Some Responses of Squirrel Monkeys to High G — Brief Duration Acceleration Profiles

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ACCIDENT SURVIVAL,[4] the experiments of Beeding,[1] Black-Schaffer,[2] Kornhauser,[5] Stapp,[6] and other work[7] have led us to speculate that men, properly supported and protected by advanced techniques, could survive much higher acceleration for longer time periods than had been previously possible. If such "unconventional" G-time loads could be tolerated, a number of operational applications in space and defense suggest themselves. But before practical application can be made, experiments must be performed to validate the hypothesis that man can accept and survive much higher accelerations for longer times than formerly thought possible, and before men are to be risked, prudence dictates the use of primates to gain data and develop equipment and techniques. We recognize that extrapolation of data from nonhuman primates to humans is unwise because of ignorance of accurate scaling relationships, but primates were selected because of their physiologic and anatomic similarity to man and their previous use as test subjects in dynamic environment studies. We desired to investigate the response of these primates to a family of acceleration profiles different than those that could be developed on conventional acceleration devices. Conventional impact devices (drop towers, sleds, swings, etc.) are characterized by high rates of change of acceleration ("jolt") over short time bases and high peak accelerative loads, while conventional long-term acceleration devices are typified by centrifuges where low peak loads can be generated and maintained for long dwell times but with the penalty of low onset values. We wished to expose primates to a family of G-time profiles characterized by: a. Rates of change variable from 10 to 1000 G per second; b. Peak G values variable from 20 to 500; c. Dwell at peak G variable from .1 sec. to several hours. But no device existed to produce repeatedly and inexpensively G-time profiles with these characteristics, so it was necessary to invent and develop a special accelerator for these studies. With the advent in 1962 of this unique articulated centrifuge, called the Space Flight Acceleration Profile Simulator (whose characteristics are reported elsewhere[3]), it became possible to produce a family of G-time profiles characterized by: a. Rates of change variable from less than 1 up to 275 G/sec.; b. Peak G values variable from 20 to 450; c. Dwell at peak G variable from .1 sec. to several hours. The great variety of curves that can be generated by this device made it necessary to select an arbitrary starting point for these preliminary investigations of the response of small primates to acceleration environments. It was decided to use profiles with the following characteristics: a. Peak G's varying from 25 to 450 G over total time bases varying from 4.5 sec. to 20 seconds; b. Rates of onset between 10 and 45 G/sec.; c. Dwells constant at .3 sec. or less.

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METHODS

In each experiment, a different squirrel monkey (*Siamiri Sciuria*) from a standardized colony was caught using a squeeze cage to minimize trauma and excitement and anesthetized with intraperitoneal aqueous pentobarbital sodium, 2.3 mgms./lb. The animal's chest and the posterior aspect of the left lower leg were soaped, shaved, and swabbed with 70 per cent ethyl alcohol. The animal was then placed in the unlined couch. The couch configuration was such that the animal's spine, upper arm and lower leg was at 90° to the G load and the upper leg and lower arm were approximately 60° relative to the load in the G_X axis. Electrocardiograph leads, consisting of a rubber ring-lead plate electrode filled with electrode jelly, were affixed using adhesive collodion spray. The electrodes were placed to the right of the manubrioclavicular junction, on the sternum at the level of fifth rib, and the indifferent lead was located on the posterior aspect of the left lower leg at the major diameter of the gastrocnemius. This typically produced a normal electrocardiographic record like that seen in Figure 1. Tape and restraint plates held the animal in place after instrumentation, as shown in Figure 2. The

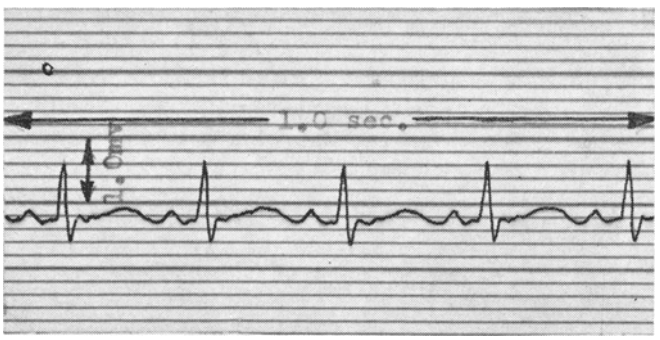

Fig. 1. Typical squirrel monkey ECG obtained through the articulated centrifuge's instrumentation system. Time and voltages are as indicated.

