Reappraisal of Biodynamic Implications of Human Ejections

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Vertebral compression represents a significant percentage of the morbidity associated with upward ejection. Vertebral and intervertebral structure reacts to and is sometimes irreversibly altered by ejection acceleration. Design and material properties of the normal vertebral column are sufficiently constant that when structural characteristics are defined and acceleration profiles known, prediction of failure may be made. Compressive load analyses of vertebra-disc complexes have demonstrated that the vertebral endplates are the initially failing structures of the spinal column. From experimental data on vertebral breaking-loads, acceptably accurate probability-of-injury curves for static loading have been generated. These data together with data describing the dynamic response characteristics of the human body permit calculation of the probability-of-injury for dynamic loading produced by exposure to impact accelerations. As an aid to the designer of ejection systems, application of these concepts should refine the estimate of "safe" acceleration profiles and minimize the risk of irreversible vertebral deformation.

SINCE THE ADVENT of aircraft ejection seat escape systems some twenty-five years ago, it has become apparent that injury to the spinal column represents a significant percentage of the morbidity associated with this mode of aircraft-pilot separation. In our present era of high performance aircraft and space travel, the problems of spinal injury persist and the efforts to design escape systems which will minimize trauma to the vertebral column have become increasingly complex. As more advanced escape systems and landing vehicles are utilized, force orientations upon the vertebral column become more variable. However, even with the injury potential of severe body twisting, rotation, and flaying resulting from the varying force directions, compression spinal fracture will probably always be an escape system problem. Review of aerospace literature reveals that relatively little effort has been expended to describe the actual sequential anatomic alterations which occur within the spinal column during ejection acceleration. Similarly, there exists little information regarding the potential long-term sequelae of injuries incurred as a result of exceeding vertebral structural tolerance. The purpose of this report is to describe the interrelated response of the vertebral bodies and intervertebral discs during static mechanical loading and during ejection acceleration and also to allude to the potential implications of unrecognized or undetected vertebral injury.

SPINAL COLUMN ANATOMY AND PHYSIOLOGY

The human spinal column is structurally comprised of a boney, cartilaginous and ligamentous complex (Figures 1 and 2) which flexibly supports the entire body and protects the spinal cord.[19] Thirty-three boney vertebral elements are separated from one another by the fibro-cartilaginous intervertebral discs which, along with vertebral joint capsules and ligaments, serve to stabilize successive vertebrae. The cephalic and caudal surfaces of adult vertebrae contain two important boney components, the "vertebral endplates." During early life these boney plates are perforated by nutrient blood vessels which by adulthood are no longer required. Fibro-cartilaginous tissue then obliterates the original channels which, however, persist as nonosseous areas of fundamentally osseous vertebral body components. Structurally the osseous tissue of mature vertebral bone is compose dof mineral apatite dispersed throughout a protein collagen matrix. Whereas apatite is characterized by relatively high compressive strength, protein collagen has relatively low stiffness. As a combined

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substance these inorganic and organic constituents yield a material with relatively high compressive-tensile stiffness properties. As bone ages, elasticity increases and compressive strength decreases. As a consequence of this fact, older vertebrae are characterized by lower stiffness values.[15] Although one is tempted to attribute such diminished stiffness and compressive strength to the decreased amount of compact bone present in older specimens, we should appreciate that other more subtle, nonapparent biochemical changes are also occurring. The altered strength characteristics of older vertebrae is in actuality probably a result of changes in both of these dynamic parameters.

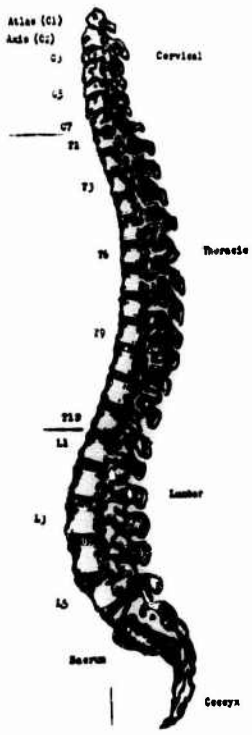


Fig. 1. Isolated human spinal column. The cervical and lumbar curves of the correctly postured spine are convex anteriorly whereas the thoracic curve is convex posteriorly.

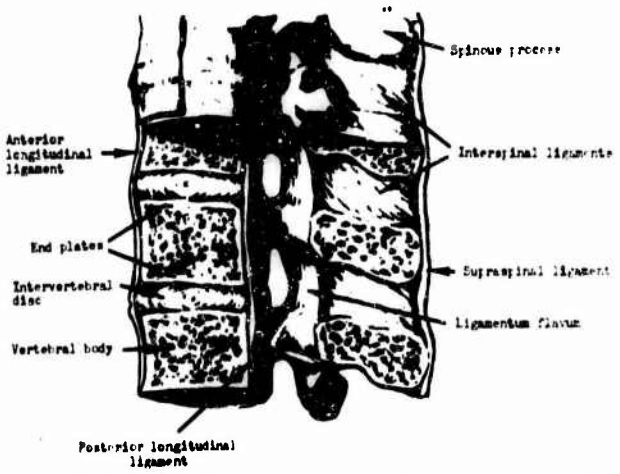


Fig. 2. Anatomy of intervertebral stabilization. Each vertebra has an anterior, weight-bearing portion, the vertebral body, and a posterior arch which shields the spinal cord and serves as an attachment point for the powerful back muscles. The anterior and posterior longitudinal ligaments are inherently attached to the vertebral bodies and the discs. As illustrated, the posterior arches are also firmly attached to one another by specialized elastic ligaments.

Between successive vertebral bodies are positioned fibro-cartilaginous intervertebral discs (Figure 3) which constitute from ¼ to ¼ of total spinal column length. Each disc is made up of three distinct but anatomically combined "parts." The *annulus fibrosis* is composed of concentrically layered fibro-elastic tissue which is inherently attached to the adjoining vertebral bodies. Within the annulus lies a watery gel, the *nucleus pulposus*. This component part of the disc is not in the anatomic center of the annulus, but lies slightly posterior where it aligns with those areas of adjacent vertebral endplates which represent the central pressure points of their respective vertebral bodies. The third anatomic entity of each disc is a pair of fine hyaline cartilage *plates* which are derived from the annulus and which form borders between the nucleus of a disc and the osseous endplates of adjacent vertebral bodies.

