Biomechanics of the Spine

Whole-body vibration and repeated shock cause mechanical disturbances of the body. Epidemiological studies point towards low back pain and injury due to accelerated degeneration of the spinal unit as a hazard of chronic vibration exposure, whereas impact injuries involve fractures of the vertebrae. Mechanical systems fracture under a single severe load. They also suffer fatigue failure in response to repeated low level loads such as are present in vibration, or other coditions involving repetitive loading. Hence, there is an obvious correlation between the effects of mechanical shock and vibration on living systems and the failure modes of engineering materials. Therefore, an attempt to gain a quantitative understanding of the physical and mechanical processes underlying the adverse effects of severe impact loading, and of repetitive loading at lower levels of stress, must begin with an investigation of the mechanical properties of tissue.

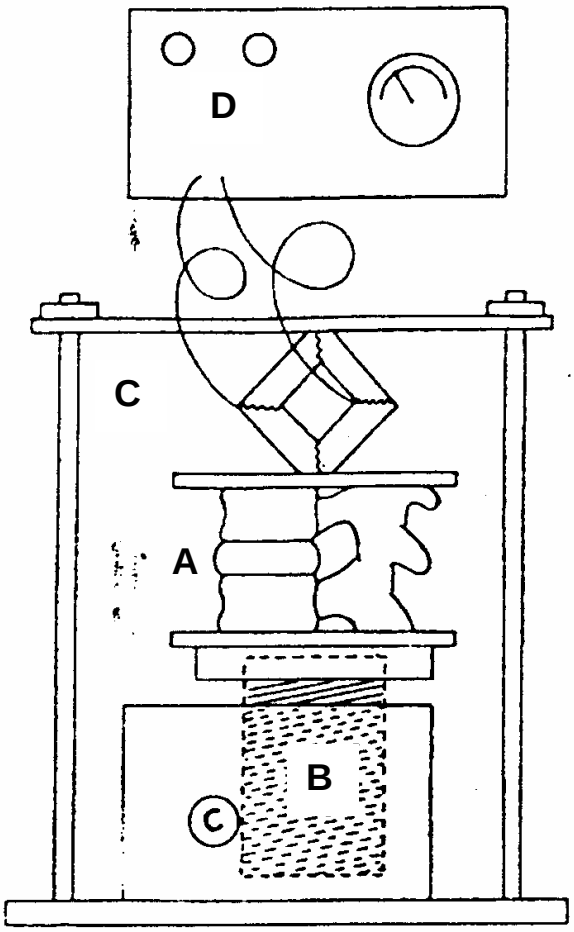
Material Properties of the Spine

The biomechanical properties of the spine have been studied extensively in vitro and to a lesser extent in vivo. Study of material properties in vitro has included the behaviour of the isolated spinal unit, which consists of an intervertebral disc and the superior and inferior vertebrae. Studies include the load deflection characteristics under conditions of axial compression, shear, torsion and bending (with both static and dynamic loading), visco-elastic behaviour, damping and dynamic stiffness.

In vitro studies provide limited information on the behaviour of the spinal column in the intact human. For example, the stiffness, damping and resonance frequency of the intact spine will be influenced by torso mass and muscle tension, and cannot be determined from in vitro studies. Basic data on the ultimate strength and fatigue failure characteristics of the spine can be obtained from in vitro measures. Knowledge of spinal loading and motion in vivo is required to relate material properties to the demands of normal activity, and working environments such as load carriage, vibration and impact.

Investigators have adopted two distinct approaches to study the biomechanics of the spine in vivo: a) direct measurement, and b) biomechanical analysis (based on external loading, tissue properties and anatomical structure).

Direct measures of spinal loading have been achieved by measurement of intra-discal pressure in vivo, while motion of the vertebra in response to whole-body vibration has been studied by insertion of steel pins to the spinous process. Visco-elastic compression during spinal loading has been studied by precise measures of stature. Invasive measures of spinal loading and motion are complex and have been limited to a few subjects. To obtain an understanding of the behaviour of the spine under conditions of dynamic loading in vivo requires the integration of information acquired through: mechanical properties measured in vitro, loading and motion measured in vivo, and biomechanical analysis.



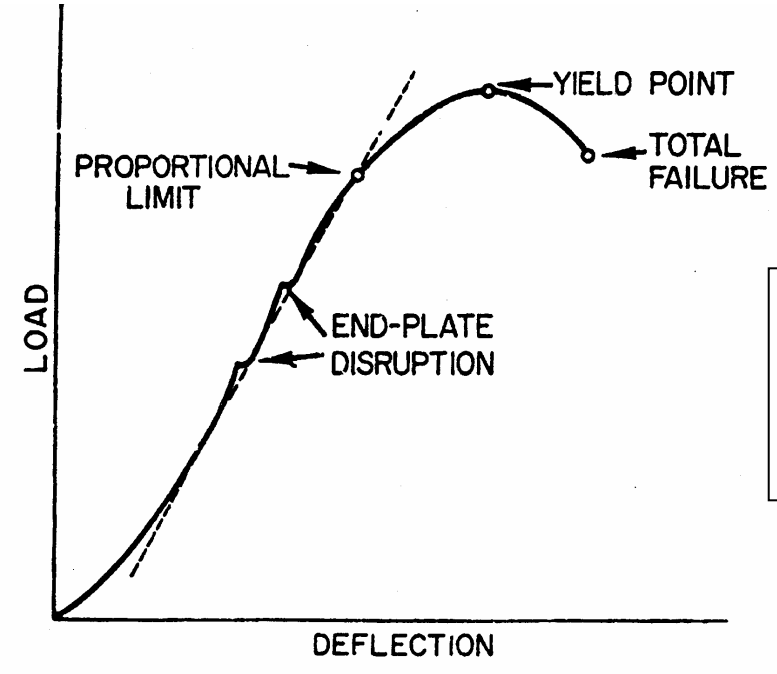
Compression apparatus in which the specimens were subjected to pressure (maximum 300 kp) recorded by a measuring brined at the same times as Röutgen plates were made.