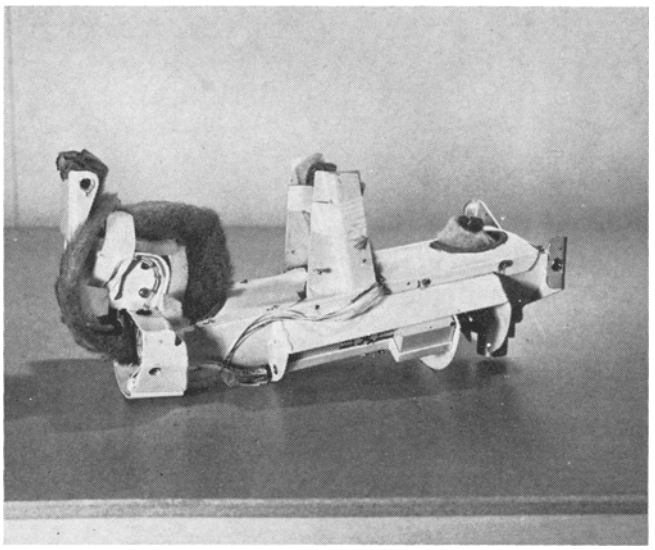

Fig. 2. Animal in couch, restrained by plates and tape at wrists and ankles. At weigh and balance, tail is taped on G_z axis of balance.

couch and animal were then weighed and balanced and weights appropriately adjusted to satisfy centrifuge dynamic requirements. Next, the couch and animal were bolted into the centrifuge cage and the cage cover attached (Figure 3). The centrifuge was adjusted to

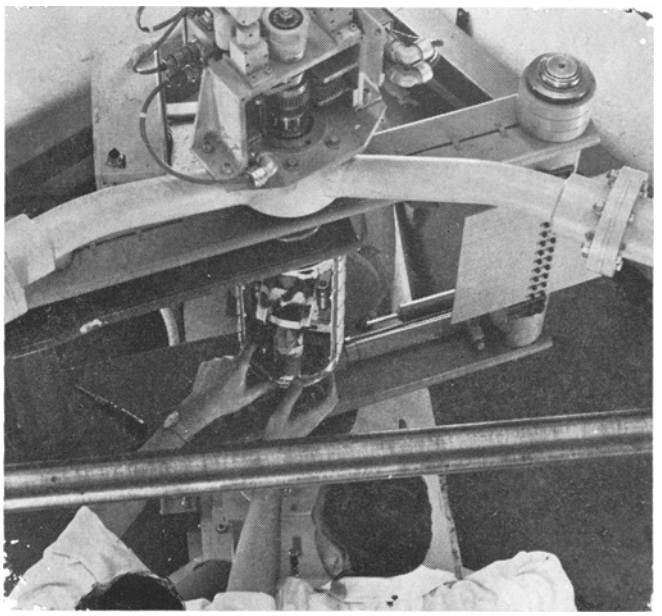

Fig. 3. Couched animal being placed in cage on articulated centrifuge.

produce the desired combination of onset rates, dwell, peak accelerative force, and decay rate, and to obtain the desired direction of force delivery. In all the experiments the force was nominally delivered $+G_X$ (AGARD standard terminology or "eyeballs in"), but a small $-G_Y$ component was almost always present ("eyeballs left") because of the difficulty of exactly aligning the couch in the cage without use of optical devices. The run was then made in accordance with a countdown to provide standardized procedures. The accelerative loads used ranged from 23 to 440, onsets ranged from 8 G/sec. to 36 G/sec. and decays ranged from 9 G/sec. to 60 G/sec. and dwells never exceeded .30 seconds. For details of the dynamic events, see Table I. Note that the experiment numbers recapitulate the dynamic parameters, with the second number being peak G and the last number being the total time in seconds.