The human vertebral column provides man with postural stability, flexion, extension, lateral bending, and rotational capability. The four prominent curves of a normal adult spine are arranged in an alternating convex-concave arrangement which contributes to the overall spinal elasticity. This also results in superb positional flexibility and a greater facility for dynamically maintaining the center of gravity which, in stationary upright man, lies just anterior to the sacral promontory.

Movement between vertebrae takes place through both the resilient intervertebral discs and the joints of the posterior arch articular processes. Although actual displacement between vertebrae is relatively small, total column motion, as outlined above, is considerable.

Pressure absorbed by a normal nucleus is hydrostatically distributed over both the adjacent endplates and the internal aspect of the annulus. The changes which a disc undergoes during life, however, alter its functional properties. Whereas the moisture content within the nucleus of a "newborn" approximates 88 per cent, it diminishes to around 68 per cent in an elderly individual.[8] Subsequent to such change, disc

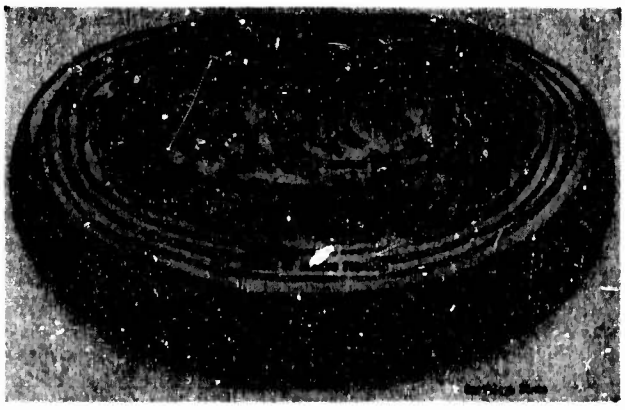


Fig. 3. Schematic illustration of an isolated intervertebral disc.

mobility is reduced and pressure transmission to adjacent vertebra changes in the manner in which it occurs. Another process, disc "degeneration" per se, occurs subsequent to both decreasing nuclear fluid content and the simultaneously occurring wear and tear of constant load stress and strain. Although "normal" for its age, the elasticity of a degenerated disc is reduced. The greater the degree of nuclear dehydration and degeneration, the greater is the proportion of the pressure that must be supported by the annulus. With proper posture and sensible load-onset rates, a healthy spine will support remarkable static and dynamic forces. If either of these factors is ignored or poorly respected, however, vertebral body or intervertebral disc injuries can be anticipated to occur.

SPINAL STRUCTURE UNDER STATIC MECHANICAL LOADING

The passive mechanical response of the system to external environmental forces is manifest by the development of internal stresses, which if of sufficient magnitude, will alter the structure, form, and functional capability of the object. More simply stated, externally applied force results in internal structural forces which ultimately will cause failure. Damage to biologic tissues resulting from mechanical forces must take place in essentially the same manner that damage to nonbiologic structures occurs as a result of such forces. If the structural characteristics of a biological material can be defined, and the magnitude and time function of a particular force applied to that material can be described, one should be able to predict whether or not failure will occur. In the investigation of material strengths, different types of applied force and different types of failure can be described. Our primary concern in this report is mechanical force directed parallel to the spinal axis. Vertebral compression fractures occurring during ejection are a direct result of such mechanical force. Definition of the occurrence of compressive failure is dependent upon specific characteristics of stress-strain (load-deflection) curves that are generated during compression testing of various materials. Three characteristics of these curves are of primary importance. These are *proportional limit*, *yield point*, and *total failure* as illustrated in Figure 4.

Statistical variability exists in the mechanical properties of all structures. We anticipate that strength analysis performed on tissues like cartilage and bone, which are continually undergoing dynamic biochemical change, will present variable results. If, however, analyses are performed on a number of particular specimens from individuals of the same sex, age, weight, general build, and degree of health, results should follow a normal, bell-shaped type of curve. The stress-strain curves for biologic materials are nonlinear and present certain analytical difficulties. In order to avoid complex mathematical analysis in such cases, one usually makes linear approximations which may set certain limitations upon the validity of calculated results.

All of the above, of course, implies structural analysis of human cadaveric material and indeed, structural analyses have been performed on human spinal columns. If such data yields valid compressive breaking load levels for the vertebrae, living humans who are exposed to loads in excess of these levels can be anticipated to incur injury.

Turning now to the injury modes in vivo, it is prudent to make a point concerning tolerance and tolerance levels. Subjective tolerance per se and objective tissue tolerance should be separate concepts. Here, we are interested in tissue tolerance. Human tolerance levels for spinal axis ejection forces must be accepted as those levels *just above which* irreversible damage will occur within the structurally most susceptible component of the spinal column.

The mechanism of absorption of compressive forces by the vertebra-disc complexes of the spinal column is fascinating. Both Brown,[3] et al. and Roaf[12] noted that early during slow column compression there is a decrease in disc volume which ranges between 1 and 2½ cm³ before one of the adjacent vertebral endplates fractures. Recognizing the fluid-retaining capability of the disc annulus and the structural porosity of the endplates, these investigators believed that this diminishing disc volume was due to two sequentially occurring events. Early during significant spinal compression, sinuses, fissures, and micro spaces, normally present in all adult discs, collapse. The diffusible intra-

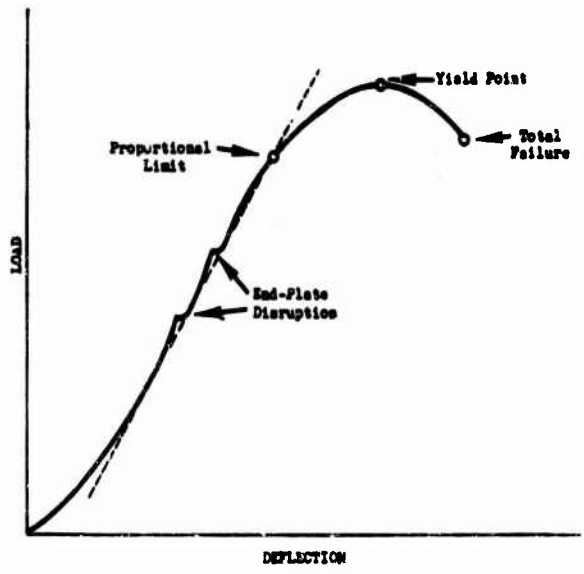


Fig. 4. Schematic load deflection curve illustrating alterations in vertebral body structure during compressive stress.