A. Specimen
B. Mechanically driven screw
C. Strain gauge
D. Measuring Bridge

A centrally situated, postmortem fracture of the end-plate

Henzel, Mohr and von Gierke (1968) provided a comprehensive review of vertebral compression due to axial loading based on in vitro observations. The authors identify four distinct events in the load deformation data of the spinal unit (see Figure below):

- end plate fractures
- proportional limit
- yield point
- total failure



Schematic Load Deflection Curve Illustrating Alterations in Vertebral Body Structure During Compressive Stress

End plate fractures have been observed to occur within the linear portion of the load deformation curve. The 'proportional limit' defines the limit of linear elastic behaviour; beyond which load-deformation becomes non-linear and there is a reduced stiffness. It represents the point at which a material begins to fail, but is able to recover its preload form on release. The yield point defines the ultimate or maximum load beyond which irreversible deformation occurs. Total failure defines the point at which the structural integrity is lost and the material collapses.

The proportional limit and yield point of spinal units (vertebra-disc complex) and isolated vertebra in axial compression have been measured by several researchers. Ruff (1950) reported yield points of 5800 N to 10500 N for thoracic and lumbar vertebrae, (T8 to L5), with a progressive increase in strength with descending position in the vertebral column Ruff's Data are presented in tne table below. Ruff's values are approximately 50% greater than those reported by Yamada (1970) and Kazarian and Graves (1977), but lower than those of Gozulov et al. (1966) – See Tables below.

Calculation of Vertebral Strengths

Vertebrae	Percentage of Body Weight Carried	Mass in kg Carried by 72.7 kg Man	Breaking Strength (N)	Breaking Stress in g's[+]	Percentage of L4 Breaking Strength
T1	9	6.5	1,605	25.0	16.6
T2	12	8.7	2,140	25.0	22.1
T3	15	10.9	2,675	25.0	27.7
T4	18	13.1	3,211	25.0	33.2
T5	21*	15.2	3,746	25.0	38.7
T6	25*	18.1	4,459	25.0	46.1
T7	29*	21.0	5,173	25.0	53.5
T8	33*	23.9	5,864*	24.9	60.7
T9	37*	26.9	6,657*	25.2	68.9
T10	40*	29.1	7,277*	25.5	75.3
T11	44*	32.0	7,580*	24.2	78.4
T12	47*	34.2	7,835*	23.4	81.0
L1	50*	36.4	7,982*	22.4	82.6
L2	53*	38.5	8,584*	22.7	88.8
L3	56*	40.7	9,636*	24.1	99.6
L4	58*	42.2	9,667*	23.4	**100.0**
L5	60*	43.6	10,550*	24.6	109.1

*Single asterisk represents data collected experimentally by Ruff. Unmarked values are calculated or assumed

[+]This value represents the acceleration of the vertebral column at which fracture occurs. It is obtained by dividing the breaking strength (N) by the mass x g. for example at L3, 9636 N / (40.7 x 9.81) gives and acceleration of 24.1 g. Ruff was interested in the acceleration tolerance of the spine during pilot ejection.

The main point is to realise that when discussing mechanical stress (force divided by area), we are talking about the material characteristics rather than any specific specimen. So despite the fact that L5 is the largest vertebrae and has the highest breaking strength, the maximum ***stress*** it can sustain is the approximately the same as all the other vertebrae.

Differences in strength reported by the various researchers can result from differences in experimental technique, the quality of the material (whether there was any previous injury, or degeneration), the ages of the vertebrae, and the rate of application of the load (strain rate),. Most of these values are considerably higher than the values used in developing the NIOSH equation, and Mital's Guide to MMH. More recent data from Hutton and Adams, 1982 and Porter et al. 1989, suggests a strength of 10,000 N for the L4/5 spinal unit, with a SD of approximately 2000N, for males in the age range 18 - 46 yr. (see data below). Biomechanical analysis suggests that some individuals can support even higher compressive forces. Biomechanical data for weight lifters are presented in the accompanying figures and table, that suggest compressive loads of 18,000 to 36,000 N are possible.