Post-run, the animal was removed from the couch and transferred to a cage for observation. Fourteen days later, the animal was sacrificed under anesthesia and necropsied if death had not occurred naturally prior to that time. In the latter case the animal was necropsied as soon as possible post mortem.

Controls were of two major types: standard controls and spin controls. Standard controls were maintained in the colony and sacrificed to provide anatomic comparison. The spin control animals were prepared exactly as were the centrifuged animals except the controls were only "spun up" radially and not exposed to the $+G_X$ loads. The control spin rates used were the same as those produced by runs with 50, 75, 100, 150, 200, 300 and 400 $+G_X$ loads.

TABLE I. DYNAMIC EVENTS

Experiment Number	Animal Number	Weight (gms.)	Sex	Spin-up (sec.)	Onset (sec.)	Dwell (sec.)	+Gx (peak)	-Gy (peak)	Decay (sec.)	Despin (sec.)
001-025-008.00				Shakedown run. No reliable data.						
002-026-005.00	2	435	M	9.9	2.80	.20	26.6	1.00	2.60	.200
003-023-005.00	3	471	M	9.0	2.70	.20	23.5	1.00	2.50	.200
004-096-012.00	5	550	M	NA *	5.95	.19	96.5	2.25	5.75	NA *
005-209-018.00	6	600	F	NA *	9.1	.20	209.0	22.0	9.15	NA *
006-330-023.00	7	383	M	NA *	12.0	.20	330.0	8.8	10.8	NA *
007-405-020.00	8	398	F	NA *	12.25	.18	405.0	12.0	7.25	NA *
008-394-015.00	9	484	F	NA *	8.0	.10	394	2.8	6.9	NA *
009-Spin Control	10	568	F	6.0	NA **	NA **	NA **	NA **	NA **	1.00
010-Spin Control	11	527	F	4.85	NA **	NA **	NA **	NA **	NA **	1.05
011-Spin Control	12	487	F	5.38	NA **	NA **	NA **	NA **	NA **	1.50
012-Spin Control	13	546	F	1.44	NA **	NA **	NA **	NA **	NA **	1.09
013-430-00-20.60	14	587	F	NA *	12.20	.20	430	3.2	8.20	NA *
014-425-00-20.45	15	475	F	NA *	12.55	.20	425	12.6	7.70	NA *
015-408-00-21.15	16	556	F	NA *	12.05	.20	408	2.4	8.90	NA *
016-428-00-20.25	17	490	F	NA *	12.55	.14	428	4.2	7.56	NA *
017-440-00-19.59	18	534	M	NA *	12.07	.22	440	5.5	7.30	NA *
018-415-00-19.41	19	721	M	NA *	12.13	.19	415	7.5	7.09	NA *
019-404-00-18.94	20	407	F	NA *	11.87	.24	404	5.0	6.83	NA *
020-400-00-18.89	21	445	M	NA *	11.89	.30	400	6.0	6.70	NA *
021-416-00-19.83	22	480	M	NA *	12.17	.30	416	3.8	7.36	NA *
022-413-00-19.27	23	503	M	NA *	12.07	.26	413	4.7	6.94	NA *

* Not applicable—SFAPS used as conventional centrifuge.
** Not applicable—centrifuge not cycled.

RESULTS

When squirrel monkeys were exposed to the dynamic experiences described in Table I, the following effects were produced typically. Upon removal from the couch, the animals were still anesthetized and appeared to be in shock, demonstrating collapse, cyanosis, hyperpnea, tachycardia, flaccid muscles, and minimal or absent reflexes, all of which persisted for two to eight hours. This gave way to a conscious, but profoundly depressed state from which the animals roused reluctantly. The animals assumed a head-down crouch ("sick monkey position") which continued for varying periods, but the animals were usually quite well and obviously more comfortable twenty-four hours post-run, and were well seventy-two hours post-run. It should be noted that in the three cases when the animal was lightly anesthetized pre-run and conscious post-run, in all cases the animal quickly slipped into stupor and apparent shock. This suggests that anesthesia may not be a factor in producing the stupor-shock syndrome.