"Proportional limit" defines elastic capability and is a representative estimate of the point at which a material begins to fail but is able, upon relief of the force, to recover its preload form free of recognizable structural damage.

"Yield point" defines irreversible deformation and is a representative estimate of that point at which a material is permanently altered in form and, though is still capable of supporting a load, will not recover its original shape subsequent to load relaxation.

"Total failure" defines material disruption and is representative of that point at which a material crumbles or crushes to the extent that structural integrity is completely lost.

disc fluid displaced during this process is "pressured across" the vertebral endplates. With continued compression and as endplate bulging is occurring, the fluid within the cancellous bone of the vertebral body (both endogenous and that transposed from the discs) is forced out into paravertebral sinuses and veins. With spinal compression above the energy-dissipating capability of this fluid-transfer mechanism, vertebral endplate bulge increases to the point of fracture. Both Roaf and Brown noted that only after endplate fracture did compressive vertebral body damage occur. They emphasized that whether a disc was histologically normal or degenerated, the same sequence of events occurred. Neither discs with degenerated nuclei nor those specimens with annular bulge secondary to early fibrous tear, "prolapsed" prior to endplate fracture and vertebral body compression. One gains an appreciation from these investigations that the precise mechanism of energy dissipation across the disc will vary depending upon the age and condition of the specimen.

Brown, after testing older specimens, documented unsymmetrical annular bulge during compression which repeatedly occurred in the anterior (strongest) area of the disc. If such compressive bulge was occurring solely as a result of intra-disc hydrostatic effects, he believed the bulge should have been most marked in the weaker posterior and postero-lateral areas of the annulus. The fact that it was most pronounced at the strongest anterior area would be against a primarily hydrostatic mechanism and in favor of direct compression of the annulus itself by asymmetrical loading due to bending. There is clinical supporting evidence that intervertebral disc annuli are in themselves capable of support and energy dissipation. Schmorl's Node is an eponym for an X-ray evident nucleus pulposus which has ruptured through an endplate into a vertebral body where it became ossified. Its "enucleated" disc, however, still maintains significant intervertebral space. We are also aware that during surgical intervention for disc "herniation," a fair amount of extruded material may be found with a minimally altered disc space. In each of these situations there is a loss of nucleus without total loss of the intervertebral space. The only structure capable of maintaining this support is the annulus of the disrupted disc.

It would appear, therefore, that although the nucleus potentiates intervertebral disc support and probably some energy-dissipating capability, it is not a requisite for either of these functions which may be handled by the annulus. It becomes apparent that the water content of a disc nucleus is related to its mechanical efficiency. In a normal disc, therefore, internal pressure is hydrostatically distributed to both annulus and vertebral endplates. This sequence results in an intervertebral pressure transfer by a highly efficient disc utilizing primarily nucleus but also the annulus. In a degenerated disc with a depleted fluid content, however, a greater proportion of the energy transmitted must be absorbed by the annulus.

Having some understanding of the anatomic biomechanics of spinal column energy transmission, we should review the significant contributions that have been made to experimental spinal biomechanics. These began in 1940 with Ruff[13] who was interested in the determination of the breaking strength of vertebrae under axial compression. In subjecting fresh cadaveric vertebral specimens to static compression loading, Ruff calculated breaking strengths from the point of the load-deflection curve at which the first peak occurred. Recalling that this represents the "yield" (irreversible deformation) type of failure, and simultaneously appreciating the "height-maintaining" and "weight-supporting" functions of vertebrae, we realize that this type of failure documentation is both clinically and biodynamically highly significant. After testing a number of vertebra-disc complexes, Ruff became aware (as Roaf and Brown later confirmed) that the vertebral body always broke before the adjacent disc incurred discernible damage. Realizing that individual vertebral body force transmission during acceleration is dependent upon the body weight supported by that particular vertebra, Ruff ascertained the per cent of total body weight supported by the individual vertebral bodies. Table I presents Ruff's data on breaking loads for T8 to L5 vertebra and also his calculated per cent-of-body-weight supported by these successive vertebrae. Extending his experimental breaking strength and per cent-body-weight-supported data, Ruff derived

TABLE I

Vertebra	Breaking Strength (kg Force)	Average (kg Force)	Average (lbs)	Per cent Body Weight Supported
T8	640, 540, 609	593	1315	33
T9	610, 720, 700	677	1493	37
T10	800, 660, 770, 730	740	1632	40
T11	750, 720, 860, 755	771	1700	44
T12	900, 690, 800, 800	797	1757	47
L1	720, 840, 900, 800, 800	812	1790	50
L2	990, 800, 830	873	1925	53
L3	900, 940, 1100	980	2161	56
L4	1100, 900, 950	983	2168	58
L5	1020, 1000, 1200	1073	2366	60

TABLE II. CALCULATED VALUES FOR A BODY WEIGHT OF 75 KG

Vertebra	Max Breaking (kg Force)	Min Breaking (kg Force)	Per cent	Max G*	Min G*
T8	640	540	33	24.9	20.8
T9	720	610	37	25.0	21.0
T10	800	660	40	25.7	21.0
T11	860	720	44	25.1	20.8
T12	900	690	47	24.5	18.6
L1	900	720	50	23.0	18.2
L2	990	800	53	23.9	19.1
L3	1100	900	56	25.2	20.4
L4	1200	900	58	24.3	19.7
L5		1000	60	25.7	21.2

*Utilizing the formulae.

$$G_{max} = \frac{100 \times P_{Br}^{max.}}{E \times W}$$

$$G_{min} = \frac{100 \times P_{Br}^{min.}}{E \times W}$$

Wherein P_{Br}^{max} and P_{Br}^{min} are the highest and the lowest breaking loads noted in initial testing; E is the per cent of body weight carried by individual vertebra; W is the body weight.

maximal and minimal G load tolerances for individual vertebrae by assuming that all specimens were representative of those tested from the spinal column of a 75 kg man. These values together with his formula are presented in Table II. The final portion of Ruff's investigation dealt with acceleration-time histories. He concluded that for exposure periods of 5 milliseconds to 1 second, structural tolerance is determined by the static compressive strength of the vertebrae most easily traumatized by such loading. For acceleration pulse durations lasting less than 5 milliseconds, structural tolerance, Ruff also concluded, is determined by the dynamic strength of the most susceptible vertebra. Figure 5 graphically illustrates the "G-time" tolerance levels derived by Ruff.