Table 10. Comparison of Compressive Breaking Loads (lb)

Vertebral Body	Yamada[18]	Gozulov et al[4]	Messerer[10]
C1	920	1760	—
C2	920	1122	—
C3	920	889	330
C4	920	898	486
C5	920	997	374
C6	920	1239	374
C7	920	1021	374
T1	814	1045	440
T2	814	959	440
T3	814	1027	418
T4	814	1148	462
T5	948	1212	462
T6	948	1362	484
T7	948	1498	550
T8	948	1813	550
T9	1417	1848	704
T10	1417	1892	792
T11	1417	2017	880
T12	1417	2319	825
L1	1606	2396	880
L2	1606	2585	935
L3	1606	2640	770
L4	1606	2640	880
L5	1606	2834	935
Strain rate	?	.04 in./min	?

Note:
I lb = 4.45 N

Compressive strength measured during mechanical testing of lumbar spinal units (2 vertebrae and intervertebral disc).
Subjects 1 - 9 from Porter, Adams and Hutton (1989). Subjects 10 - 17 from Hutton and Adams (1982).

		Compressive Strength (N)				
Subject	Age	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
1	20		8500		11600	
2	18		10206		10330	
3	25		13954		10140	
4	20		8179		7631	
5	24		9460		9760	
6	19		7310		7710	
7	16		5120		8680	
8	23		7675		9206	
9	32		8404		11322	
10	32		6857		8088	
11	33		9024		10780	
12	46		10240		11237	
13	31				8710	
14	46	8553		9259		12981
15	31	11567		11895		10802
16	22			10780		12740
17	26					9987

Mean Compressive Strength (N) Estimated for L4/L5 Spinal Unit from Mechanical Testing of Lumbar Spinal Units (males 18-46 yr., n = 17). Data adapted from Porter, Hutton and Adams, 1989: and Hutton and Adams, 1982

	Age	Compressive Strength (N)
Mean	28	10,093
Std. Dev.	9	1,924

Table 1. The Age, Body Weight, Height, Bone Mineral Content (BMC) in L3, Maximum Lifted Weight, Calculated Load on L3 and Annually Lifted Weight in the Eight Analyzed Power Lifters

Power lifter	*Age (yrs)*	*Body weight (kg)*	*Height (cm)*	*BMC L3 (g/cm)*	*Maximum lifted weight (kg)*	*Load on L3 (kN)*	*Annually lifted weight (ton)*
1	30	93	177	7.76	335	27.5	2,150
2	40	84	165	6.25	290	27.0	450
3	33	59	160	6.11	212	18.8	300
4	24	60	156	6.70	230	28.9	1,000
5	25	69	165	6.44	305	22.9	1,200
6	30	90	172	8.42	335	36.4	4,000
7	24	88	183	7.98	-	-	5,000
8	23	90	172	6.86	-	-	2,000

* Did not participate in the world championship.

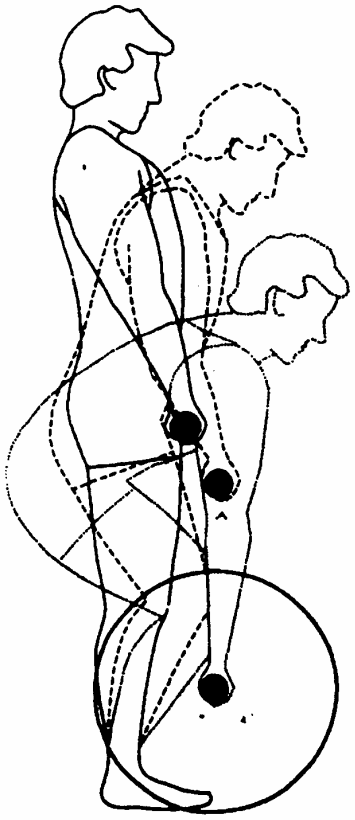


Fig 1. One of the power lifters performing a ground lift.

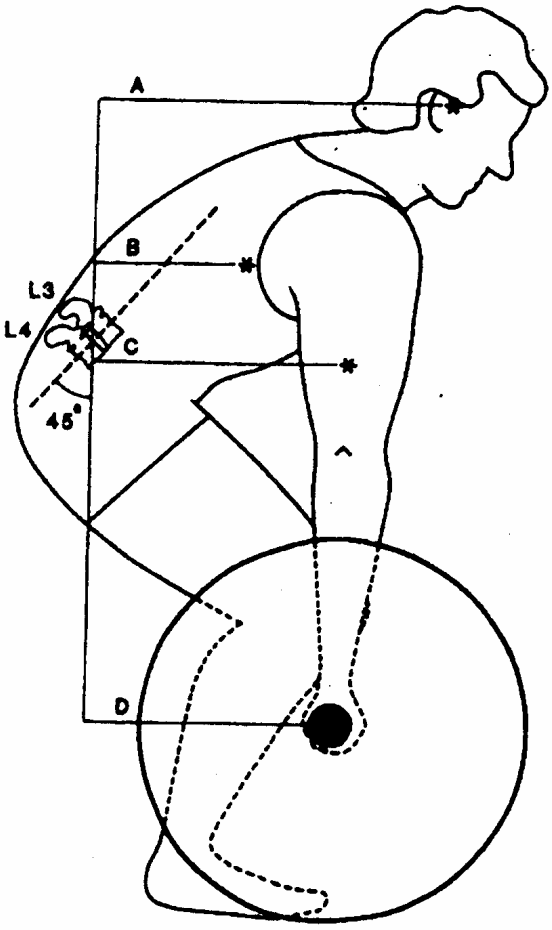


Fig 2. The lever arms used for calculation of the forces acting at the L3-L4 level. In this example the lumbar spine is flexed 45°. A, B, C, and D represent the lever arms from the center of L3-L4 to the mass centers of the head, trunk, arms, and the lifted weight, respectively.