Neurologic signs usually accompanied the "sick mon-

TABLE II.

Experiment Number	Animal Number	Weight change in per cent, Pre-run versus Necropsy	Death, Days Post-run S = Sacrifice	Gross Pathology, Post Mortem: Hepatic	G.I.	Pulmonary	Cardiac	CNS	Adrenals	Other
001-025-008.00	1	–	–	Shakedown run—data not reliable.						
002-026-005.00	2	–	14S	– *	–	–	–	–	–	–
003-023-005.00	3	– 10.6	14S	–	–	++	–	–	–	–
004-096-012.00	5	– 13.5	12S	–	–	+	–	–	–	–
005-209-018.00	6	+ 3.8	14S	–	–	++	–	–	–	splenic cyst
006-330-023.00	7	– 11.3	14S	+ **	–	++	–	–	+	pale spleen (exsanguination?)
007-405-020.00	8	– 10.3	2	+	–	+	+	++++	–	shock dehydration, cerebral edema
008-394-015.00	9	– 5.2	14S	+	–	–	–	+	–	–
013-430-020.60	14	– 7.0	14S	+	+	–	–	–	–	omental cysts
014-425-020.45	15	– 1.3	14S	+++	–	+++	–	–	–	–
015-408-021.15	16	– 4.9	14S	–	–	–	–	–	–	–
016-428-020.25	17	– 10.2	14S	–	–	–	–	–	–	–
017-440-019.59	18	– 2.8	14S	–	–	–	–	+	–	omental cysts, splenomegaly?
018-415-019.41	19	– 8.6	14S	–	–	–	–	–	–	–
019-404-018.94	20	– 2.0	14S	++	–	+	–	–	–	–
020-400-018.89	21	– 3.6	14S	–	–	–	–	–	–	–
021-416-019.83	22	– 6.4	14S	–	–	–	–	–	–	omental cysts
022-413-019.27	23	– 6.0	14S	++	–	+	–	–	+	–

* "–" sign here used means "no gross pathology"
** "+" sign here used means "discernible gross pathology" on a one-to-four "judgment unit" scale of increasing severity.

key position" and were those usually associated with vestibular disturbance. Included were: "scrambling" movements upon mechanical stimulation; vertigo (evidenced by the animals' need to grasp the cage, without the support of which they fell backward or to the left and back); falling to the left from the "sick monkey" position, and vomiting. Occasionally other signs were seen. For example, in one case, a "thalamus animal" effect was observed with the animal on its left side, the lower limbs in extension, and the upper limbs in flexion. Also on occasion, fine and gross extremity tremors were seen, but these manifestations were the exception rather than the rule.

The lethality of the accelerative experience and the evidence of pathology are summarized in Table II. This table shows only one animal, Number 7, died as a result of the accelerative experience. The cause of death was severe cerebral edema and dehydration (evidenced by sunken eyeballs, dry mucosa, loss of tissue turgor and loose skin). Not all sixteen remaining animals autopsied sustained injury due to the accelerative experience. Six animals had no visible pathology as a result of the accelerative experience fourteen days after the experiment, and five of these six experienced 400 G or more. Eleven evidenced some degree of injury varying from slight to severe. Hepatic and pulmonary damage were most frequent, occurring seven and eight times, respectively, with the pulmonary damage judged to be slightly more severe. Central nervous system damage was seen twice, adrenal hypertrophy was seen twice, and gastrointestinal and cardiac damage were seen once each.

The most noteworthy single result was the electrical arrest of the heart with spontaneous resumption after removal of the G forces. Spot checks with a cardiophone indicated that the contractions of the heart stopped when loads of more than 200 G were applied.When the accelerative loads began to approach 400 G, a progressive electrobradycardia occurred, accompanied by reduction in heart voltage amplitudes. The reduction in ECG frequency and amplitude became progressively greater above 400 G and in six cases apparently complete cessation of all electrical discharge from the heart occurred coincident with the peak G load. This arrest was typically followed by a spontaneous resumption of the electrical activity characterized by: persistent bradycardia; gradual increase of electromyopotentials to normal levels, and then an increase to supranormal amplitudes which persisted; and absence of the fibrillation which sometimes accompanies reinitiation.