The second outstanding biomechanical investigation of the spinal column was that which Perey[11] of Sweden published in 1957. His analyses differed from those of Ruff in three ways: (1) he utilized "proportional limit" instead of "yield" point, (2) he did dynamic as well as static loading and (3) his specimens were more exactly representative of specific anatomic entities than were Ruff's. In utilizing proportional limit, Perey's estimates of vertebral strength are naturally anticipated to be lower than Ruff's. Although they would appear to be safer by virtue of the definition of proportional limit, i.e., reversible "damage," we should appreciate that Perey noted that fracture of the anatomically distinct vertebral endplate occured *at levels below the vertebral body proportional limit.* By injecting radiopaque media into discs of test complexes and then taking roentgenograms of dynamic compressive alteration, Perey was able to document the "weakest links" at the moment of damage. During one group of dynamic loadings to the proportional limit, Perey identified twenty instances of vertebral endplate fracture as compared to six instances of irreversible vertebral body compression. Perey warned that many of these endplate fractures could not be visualized on X-rays that were experimentally analogous to the routinely obtained views in the clinical situation. Many of these X-ray "misses" were documented rather easily, however, by discography and laminography. Subsequent to his dynamic testing, Perey investigated static loading of two and three vertebrae complexes. In 40, two vertebrae specimens subjected to static compression, a definite relationship between age and "proportional limit" was noted. For vertebrae over age 60, average "breaking strength" was 425 kp (935 pounds force). Endplate fractures in the static test of excised specimens composed of two vertebral segments were microscopically evident in 13 instances (32 per cent). Following his preliminary dynamic and static testing, and appreciating the significance of the difference in breaking strength between endplates and vertebral bodies, Perey was naturally interested in comparing these two sets of values. Tables III and IV illustrate average results obtained. In this portion of his investigation Perey also ascertained that vertebral bodies compress an average 16 per cent of their total height before the proportional limit is reached. Realizing that the actual point of vertebral body damage lies closer to the yield point (Perey was testing to proportional limit), we are able to appreciate that in reality greater than 16 per cent reversible compression probably occurs prior to vertebral body damage. Also, no matter how fresh cadaver specimens are, there has been some fluid loss. Consequently, for the in-vivo case there is probably still another added increment of reversible compression prior to fracture. Finally, Perey was able to ascertain from his investigation that endplate strength is similar in peripheral and central areas. This helps explain the lack of any particular uniformity to the area of endplate failure that occurred during his testing.

Two facts gleaned from Perey's work stand out as being particularly important in the problem of human ejection: *endplate fractures occur at lower level loading than is required to reach the proportional limit and by the time 16 per cent compression of a vertebral*

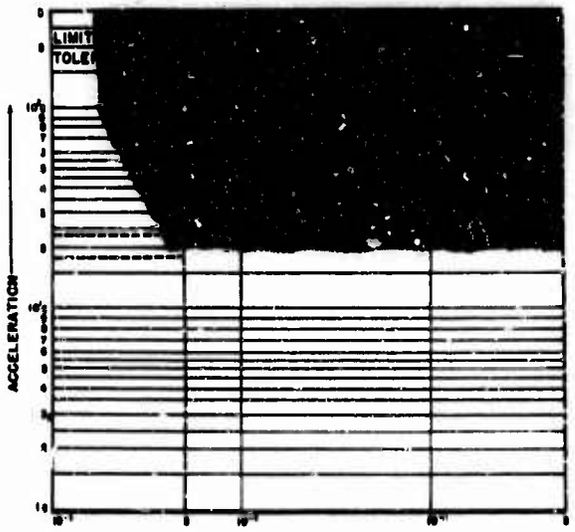


Fig. 5. Ruff's dynamic and static vertebral column tolerance as related to acceleration profile.

TABLE III. LUMBAR VERTEBRAL BODY RESISTANCE WITH RESPECT TO AGE

Vertebra	Under 60 yrs		Over 60 yrs	
	Kiloponds	Pounds	Kiloponds	Pounds
L1	520	1144	270	594
L2	600	1320	260	572
L3	635	1397	250	550
L4	650	1430	270	594
L5	590	1298	240	528

TABLE IV. MEDIAN BREAKING POINTS (KILOPONDS PER SQUARE CENTIMETER AND POUNDS PER SQUARE INCH) FOR 223 VERTEBRAL ENDPLATES TAKEN FROM SPECIMENS OF L1 THROUGH L5

Age (yrs)	Median Breaking Point	
	kp/cm²	*psi*
20-30	107	1530
31-40	98	1400
41-50	76	1085
51-60	77	1100
>60	43	614

body has occurred, one or both endplates have usually exceeded their breaking points. One can conclude that acceptance of "yield point" as being equivalent to irreversible compressive deformity implies even greater differences between endplate and vertebral body breaking points than Perey ascertained with proportional limit criteria. We can begin to appreciate not only that endplate disruption occurs at levels appreciably below irreversible vertebral body compression, but also (and of greater concern) that a number of spinal column endplates may incur "loss of structural integrity" prior to demonstrable fracture of the most susceptible vertebral body. When transposed to the live ejection situation, this bit of knowledge takes on pertinent and important clinical implications.

The investigations of Ruff, Perey, Roaf and Yorra[20] have led us to realize that endplate and vertebral body damage is far more apt to occur during spiral axial loading than is intervertebral disc disruption. This appears to be substantiated by the infrequent reports of disc trauma contained in the ejection literature.