The mechanism of compressive deformation and failure of the vertebra-disc complex has been reported by several investigators. Axial compression of the spinal unit results in a loss of height measured between the vertebrae. As the disc material itself is essentially incompressible, height decrease must result in a radial bulge of the disc. Measurements indicate an increase of disc compressive load from 1000 to 2000 N causes a disc radial expansion of 0.2 mm. Radial disc bulge is accompanied by a corresponding axial disc bulge (an inward deformation of the vertebral end plates). Axial bulge has been reported to be of the order of 0.2 to 0.5 mm under physiologically safe loads, increasing to 1.0 mm at yield point. Thus, as the rims of the vertebra approach, the end-plates deform in the opposite directions such that the height at the centre of the disc remains almost unchanged.

At yield point, a deformation of 1.0 mm of the end plate will produce a mean compressive strain of approximately 7% in trabecular bone, which approximates the yield strain for that material. Brinckmann (1988) reported that axial overload, and repetitive loads below the yield point damage the end plates and adjacent trabecular structure, whereas the annulus remains intact unless the physiological limits of flexion or bending are exceeded. This suggests that disc herniation is caused by fatigue failure of the disc structure, (due to repetitive loading within the elastic limit of the material) rather than by a single mechanical overload.

The range of ultimate stress for trabecular and cortical bone is shown below.

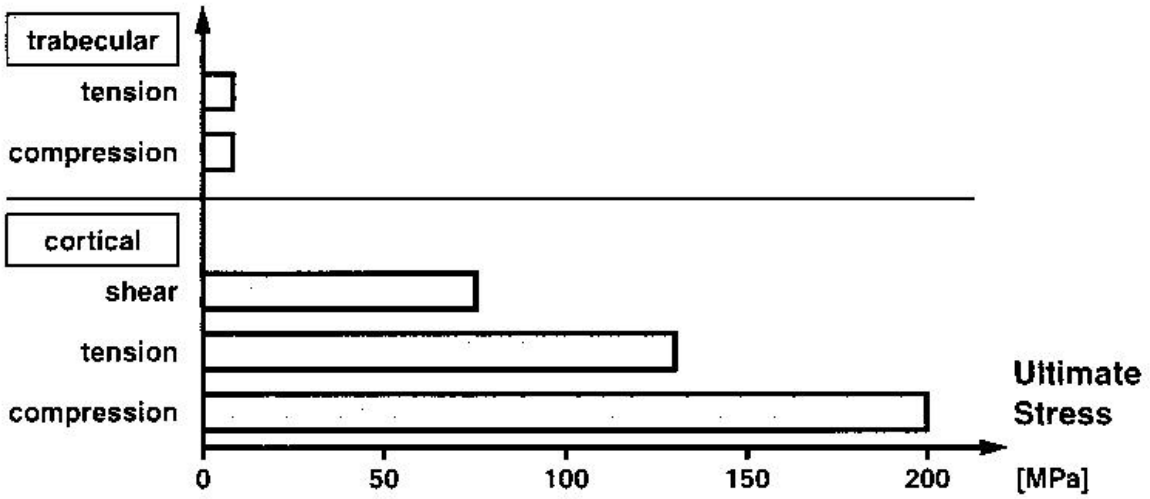


Figure 2.3.2 **Order of magnitude of ultimate stress for cortical and trabecular bone (based on data from Yamada, 1970; Steindler, 1977; Reilly and Burstein, 1975; Martin and Burr, 1989, with permission of Williams & Wilkins, Baltimore, Maryland).**

A detailed investigation of the mechanical properties of the spine in vitro was reported by Markolf (1970). In vitro specimens taken from the thoracic and lumbar spine were and lateral bending. Stiffness in axial compression was measured to be 1200 to 3300 $N.mm^{-1}$ at moderate loads of 200 to 600 N. Stiffness in axial tension was half of this value, while stiffness in shear was 10% to 15% of that in axial compression (100 - 500 $N.mm^{-1}$). The initial rotary stiffness for lateral bending, flexion and extension were all of similar magnitudes (0.7 to 4.7 Nm/degree) and did not vary significantly between lumbar and thoracic regions. Stiffness in axial torsion showed a discontinuity between the tenth and twelfth thoracic vertebrae, with a much greater stiffness occurring in the lower thoracic and lumbar regions. Stiffness in axial torsion and extension were found to be dependent on the posterior facets and ligaments.

Crocker and Higgins (1967) measured the dynamic stiffness of L1 - L2 spinal units. The vertebrae were compressed at 0.1 and 0.7 $mm.s^{-1}$ and finally at 4 $mm.s^{-1}$ to failure. Stiffness was found to increase at higher strain rates (viscous properties). At compression rates of 0.7 and 4.0 $mm.s^{-1}$ the spinal units displayed linear characteristics with a stiffness of 6000 and 12000 $N.mm^{-1}$ respectively. At a low compression rate and moderate loads (< 1000 N) stiffness was comparable with the measures of Markolf.