Table III summarizes the occurrence of electrocardiac arrest and the percentage reduction in frequency of the heart beat during three seconds at peak accelerative load, relative to the baseline rate. Figure 4 illustrates a typical electrocardiac arrest.

Table IV summarizes the results of the spin control

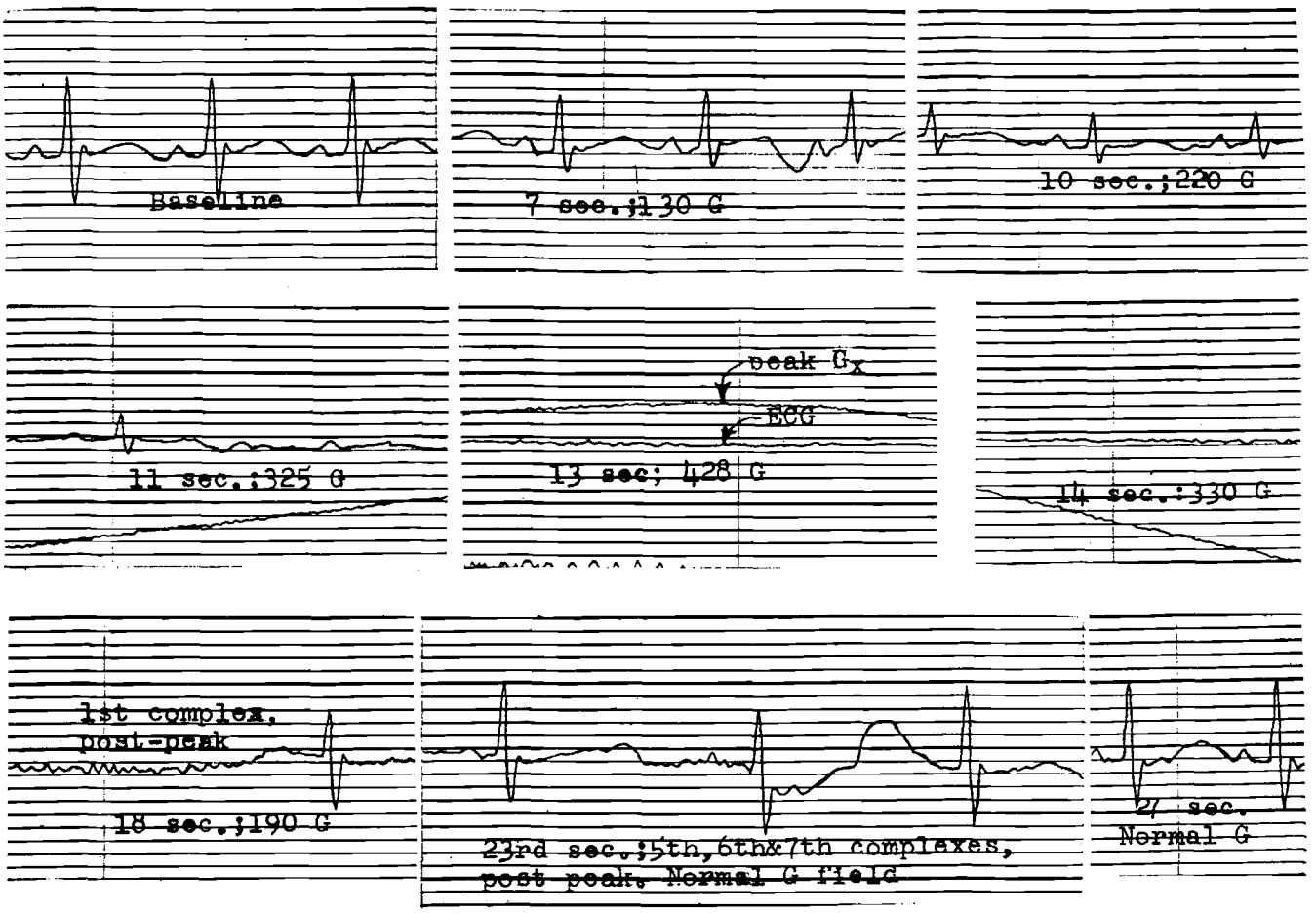

Fig. 4. ECG changes during Experiment 016-428-2025. Samples are at 7, 10, 11, 13, 14, 18, 23 and 27 seconds after start of run. Accelerations are as noted on the samples. The post-run bradycardia and amplitude increase are not pronounced here as in some.