Stech[14] has recently extended Ruff's original experimental data to calculations on the remaining thoracic vertebrae. This author, appreciating that Ruff found a relatively constant increase in per cent of body-weight-supported by each successive vertebral segment from T8 through L5 vertebra, postulated that the same relationship exists upward from T8. Extrapolating upward in a constant 3 per cent decrease per vertebrae, Stech arrived at a 9 per cent value for T1. The head and neck, which theoretically is all that T1 does support, has indeed been measured as being approximately 9 per cent of total body weight. By making what to us appears to be acceptable and relatively accurate approximations, Stech calculated both the breaking strengths of T1-T7 and the per cent-body-weight supported by these individual vertebrae. Table V presents Ruffs original T8-L5 data along with Stech's extrapolated T1-T7 values. Having these data, Stech utilized the concept of probability to define spinal acceleration tolerance (injury) levels. Utilizing this concept of relative probability of injury, one can determine levels up to which the incidence should be very low and above which the incidence of injury can be expected to increase rapidly with each added increment of acceleration. We are in agreement with his cautioning statement that, "the most important fact to be realized, appreciated, and respected with regard to tolerance curves and injury probability is that the levels represented are *risk* levels." Due to the variables which he realized affected the data gathered by Ruff and Perey, Stech was obligated to make certain necessary assumptions in constructing his risk curves. As a consequence, Stech estimated mean breaking level values on the low side and utilized variances that are almost assuredly higher than the actual unknown variances. Table VI represents Ruff's individual vertebra

TABLE V

Vertebra	Weight Carried: Per cent Body Weight Carried	Weight Carried: 160 lb man in Pounds	Breaking Strength in Pounds	Breaking Load in G
T1	9	14.4	360	25
T2	12	19.2	480	25
T3	15	24.0	600	25
T4	18	28.8	720	25
T5	21	33.6	840	25
T6	25	40.0	1000	25
T7	29	46.4	1160	25
T8	33	52.8	1315	24.9
T9	37	59.2	1493	25.2
T10	40	64.0	1632	25.5
T11	44	70.4	1700	24.2
T12	47	75.2	1757	23.4
L1	50	80.0	1790	22.4
L2	53	84.8	1925	22.7
L3	56	89.6	2161	24.1
L4	58	92.8	2168	23.4
L5	60	96.0	2366	24.6

TABLE VI. RUFF'S DATA IN REDUCED FORM

Vertebra	Average Strength (kg Force)	Standard Deviation (kg Force)	Coefficient of Variation
T8	534	16.5	32.2
T9	618	40.8	15.1
T10	647	69.5	9.3
T11	688	49.1	14.0
T12	706	35.4	12.7
L1	721	47.7	15.1
L2	761	75.6	10.1
L3	862	70.9	12.2
L4	855*	—	—
L5	898	72.7	12.4

*Single data point.

TABLE VII. RUFF'S DATA IN FINAL REDUCED FORM

Vertebra	Average Strength (Pounds)	Standard Deviation (Pounds)
T8	1175	79.6
T9	1363	92.2
T10	1427	96.4
T11	1517	102.5
T12	1557	105.2
L1	1580	107.4
L2	1678	113.3
L3	1901	128.3
L4	1940	131.2
L5	1980	133.8

TABLE VIII. PEREY'S DATA IN STECH'S FINAL REDUCED FORM (FOR AGE 27.9)

Vertebra	Mean Breaking Strength (Pounds)	Standard Deviation (Pounds)
L1	1266	362
L2	1383	395
L3	1395	399
L4	1415	404
L5	1661	475

TABLE IX. DISTRIBUTION OF ENDPLATE BREAKING STRENGTH (STECH)

Vertebra	Mean Breaking Strength (Pounds)	Standard Deviation (Pounds)
L1	982	280
L2	1063	305
L3	1112	316
L4	1178	333
L5	1194	343

data which Stech corrected for age, location in the spinal column, and body weight. This table also contains an estimate of the standard deviations for these vertebrae derived on the basis of the number of specimens that Ruff tested. Table VII gives this same data in pounds with the standard deviations re-estimated utilizing the average coefficient of variation. Stech then did a similar analysis on Perey's vertebral body and vertebral endplate breaking strength data. Tables VIII and IX present vertebral body and vertebral endplate breaking strengths for L1 through L5 together with their respective standard deviations, corrected for a 28-year-old specimen. Figure 6 shows Stech's curves for T8-T12 vertebrae describing the probability of damage in response to applied steady state acceleration loads. Figure 7, which presents simultaneously plotted curves for L1 to L5 proportional limits, compressive limits, and endplate limits, graphically demonstrates one of the points that we have been striving to emphasize. *Endplate fractures occur at load levels significantly below those required to produce compressive vertebral body fracture.* Stech recognized that his curves are representative of the response of particular vertebra for a specified age group and could not be directly applied to estimate hazards in the operational situation. In the final analysis of available data, Stech,

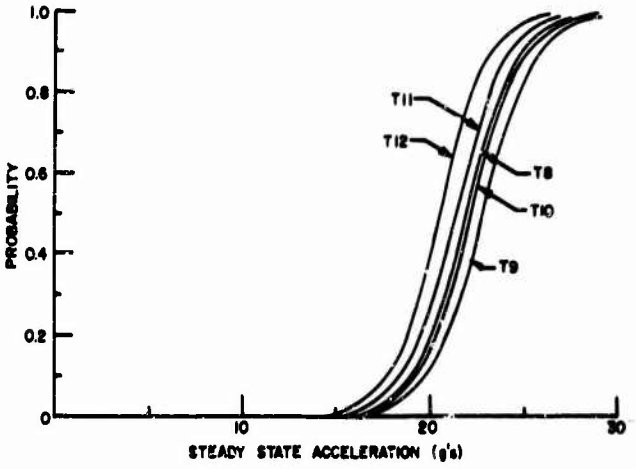


Fig. 6. Probability of damage for T8-T12 vertebrae during steady state acceleration.

appreciating the high incidence of T12 and L1 ejection fracture but simultaneously realizing that other vertebrae do fracture, wished to transpose probability of injury curves for single vertebrae to information on the entire column. Since this total susceptibility to injury is variable and probability of injury curves for T12 and L1 cover the majority but not all cases, one