Kazarian and Graves (1977) investigated the mechanical properties of the isolated vertebral centrum subject to axial compression. Their results are shown in the Figures on the next page. P1, 2, 3 and 4 represents the position in the vertebral column, and R1, 2, and 3 represents the strain rate. Results show that the ultimate load (yield point) and stiffness varied with position in the spinal column, and the strain rate. At low strain rates (R_3 = 0.09 $mm.s^{-1}$ see top figure) the ultimate load increased from 2700 N at T1 - T3 to 5600 N at T10 - T12. Both ultimate load and stiffness increased linearly with strain rate. In the T10 - T12 region ultimate load increased from 5600 N to 8900 N at strain rates of 900 $mm.s^{-1}$ (R_1 in top figure). Note that in these figures, the strain rate R_1 is greatest and the strain rate R_3 is smallest. So, the axis of the graph really represents the inverse of strain rate: i.e. $(\text{rate of strain})^{-1}$.

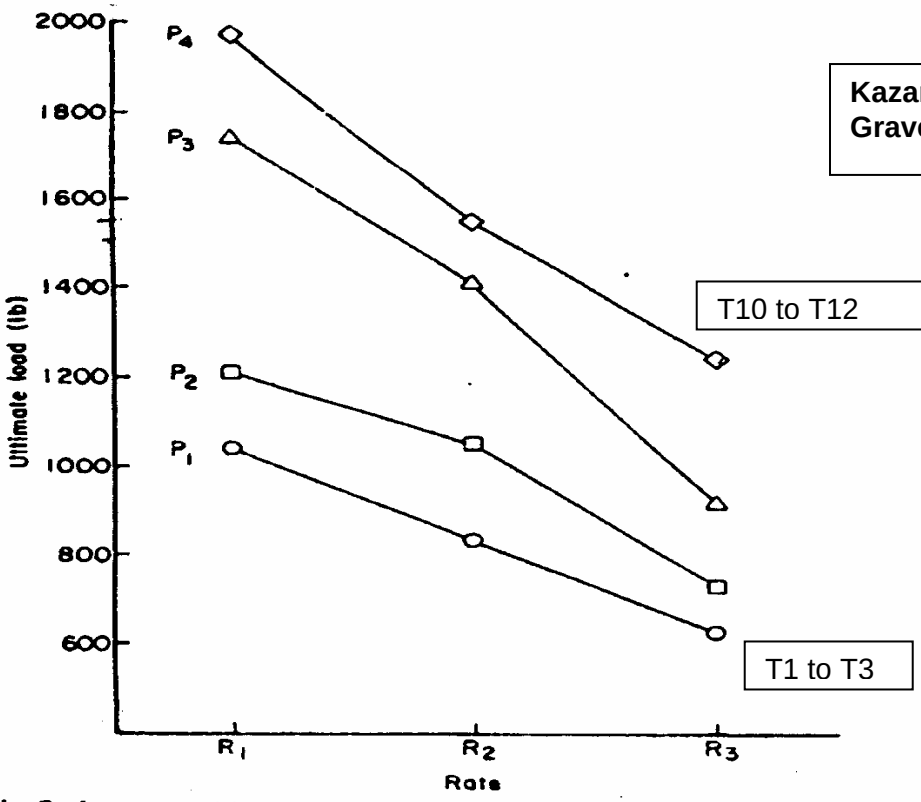


Fig 5. Average ultimate load versus displacement rate for each position (R_1 = 2100 inches/min, R_2 = 21 inches/min, R_3 = 0.21 inches/min).