TABLE III.

Experiment Number	Arrest	Per Cent Rate Reduction
007-405-020.00	No	27.0
008-394-015.00	Yes	100.0
013-430-020.60	No	65.5
014-425-020.45	Yes	100.0
015-408-021.15	Yes	100.0
016-428-020.25	Yes	100.0
017-440-019.59	Yes	100.0
018-415-019.41	No	13.7
019-404-018.94	No	35.3
020-400-018.89	Yes	100.0
021-416-019.83	No	13.7
022-413-019.27	No	65.5

series. Almost no gross pathology was found in these animals except for the pulmonary petechia and adrenal hypertrophy found in Animal M-1. The data from M-1 is questionable because the animal died during what was probably a colony epidemic. The findings may be related to a disease process rather than radial acceleration. The typical response to the spin control included: strong vertigo, pronounced nystagmus persisting for 15-20 seconds, moderate depression, some difficulty in arousal, and occasional assumption of the "'sick monkey position," the latter persisting for 2 to 24 hours. The shock and stupor always seen as a result of high G loads was never seen in the controls.

DISCUSSION

When squirrel monkeys were exposed to the time-acceleration environments used in this study, death was an infrequent response, occurring but once in seventeen experiments, and the injuries produced in the remaining animals were not really severe. Pulmonary and hepatic damage were most frequently encountered and the etiology probably can be related to organ density and displacement.

The lungs, the least dense organs in the body, offer little structural resistance to accelerative loads and little support to the thoracic cage. Possibly the cage distorts badly and presses the lungs against the posterior ribs and into the costovertebral gutter or the fluid trapped in the pulmonary circulation is displaced, distorting the tissue. This deformation and compression against the bony structures probably ruptures pulmonary capillaries, resulting in minute petechial hemorrhages. The subpleural bloody effusions may be caused by the coalescence of many of these petechia, since in no case was a really organized clot seen. The deformation of the lungs may also be responsible for the atelectatic consolidations observed. The mechanism here may be that compression of the lungs is actually severe enough to cause the bronchiolar and alveolar inner surfaces to touch. The elastic forces tending to keep the alveoli and bronchioles open is probably less than the adhesive forces tending to keep them approximated once they have touched. This mechanical compaction is aggravated by the uptake of oxygen in the resulting sealed spaces. The oxygen uptake produces a negative pressure, further solidifying the lung. Thus the lungs, once "pressed flat" by accelerative loads may have difficulty in re-expanding, resulting in a functional compression atelectasis.

The damage to the liver probably can be explained on the basis of distortion due to displacement. Because the loads are delivered $+G_X$, we envision an upward and backward displacement of the liver against the diaphragmatic dome. This would cause the hepatic borders to be under more stretch than the mass of the lobes near the falciform ligament and would explain the split hepatic margins and the yellow and purple bruise-like "stress lines" radiating from the ligament to the borders.

The two single examples of hemorrhage into the small intestine and into the anterior mediastinum can be explained by displacement distortions exceeding the stretch modulus of the local vessels.

The hypertrophied adrenals are probably a response to the generalized stress represented by caging, capture, and acceleration.

The three examples of central nervous system pathology are less easy to explain. In one case, the superficial vessels were engorged and in two others the brain was thought to be edematous. It cannot be doubted that the brain is distorted by the accelerative forces, and at a rate of distortion different from the skull. It can be speculated that this distortion is enough to permit leakage of non-formed elements of the blood into the brain from the cerebral capillaries without evident bleeding.