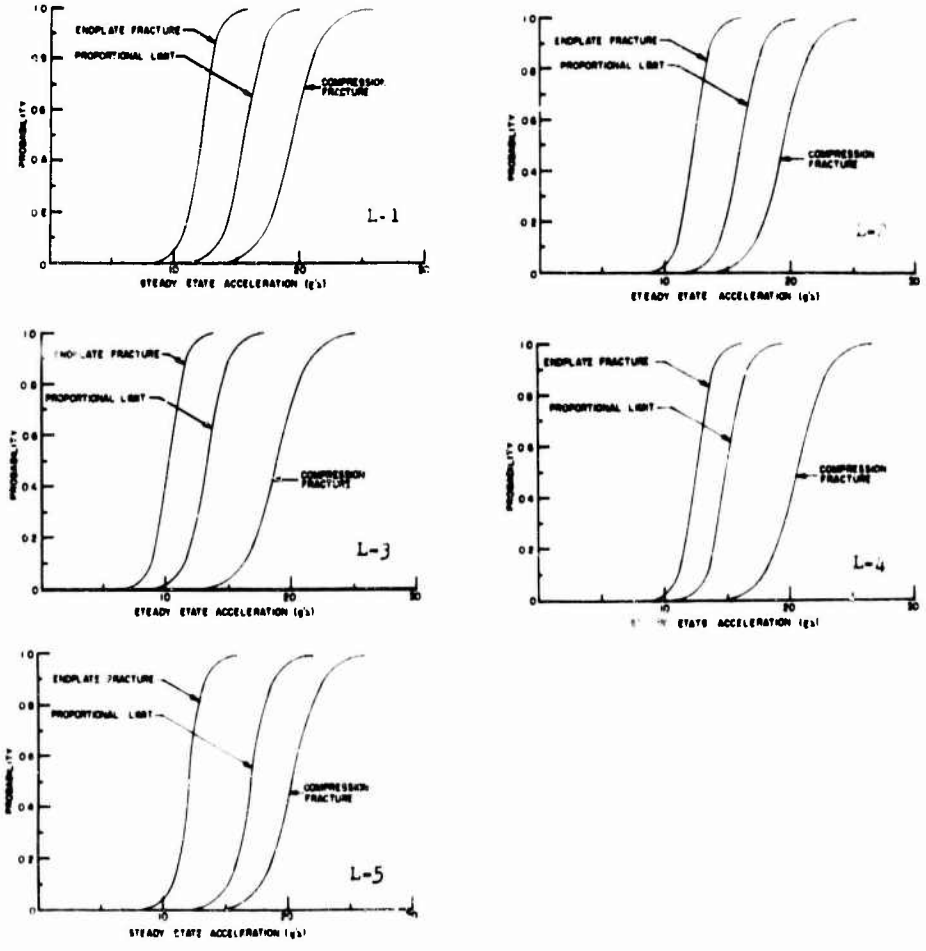


Fig. 7. Stech's curves for L1-L5 vertebrae. Endplate fracture occurs at levels below compressive fracture levels.

can look at "the other side of the coin" and examine the probability of no injury. Utilizing the product of the probabilities of no injury for the entire seventeen thoracic and lumbar vertebrae, Stech calculated the probability of injury curves for the entire dorso-lumbar column. He then applied age specific corrections to derive overall spinal fracture risk figures for a population group representative of the aircrew population. Figures 8 and 9 illustrate these curves for "live" spinal columns estimated in an aircrew-representative population and for specific populations of ages 20, 25, 30, 35 and 40.

As we mentioned prior to describing these probability of injury curves, Stech estimated mean values on the low side and used variances that are probably higher than the true variances. We agree with his postulate that such curves probably indicate a higher probability of vertebral body fracture at a given acceleration level than is true in actuality. We are also in agreement that the curves should be used cautiously for probabilities below 0.1 or above 0.9 and that the age distribution of the potential ejectee population should be utilized to generate the operational curves for such group.

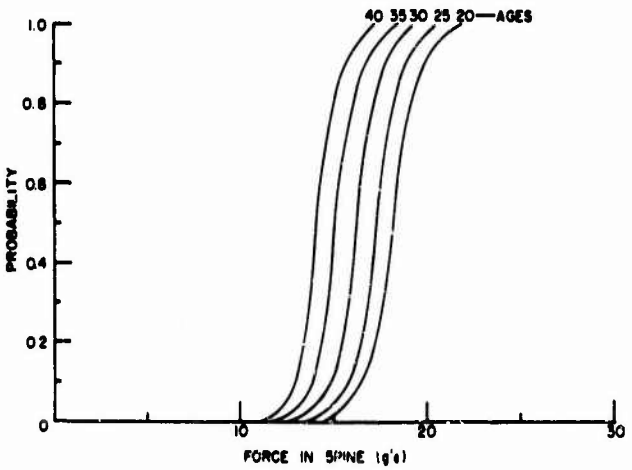


Fig. 8. Probability of injury for entire spinal column for specific ages.

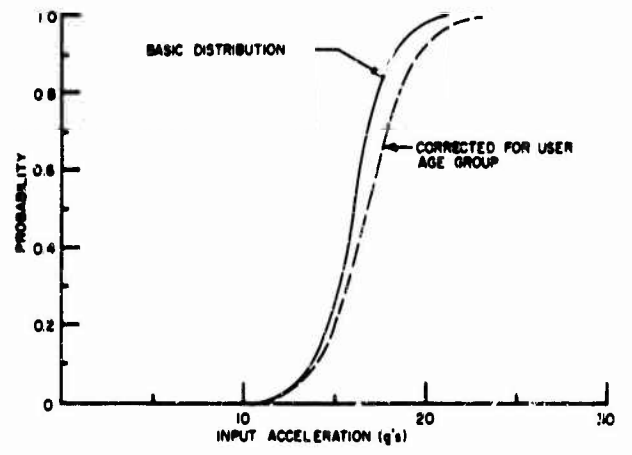


Fig. 9. Distribution of injury probability—basic and corrected for aircrew population age factor.

SPINAL STRUCTURE UNDER DYNAMIC MECHANICAL LOADING

The various types of failure of spinal structures were discussed in the previous section for the case of static mechanical loading. The probability of injury curves evaluated and presented are directly applicable only to steady state (sustained) acceleration loading of the spine in the head-to-foot direction. For transient acceleration-time patterns the dynamic loading of the spine as part of the overall dynamic response of the body must be considered.