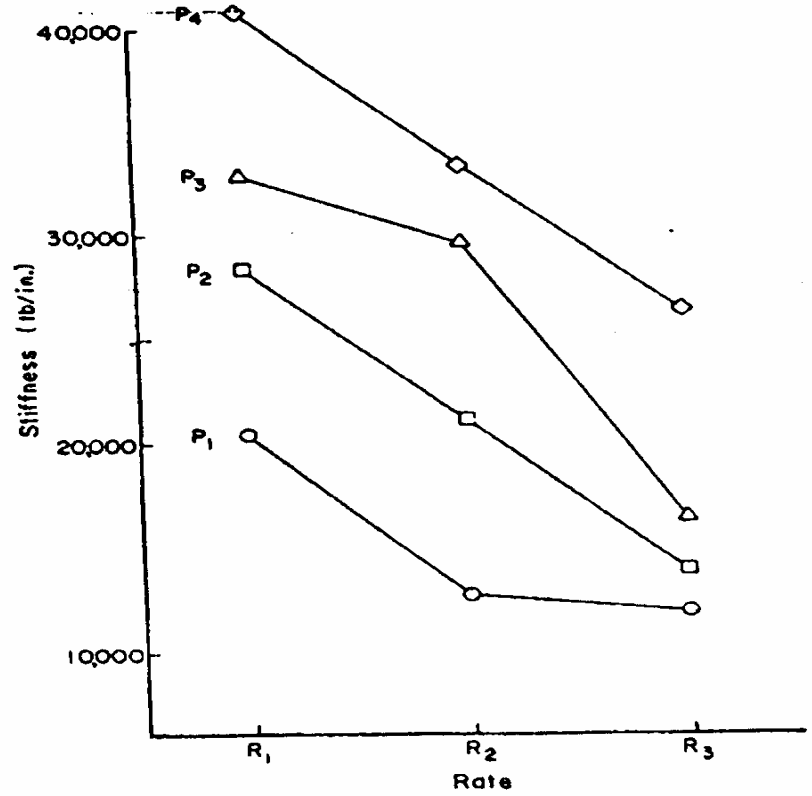


Fig 9. Average stiffness versus displacement rate for each position (R_1 = 2100 inches/min, R_2 = 21 inches/min, R_3 = 0.21 inches/min).

Loss of Stature: Spinal Creep

It has been reported that people lose approximately 1% of stature during the course of a day (Reilly, Tyrrell, and Troup, 1984). This loss of stature is then regained during the hours of sleep. Loss of stature occurs primarily through change in height of the intervertebral discs, and is exponential in form, the most rapid change occurring during the first hour after rising (Corlett et al. 1987).

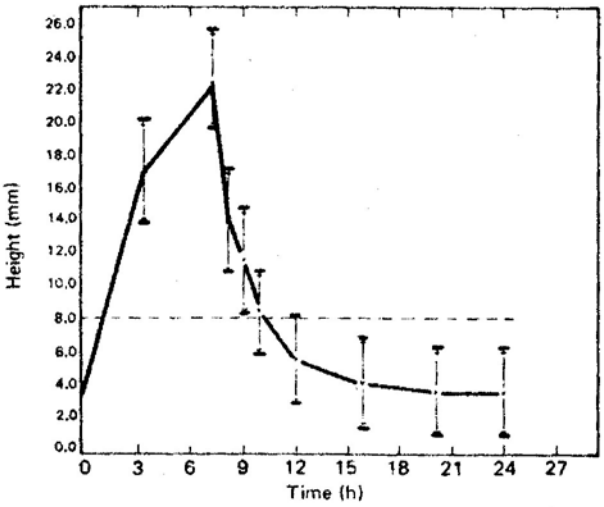


Fig. 1 Height changes of eight healthy male adults over a 24 h period. The midnight baseline is set at 3·5 mm. The unbroken line indicates the mean values and 95% confidence limits. The dashed line represents the cosinor curve fitted to the data. (Reilly *et al*, 1984, Fig. 2.)

Various investigators have shown that the decay in stature can be accelerated by such factors as static spinal loading, dynamic lifting, running, and different types of seating. Lifting a 50 kg load repeatedly for 20 minutes would cause a shrinkage equivalent to the entire diurnal loss of stature (Tyrrell, Reilly, and Troup, 1985; Corlett et al. 1987). By comparison, sitting on a stool caused a height loss of 3 $mm.hr^{-1}$, and sitting in an office or easy chair with back support caused no height loss over 90 minutes (Eklund and Corlett, 1984). It is probable that this difference is due to variations in posture and muscle activity, which will produce changes in spinal load. Evidence of a strong relationship between spinal load and height loss has led researchers to investigate whether stature loss can be used as an index of the effects of whole-body vibration. Results are inconclusive with some researchers reporting a height loss in response to vibration, and others reporting a height gain.

Haslegrave, Shearing and Corlett (1989) measured a height gain of 0.8 to 6.1 mm in response to one hour of vibration at 4 Hz and 1 $m.s^{-2}$ magnitude. Bonney and Corlett (1988) also report a gain of 1.9 mm in a one hour exposure at 4 Hz and 1 $m.s^{-2}$, but a loss of 1.0 mm with no vibration, and no change at 6 and 8 Hz. Sandover et al. (1991) have reported a gain in height of 1.1 and 1.9 mm following 30 minutes of sitting with and

without vibration. The subjects returned to their pre-exposure height within 10 minutes of the end of exposure. Results of Sandover et al. (1991) do not support the concept of an inflammatory response proposed by Sullivan and McGill (1990). In contrast to the above, Klingsterna and Pope (1987) and Sullivan and McGill (1990) have reported a loss of stature in response to vibration exposure.

The conflicting nature of results may arise from the difficulty in obtaining accurate measures. The results are sensitive to the exact conditions of posture and timing under which measures are obtained immediately prior to and after vibration exposure. For example, when measures are taken in an erect posture after a seated exposure the effect of heel pad compression, reported to average 4.5 mm over 90 seconds, may confound results (Haslegrave, Shearing, and Corlett, 1989). Similarly, the large loss of stature reported by Sullivan and McGill (1990) who measured sitting height in response to vibration, may derive from visco-elastic compression of soft tissues of the buttocks, rather than the intervertebral discs. Hence, until more consistent data become available, and an explanation of stature change in response to vibration is elucidated, it is unlikely that the measure of stature will prove a useful means of estimating the effects of vibration and impact on the spine.