The electrical events of the cardiac cycle were abolished in one-half of all the animals exposed to 400 or more $+G_X$. (One of the arrests was seen in Experiment 8, which reached only 394 G, but this is close enough to 400 G to be classed with the true "400 and over" expo-

TABLE IV.

Experiment Number	Animal Number	Weight change in per cent, Pre-run versus Necropsy	Death, Days Post-run S = Sacrifice	Gross Pathology, Post Mortem: Hepatic	G.I.	Pulmonary	Cardiac	CNS	Adrenals	Other
000-Spin Control	M-1	- 1.0	204	-*	-	+**	-	-	+	-
000-Spin Control	M-2	+ 2.0	25S	-	-	-	-	-	-	-
009-Spin Control	10	- 5.2	14S	-	-	-	-	-	-	-
010-Spin Control	11	+ 4.5	14S	-	-	-	-	-	-	Pale spleen (exsanguination?)
011-Spin Control	12	+ 4.5	14S	-	-	-	-	-	-	-
012-Spin Control	13	+ 1.0	14S	-	-	-	-	-	-	-

* "-" sign here used means "no gross pathology."
** "+" sign here used means "discernible gross pathology."

sures.) The mechanism by which this occurs is obscure, but before discussing a possible answer, it should be said that this event is not an artifact. Instrument error was suspected immediately. We thought the electrodes might have slipped or distorted, or the instruments' electronic components might have been affected by the accelerative forces. Accordingly, an electromechanical ECG signal generator was fabricated, placed in the couch, and instrumented with the usual instrumentation. Runs above 400 G showed only a 5-7 per cent reduction in signal amplitude and no diminution in signal frequency. This control demonstrates that the electrocardiac arrest seen is physiologic in origin and not an instrument artifact.

The mechanism of arrest, as already observed, is obscure. It is known that the intrinsically rhythmic mammalian sinoauricular node maintains its membrane potential and undergoes depolarization by the same mechanism as other cell varieties. We do not know how this intrinsic rhythmicity is suppressed by accelerative forces. It may be that the dimensional morphology of the nodal cell is related to the ability of the cell to maintain the ion imbalance required to generate a membrane potential. On this basis, it can be hypothesized that the cell has certain critical internal distances which are involved in the "ion-pump" mechanism and that, when these distances are disturbed by distorting mechanical forces, the "ion-pump" no longer works and the intrinsic rhythmicity is abolished. In this scheme, when the distorting load is removed, the inherent elasticity of the cell restores the critical distance relationships and the potential can be reestablished and then the cell depolarizes as usual. This essentially mechanistic hypothesis explains why the rhythm can return without extrinsic stimulus or without the fibrillation sometimes encountered after "physiologic" suppression of nodal activity. Other explanations are possible of course, but this hypothesis seems to fit the observations better than others. The theory does not explain why it only happens in half the animals tested or predict what will happen if the same loads are continued for long periods or more load is applied for the same period.

Finally, consider the organism in its entirety and its ability to accept accelerative loads. Monkeys have good inherent tolerance to impact-type accelerations, but their ability to deal with conventional G loads is not remarkable. Thus it was enlightening to observe these unprotected animals accept ever higher G-time experiences without greater mortality and without evidence of really major trauma. Certainly they were severely affected; cardiovascular collapse, loss of consciousness, and severe vestibular effects were all seen, but the animals survived and were usually well soon. The explanation for this exceptional acceleration durability must be speculative until more experiments are performed to determine the force-time situations which will produce death or significant non-lethal damage. Finding out what forces are required to kill these animals may illuminate how they survive these lesser, but still formidable, accelerations.

SUMMARY

Squirrel monkeys were exposed to unconventionally high force-time profiles on a unique articulated accelerator. Thirteen tests above 300 $+G_x$ over approximately 20-second time bases were conducted of which eleven exceeded 400 G. One death and some injuries occurred, and a characteristic general response, including cardiovascular and neurologic signs was observed. The possible mechanism of the injuries is discussed. The electrocardiac arrest with spontaneous reinitiation of rhythmic contraction is also discussed. Additional work must be done to investigate further the response of primates to force-time situations of similar dimensions to establish the basis for their survival.

ACKNOWLEDGMENTS

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