By virtue of the structural composition of the human body, the vertebral column is part of an elastic system capable of a "dynamic response." The elasticity arises out of the flexion, compression, and expansion properties of biologic tissue. Being part of an elastic system, and being in itself elastic, the column in connection with the body masses coupled to it responds to high-onset accelerative forces transmitted to its caudal end by compression and bending and by subsequent expansion. Depending upon the rate of onset of acceleration during the initial phase of ejection, motion of the upper torso supported by the spine may lag the forced motion of the seat pan with accompanying spinal compression. Up to this stage, under such conditions the seat has a greater velocity than the upper torso, which afterwards requires that the upper torso undergo an acceleration which exceeds seat acceleration in order to reach terminal seat velocity. The additional component increases the inertial loading of the spine, resulting in additional spinal compression to the point where fracture may occur. The resulting "dynamic response" or overshoot can result in accelerations on parts of the subject that are higher than those on the seat.[6] The "overshoot" can be magnified, as has been observed and reported by many investigators, if an elastic seat cushion is placed between subject and seat pan. Since Latham's early work on the dynamic response function of seated human subjects, considerable progress has been made in the measurement, interpretation, and analytical expression of this dynamic response.[10,16,17] Today the injury potential of complex acceleration time functions, untested with respect to their biological hazard, is probably best evaluated by means of these analytical methods and the dynamic mechanical model concepts on which they are based. Special analog computers are available to calculate the dynamic response of the seated subjects when using different types of seat cushions or restraints.[10] It is not the purpose of this paper to review this area of impact research. However, assuming the general validity of the method, it can be readily shown that for a given spinal injury risk, the short duration impact limit is much higher than the static load limit if the duration of exposure is sufficiently brief. This conclusion is evident even from Ruff's early work as shown in Figure 5. The injury potential of acceleration-time patterns with long rise times is less (in a quantitatively predictable way) than the injury potential of patterns with short rise times and equal peak acceleration. Utilizing the injury probability curves for static loading discussed in this paper together with the present

knowledge on overall body dynamics, mathematical models have been derived by means of which the risk of spinal injury can be estimated for exposures to any particular acceleration environment produced by any specific ejection system.

OPERATIONAL EXPERIENCES AND CLINICAL FINDINGS

The following brief review of the acceleration levels that have been suggested and utilized during the past 25 years of ejection seat design and of injury statistics is included to emphasize the importance of considering both the static spinal injury mechanisms as well as the dynamic response concept.

Although early German development was fraught with complications, by 1945 their ejection velocities had substantially increased above earlier 8-9 meters/sec and they were tolerating 18-20 G peaks with relatively little documented spinal trauma. Swedish-catapults, designed between 1944-47, had a peak acceleration of approximately 21 G. With their seat, acceleration reached peak level in approximately 70 milliseconds, during which time the onset rate didn't exceed 300 G/sec. The velocity change of these systems was about 17 meters/sec. British experience began with the use of the Martin-Baker[6] ejection seat. In early 1945, after a compression fracture was incurred at a level below 12 G, critical investigation and analyses revealed that the onset rates of some early ejection tower exposures were frequently 600-800 G/sec. As a result of studying additional exposures at substantially lower onset rates the British, in late 1945, accepted the following parameters for catapult design: peak acceleration should not exceed 21 G, the time duration at peak acceleration should be less than 100 msec, and the onset rate should not exceed 250-300 G per second. U. S. ejection catapult acceleration specifications were defined between 1945 and 1947. Ames,[1] aware of the dynamic overshoot that occurs with high onset rates, cautioned during this period that any overshoot would be negligible if onset rates were held below 200 G/sec. In 1947 ejection seat equipment developed by the Army Air Forces and the Ordinance Department provided a terminal velocity of 60 ft/sec with a maximum of 14-16 G on the subject at a rate-of-application of 175 to 200 G per second. After 1947, subsequent to continued investigation, further recommendations were made. Although Watts[10] documented three instances of vertebral fracture at 16-19 G levels, he concluded that ejection seats designed to peak at 18-21 G should be tolerated without injury by the majority of the pilot population. In 1948 Ames[2] advised 20 G upper limit as did Glasser in 1950. In 1955, Mr. J. Martin stated that both the 60 ft/sec and 85 ft/sec Martin-Baker seats had onset rates of 200-250 G/sec and peak levels of 18-21 G. Barach, in 1956 empirically stated that tolerance limit is about 20 G for 100 msec or 25-28 G for 10 msec.

Injury data derived from operational experience with ejection systems designed according to these acceleration profile specifications have slowly accumulated. Between 1950 and the present, a number of reports have appeared in the aerospace literature documenting injuries received utilizing seats designed to operate within a specified envelope of acceleration environments. Three of the most enlightening of these are the reports of Laurel and Nachemson,[7] Fryer,[4] and Jones.[5] Laurel described 23 ejection profiles in which 15-20 G peaks were tolerated without a single case of fracture while there were 12 cases in 29 ejections involving 20-25 G profiles. Fryer's description of British experience with the Martin-Baker seat (18-21 G peak) between 1949 and 1960 documented 41 cases of x-ray proven fracture out of 200 ejections (20.5 per cent). In this series of 41, there was an average of two fractures per spinal column. Jones documented the 1958-1963 incidence of spinal fracture from the 18-21 G Martin-Baker seat utilized by the British, U.S., and Swedish Air Force during this period. Frequency of fracture was comparable for the British and U.S. being 20.5 and 21 per cent, respectively. The Swedish incidence, however, was listed as 46 per cent.

The acceleration profile produced by most present-day upward catapult seats exhibit 200-300 G/sec onset, 12-22 G peak, 70 $\pm$ 10 ft/sec terminal velocity, and 0.01-0.08 sec of peak G exposure. Under operational conditions utilizing such systems, there is an incidence of spinal column compression that averages about 6 per cent.

It must be emphasized that these reports reveal only the incidents of demonstrable decreased vertebral height. Although Fryer did record 28 incidences of "minor" injury which be defined as painful spinal symptoms or signs in the presence of "normal" X-rays, one has no way of knowing how many undetected endplate fractures (some painful and others asymptomatic) occurred during these ejections.