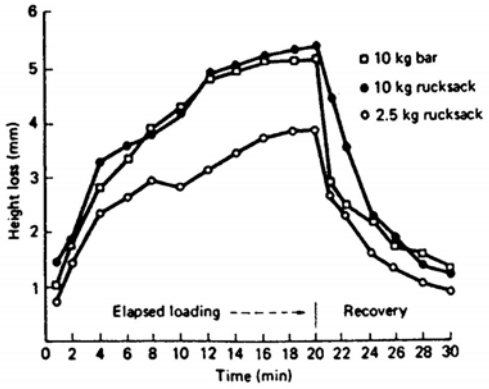


Fig. 3 Results of static load with 10 kg barbell and rucksacks of 2·5 and 10 kg while standing for 20 min followed by standing recovery. Mean values from eight male subjects at 2 min intervals (Tyrrell *et al*, 1985, Fig. 2).

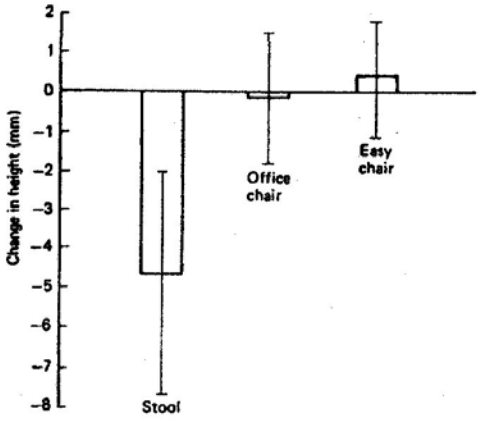


Fig. 8 Height losses after 1½ h of sitting in each of three chairs. Means and standard deviations from three subjects (Eklund and Corlett, 1984, Fig. 6).

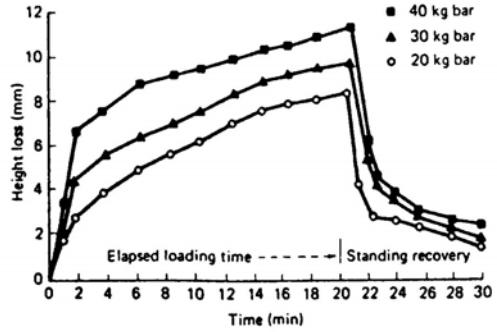


Fig. 4 Static shoulder loading with barbells of 20, 30 and 40 kg in standing position for 20 min followed by standing recovery. Mean values at 2 min intervals from eight males (Tyrrell *et al*, 1985, Fig. 3).

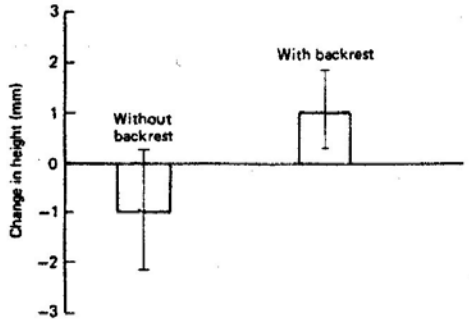


Fig. 9 Height losses after a 30 min pushing task while seated with and without a backrest. Means and standard deviations from four subjects (Eklund and Corlett, 1984, Fig. 7).

Repeated Loading and Fatigue Failure of the Spine

Compression fracture is the common failure mode of the vertebra-disc complex in severe axial loading. This mechanism does not apply to repetitive loading within the linear portion of the stress-strain curve. Low back pain and back disorders associated with whole-body vibration and repeated shocks point to a chronic degeneration of tissues, rather than acute failure.

Two mechanisms have been proposed to relate vibration exposure to degenerative changes of the spine:

- impairment of nutrition; and
- mechanical fatigue due to repetitive loading.

Hansson and Holm (1991) speculate that disc nutrition may be disrupted by vibration. They identify two mechanisms for transfer of nutrients: molecular diffusion through the tissue matrix, and fluid transfer due to the pumping action of loading and unloading the disc. These mechanisms act across both the annulus fibrosis and the cartilaginous end plates of the disc. Although the cause of disc degeneration is multifactorial, in addition to ageing, mechanical factors may initiate degenerative processes. A possible mechanism is the disruption of blood flow in vessels surrounding the annulus fibrosis and under the end plate cartilage that will affect solute transport by both diffusion and fluid transfer.

Several researchers have proposed the role of mechanical fatigue as a factor in chronic degeneration of the spine. Troup (1975) points to evidence of fatigue failure of the neural arch in athletes subjected to repetitive loading. Brinckmann (1985) observes that detached pieces of annular material and sometimes fragments of cartilaginous end plate cause clinical symptoms of disc herniation.

Sandover (1983) proposed two hypotheses to relate fatigue-induced failure of vertebral tissue to disc degeneration. In the first, dynamic compressive loading of the joint leads to fatigue induced micro fractures of the end plate or subchondral trabeculae. Callous formed during the repair process leads to reduced nutrient diffusion. In the second hypothesis, dynamic shear, bending or rotational loading of the joint leads to fatigue induced failure within the annulus, either as tensile failure of the collagen fibres, or as failure of cohesion between fibres or lamellae.

