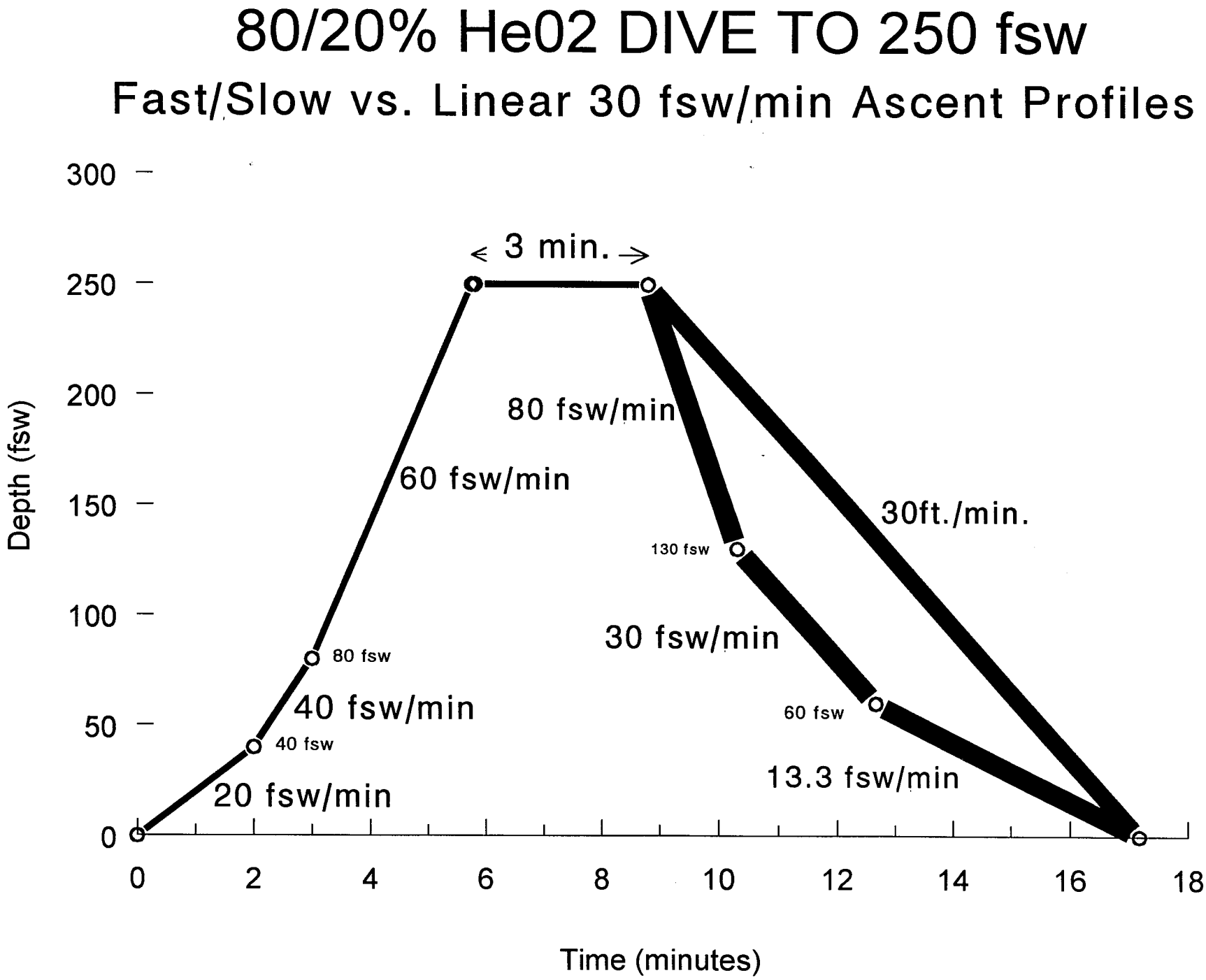

200 fsw AIR DIVE
Linear 20 fsw Ascent vs. Fast/Slow Ascent Profile
Depth (fsw)
200
160
120
80
40
0
19 min
60 fsw/min
60 fsw/min
110 fsw
20 fsw/min
80 fsw
40 fsw/min
12.9 fsw/min
40 fsw
20 fsw/min
0
5
10
15
20
25
30
35
Time (minutes)

Fig. 2


80/20% He02 DIVE TO 250 fsw
Fast/Slow vs. Linear 30 fsw/min Ascent Profiles
300
250
200
150
100
50
0
Depth (fsw)
3 min.
80 fsw/min
60 fsw/min
30ft./min.
130 fsw
30 fsw/min
80 fsw
40 fsw/min
60 fsw
40 fsw
13.3 fsw/min
20 fsw/min
0
2
4
6
8
10
12
14
16
18
Time (minutes)

ANNEX A:

DATA FROM AIR DIVE TO 200fsw: LINEAR vs. FAST/SLOW ASCENT

| PIG # | WT. (KG) | ASCENT TYPE | SKIN BEND | NEURO HIT? | ONSET TIME AND NEUROLOGICAL CLINICAL FEATURES | NEURO SEVERITY |
|---|---|---|---|---|---|---|
| 953 | 20.20 | L | MOD | NO | ---------- | ----- |
| 851 | 19.80 | L | MOD | YES | 7:20 BILATERAL HIND LEG WEAKNESS | SEV |
| 850 | 18.40 | L | MIN | NO | ---------- | |
| 874 | 20.60 | L | SEV | YES | AT SURFACE - FORELEG PARESIS | SEV |
| 895 | 17.50 | L | SEV | YES | AT SURFACE - FORE AND HIND LEG PARESIS/ATAXIA | SEV |
| 893 | 17.65 | L | MIN | NO | ---------- | ----- |
| 900 | 19.60 | L | MIN | NO | ---------- | ----- |
| 899 | 18.70 | L | MOD | YES | 3:30 PROGRESSIVE HIND LEG WEAKNESS | SEV |
| 896 | 18.30 | L | MIN | NO | ---------- | ----- |
| 894 | 19.20 | L | MIN | YES | 18:00 CAN STAND BUT LEFT HIND LEG WEAKNESS | LAME |
| 946 | 19.30 | F/S | MIN | YES | AT SURFACE - MILD ATAXIA PERSISTED, ABLE TO TREADMILL | ATAXIA |
| 944 | 20.00 | F/S | NO | NO | ---------- | ----- |
| 945 | 18.30 | F/S | MIN | NO | ---------- | ----- |
| 947 | 20.60 | F/S | MOD | YES | AT SURFACE - PERSISTENT MILD ATAXIA, ABLE TO TREADMILL | ATAXIA |
| 948 | 20.30 | F/S | MIN | NO | ---------- | ----- |
| 949 | 19.60 | F/S | NO | NO | ---------- | ----- |
| 979 | 18.00 | F/S | MOD | YES | 8:22 HIND LEG WEAKNESS RESOLVED BY 1H, NORMAL TREADMILL | MILD |
| 978 | 19.00 | F/S | MIN | NO | ---------- | ----- |
| 981 | 19.30 | F/S | MIN | NO | ---------- | ----- |
| 980 | 19.60 | F/S | MIN | NO | ---------- | ----- |
| 983 | 18.00 | L | MOD | YES | AT SURFACE - RIGHT FORELEG PARESIS | SEV |
| 982 | 20.00 | F/S | MOD | NO | ---------- | ----- |
| 505 | 18.80 | F/S | NO | NO | ---------- | ----- |
| 503 | 20.10 | L | SEV | YES | AT SURFACE - TETRAPARETIC | SEV |
| 507 | 20.80 | F/S | SEV | YES | 10:00 SENSORY SIGNS IN HIND LEGS - NO WEAKNESS | MOD |
| 506 | 21.10 | L | NO | NO | ---------- | ----- |
| 504 | 21.10 | F/S | MIN | NO | ---------- | ----- |
| 502 | 22.10 | L | MOD | YES | 12:20 BILATERAL HIND LEG WEAKNESS | SEV |
| 533 | 18.90 | L | MOD | YES | 13:00 UNEQUIVOCAL RIGHT HIND LEG WEAKNESS, RESOLVED BY 1H | MILD |
| 536 | 20.20 | F/S | SEV | YES | AT SURFACE - FORE AND HIND LEG PARESIS | SEV |
| 532 | 19.30 | F/S | MOD | YES | 2:00 CAN TREADMILL BUT LAME RIGHT HIND LEG | LAME |
| 534 | 20.00 | L | NO | NO | ---------- | ----- |
| 537 | 18.50 | F/S | NO | NO | ---------- | ----- |
| 535 | 18.30 | L | SEV | YES | AT SURFACE - TETRAPARETIC. DIED AT 10 MIN. | SEV |
| 262 | 19.90 | F/S | NO | NO | ---------- | ----- |
| 260 | 19.70 | L | MOD | NO | ---------- | ----- |
| 261 | 18.60 | L | MIN | NO | ---------- | ----- |
| 257 | 19.30 | F/S | MIN | NO | ---------- | ----- |
| 258 | 18.50 | F/S | NO | NO | ---------- | ----- |
| 259 | 18.40 | L | SEV | YES | AT SURFACE - FORE AND HIND LEG WEAKNESS | SEV |

ANNEX B:

DATA FROM 80/20% HELIOX DIVE TO 250fsw: LINEAR VS. FAST/SLOW ASCENT

| PIG # | WT. (KG) | DECOMP TYPE | SKIN BEND | NEURO HIT? | ONSET TIME AND NEUROLOGICAL CLINICAL FEATURES | NEURO SEVERITY |
|---|---|---|---|---|---|---|
| 734 | 20.35 | L | MOD | YES | 4:22 HIND LEG PARESIS | SEV |
| 738 | 21.30 | F/S | MIN | NO | ---------- | ---------- |
| 733 | 20.50 | F/S | NO | YES | 5:20 ABLE TO STAND BUT WEAKNESS AND ATAXIA OF HIND LEGS | MOD/ATAXIA |
| 735 | 20.30 | L | MOD | NO | ---------- | ---------- |
| 736 | 20.20 | L | MOD | YES | AT SURFACE - ABLE TO STAND BUT HIND LEG ATAXIA | ATAXIA |
| 737 | 19.60 | F/S | NO | NO | ---------- | ---------- |
| 923 | 20.65 | L | SEV | YES | 3:04 WEAKNESS OF BOTH HIND LEGS | SEV |
| 927 | 19.15 | F/S | NO | NO | ---------- | ---------- |
| 926 | 19.90 | L | SEV | NO | ---------- | ---------- |
| 929 | 17.90 | F/S | NO | NO | ---------- | ---------- |
| 925 | 19.20 | F/S | MIN | YES | 4:26 UNEQUIVOCAL LEFT HIND WEAKNESS, RESOLVED BY 1 H. | MILD |
| 928 | 19.10 | L | MOD | YES | AT SURFACE - PARAPLEGIC | SEV |
| 420 | 20.50 | L | MIN | YES | AT SURFACE - TETRAPLEGIC | SEV |
| 421 | 20.60 | F/S | MIN | NO | ---------- | ---------- |
| 417 | 22.80 | L | SEV | NO | ---------- | ---------- |
| 418 | 19.50 | F/S | MIN | YES | 1:30 PROGRESSIVE HIND LEG WEAKNESS | SEV |
| 821 | 20.80 | L | MOD | YES | 1:40 HIND LEG ATAXIA/ABLE TO WALK BUT LAME | ATAXIA |
| 823 | 19.60 | F/S | MOD | YES | 11:00 SENSORY SIGNS BOTH HIND LEGS, NO WEAKNESS | MOD/SENSORY |
| 822 | 22.40 | L | MOD | YES | 2:00 HIND LEG WEAKNESS PROGRESSED TO TETRAPLEGIA | SEV |
| 824 | 22.20 | F/S | NO | NO | ---------- | ---------- |
| 9 | 19.90 | L | SEV | YES | 4:00 FORE AND HIND LEG WEAKNESS | SEV |
| 10 | 21.10 | F/S | NO | NO | ---------- | ---------- |
| 7 | 19.80 | F/S | SEV | YES | 2:00 FORE AND HIND LEG WEAKNESS | SEV |
| 11 | 20.50 | L | SEV | YES | 2:00 ATAXIA AND RIGHT HIND WEAKNESS. | SEV |
| 8 | 20.40 | L | MIN | NO | ---------- | ---------- |
| 12 | 21.90 | F/S | MOD | NO | ---------- | ---------- |
| 28 | 21.20 | L | MIN | YES | 2:00 WEAKNESS RIGHT HIND LEG.CAN STAND | LAME |
| 33 | 20.20 | F/S | NO | NO | ---------- | ---------- |
| 29 | 20.70 | F/S | MIN | YES | AT SURFACE - TETRAPARESIS | SEV |
| 30 | 20.70 | L | SEV | YES | 1:30 FORELEG WEAKNESS PROGRESSED TO TETRAPARESIS | ---------- |
| 31 | 21.05 | L | MOD | YES | 2:12 HIND LEG WEAKNESS AND ATAXIA CAN WALK BUT ATAXIC | ATAXIA |
| 32 | 20.65 | F/S | MIN | NO | ---------- | ---------- |
| 38 | 18.60 | F/S | MIN | YES | 1:30 RIGHT FORELEG WEAKNESS. ABLE TO STAND | LAME |
| 39 | 21.40 | L | MOD | YES | AT SURFACE - PARAPLEGIA | SEV |
| 34 | 21.10 | F/S | NO | NO | ---------- | ---------- |
| 35 | 21.60 | L | SEV | YES | AT SURFACE - TETRAPLEGIA. DIED - RESPIRATORY ARREST | SEV (DIED) |
| 36 | 18.60 | F/S | MIN | NO | ---------- | ---------- |
| 37 | 21.20 | L | NO | YES | 1:30 FORELEG PARESIS | SEV |
| 41 | 20.20 | F/S | MIN | YES | 6:00 HIND LEG WEAKNESS. ABLE TO STAND | MOD/WEAK |
| 42 | 20.40 | L | MOD | YES | 4:12 PROGRESSIVE HIND LEG WEAKNESS | SEV |