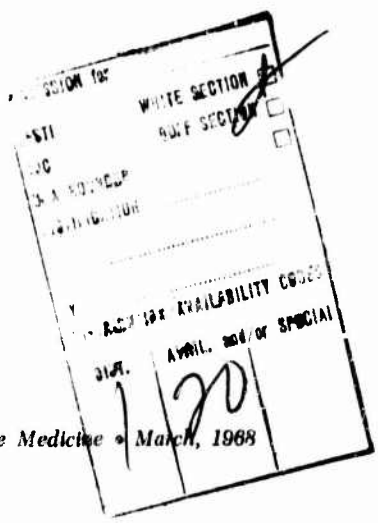
We should realize that we have little knowledge at present about the possible long-term sequelae of undetected endplate fracture. It is significant, as Jones[5] notes, that of the first seven Martin-Baker ejections with vertebral fractures, five were retired for radiculoneuritis, degeneration of intervertebral disc with localized arthritis and arthritis with muscle spasm. One cannot help but wonder how many undetected endplate fractures, with or without later compression, will have similar difficulties at some future date.

When human exposure to severe environmental stress is necessary, well-defined tolerance limits for the most susceptible organ system are necessary for design of optimal protection systems. Because of the multiple variables governing both the human response (congenital vertebral defect, restraint and posture during ejection) and the imposed acceleration environment (acceleration-time function, altitude and temperature, aircraft orientation); precise definition of these limits are not completely satisfactory for the emergency escape environment. The ideal solution would be to outline ejection tolerance curves that will be "safe" for the average ejectee population. The word "safe," however, requires qualification. One realizes from the evidence outlined above that any tolerance levels defined for maintaining "functional" spinal column in-

tegrity will, for the most part, be above breaking strength levels of the "weakest link" in the column, i.e., the vertebral endplate. At the present time, unfortunately, this injury response is not sufficiently appreciated by engineers and not always thought of (much less diagnosed) by medical personnel. Realizing that ejection is usually a life-saving situation without alternatives, and appreciating that endplate fracture doesn't usually result in acute functional disturbance, vertebral body tolerance curves are commonly accepted. Indeed, they are to be recommended if properly executed ejections utilizing optimally designed systems can not otherwise safely clear a pilot from a disabled aircraft. Unfortunately, escape system performance will always be somewhat hindered by biologic limitations. The challenge is to precisely define and utilize these end points to their maximum benefit.

SUMMARY

In this report we have attempted to review, elucidate, and extend the body of biodynamic information to the point where the engineer and physician will appreciate that a carefully designed ejection system can be utilized by the air crewman, under specified conditions, such that a predictable spinal injury rate will prevail. The unexpected compressive vertebral body fractures that may occur should for the most part result because of factors outside of objective control. Such factors are the congenitally abnormal vertebrae and the hyperdynamically responsive spine both of which preclude objective human control. Presently accepted ejection acceleration levels generally exceed the structural breaking levels of the vetebral endplates. The immediate implications of such injury are usually benign but long-term follow-up is mandatory if the occurrence of delayed effects is to be detected. Spinal trauma during ejection is not always a short-term affliction with minimal sequelae. *Any* spinal axis injury may possibly result in future physical disability, leading to mental anguish, physical pain, and financial loss. The engineers who design ejection systems and the medical personnel who care for the air crew share the responsibility for understanding the pathogenesis of spinal injury and for taking all possible precautions to minimize both the occurrence and the potential complications of this type of injury.



REFERENCES

1. Ames, W. H., Sweeney, H. M., and Savely, H. G.: Human Tolerance to Acceleration in Pilot Ejection. *J. Aviat. Med.*, 18:548, 1947.
2. Ames, W. H.: Human Tolerance to High Linear Accelerations of Short Duration. *Military Surgeon* 103:96-99, 1948.
3. Brown, T., Hansen, R. V., and Yorra, A. S.: Some Mechanical Tests on the Lumbosacral Spine with Particular Reference to the Intervertebral Discs, A Preliminary Report. *J. Bone Joint Surg.*, 39:1135-1164, 1957.
4. Fryer, D. I.: Operational Experience with British Ejection Seats. *F.P.R.C.*, 1166, 1961.
5. Jones, W. L., Madden, W. F., and Luedeman, G.: Ejection Seat Accelerations and Injuries. *Aerospace Med.*, 35:559, 1964.
6. Latham, F.: A Study in Body Ballistics. *Proc. Royal Soc. London*, 147:121, 1957.
7. Lauzell, L., and Nachemson, A.: Some Factors Influencing Spinal Injuries in Seat Ejected Pilots. *Aerospace Med.* 34:726, 1963.
8. Lewin, P.: *The Back and Its Disc Syndromes*, Lea and Febiger, 1955.
9. Martin, J.: Ejection from High Speed Aircraft. *J. Royal Aeronaut. Soc.* 60:659-668, 1956.
10. Payne, P. R.: Dynamics of Human Restraint Systems. Proceedings of a symposium on Impact Acceleration Stress, NAS-NRC, 97, 1962.
11. Perey, O.: Fracture of the Vertebral Endplate in the Lumbar Spine. Acta Orthopaedica Scandinavica Supplement No. XXV, 1957.
12. Roaf, R.: A Study of the Mechanics of Spinal Injuries. *J. Bone Jt. Surg.* 42:810, 1960.
13. Ruff, S.: Brief Accelerations: Less than One Second. Ch. VI-C, German Aviation Medicine, World War II, Vol. 1, Wash., D. C.
14. Stech, E. L.: The Variability of Human Response to Acceleration in the Spinal Direction. Frost Engineering Devel. Corp. Rept. No. 122-109, May 1963.
15. Stech, E. L., and Payne, P. R.: The Effect of Age on Vertebral Breaking Strength, Spinal Frequency and Tolerance to Acceleration in Human Beings. Report No. 122-101, Frost Engineering Development Corporation, Jan. 1963.
16. Stech, E. L., and Payne, P. R.: Dynamic Models of the Human Body. AMRL-TR-66-157 Aerospace Medical Research Laboratories, Wright-Patterson Air Force Base, Ohio. (In press).
17. von Gierke, H. E., and Coermann, R. R.: The Biodynamics of Human Response to Vibration and Impact. *Indust. Med. and Surg.* 32:30-32, 1963.
18. Watts, D. T., Mendelson, E. S., *et al.*: Tolerance to Vertical Acceleration Required for Seat Ejection. *J. Aviat. Med.* 18:554, 1947.
19. Woodburne, R. T.: Essentials of Human Anatomy, Oxford Univ. Press, 1957.
20. Yorra, A. J.: The Investigation of the Structural Behavior of the Intervertebral Disc. Master's Thesis, M.I.T., May 1956.