The fatigue characteristics of bone have been studied several researchers. Lafferty (1978) derived relationships between fatigue life of bone (number of cycles to failure), and peak stress. Sandover (1983; 1985) proposed a model of fatigue-induced failure of the intervertebral joint using the data of Lafferty and others:

$$N = (S_U/S_p)^X \qquad \log N = \log(S_U/S_p)^X$$

where: N = number of cycles to failure, S_U = static failure stress,
S_P = applied repetitive stress, and x = constant.

The value of exponent x varies between biological tissues and test method from x = 5 for cortical bone (Carter et al. 1981) to 20 for cartilage (Weightman, 1976).

The risk of fatigue-induced failure can be estimated by application of the Palmgren-Miner hypothesis. This hypothesis states that the degree of fatigue damage is given by the

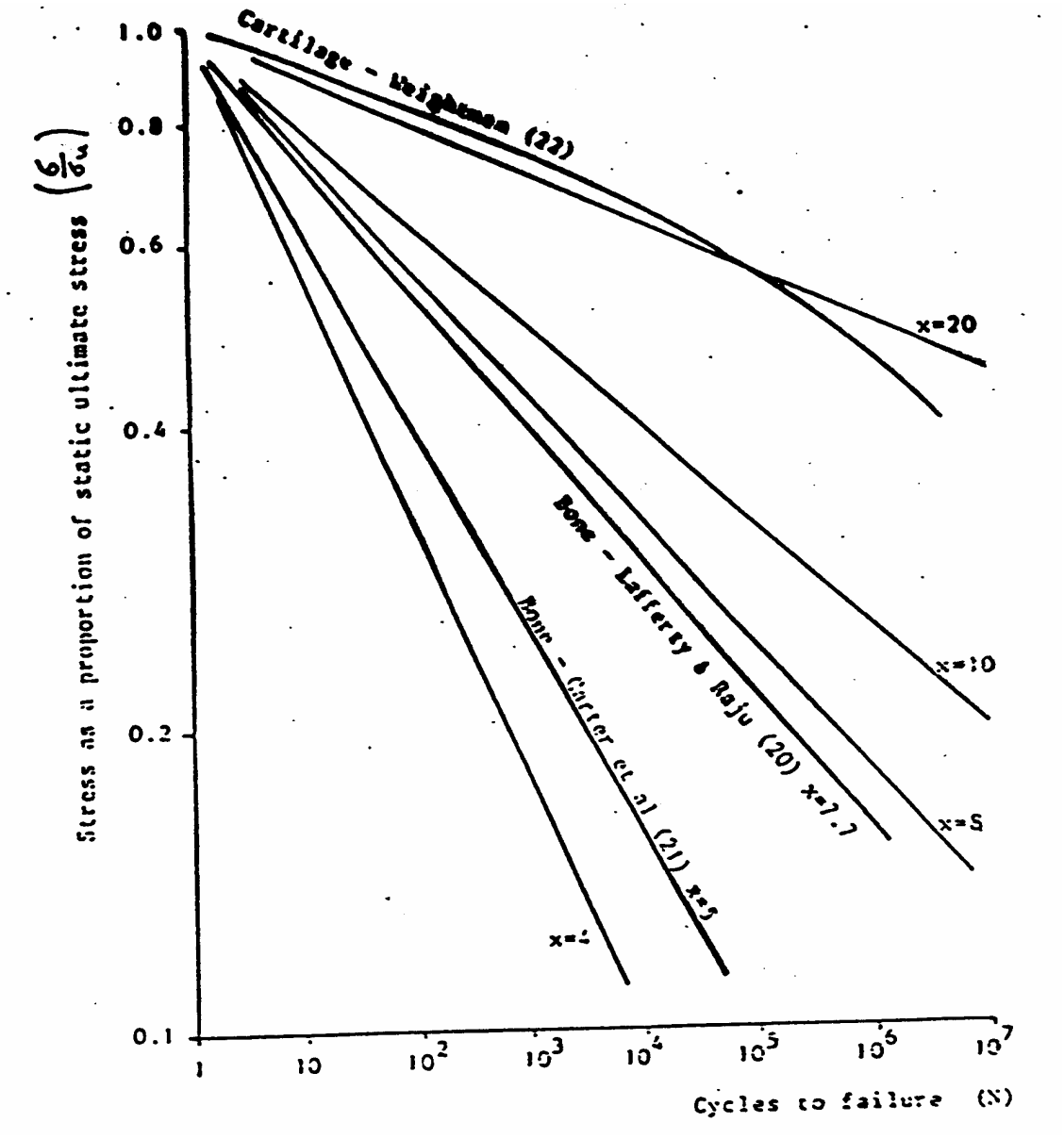
summation of n_i/N_i: where n_i is the number of cycles at a particular stress level, and S_i and N_i are the number of cycles to failure at that stress.
The effect of a particular vibration environment can be estimated in terms of a "dose" value as:

$$D = n_i/N_i \qquad \text{or, } D = n_i (S_i/S_u)^X$$

where D = fatigue dosage index, and i = magnitude of stress

In this system, a dosage value of D = 1.0 represents the accumulated exposure at which fatigue failure is expected. The theory can be extended to include stresses of different magnitudes i, where i = 1 to j, and

$$D = \sum [\, n_i (S_i/ S_u)^X \,]$$

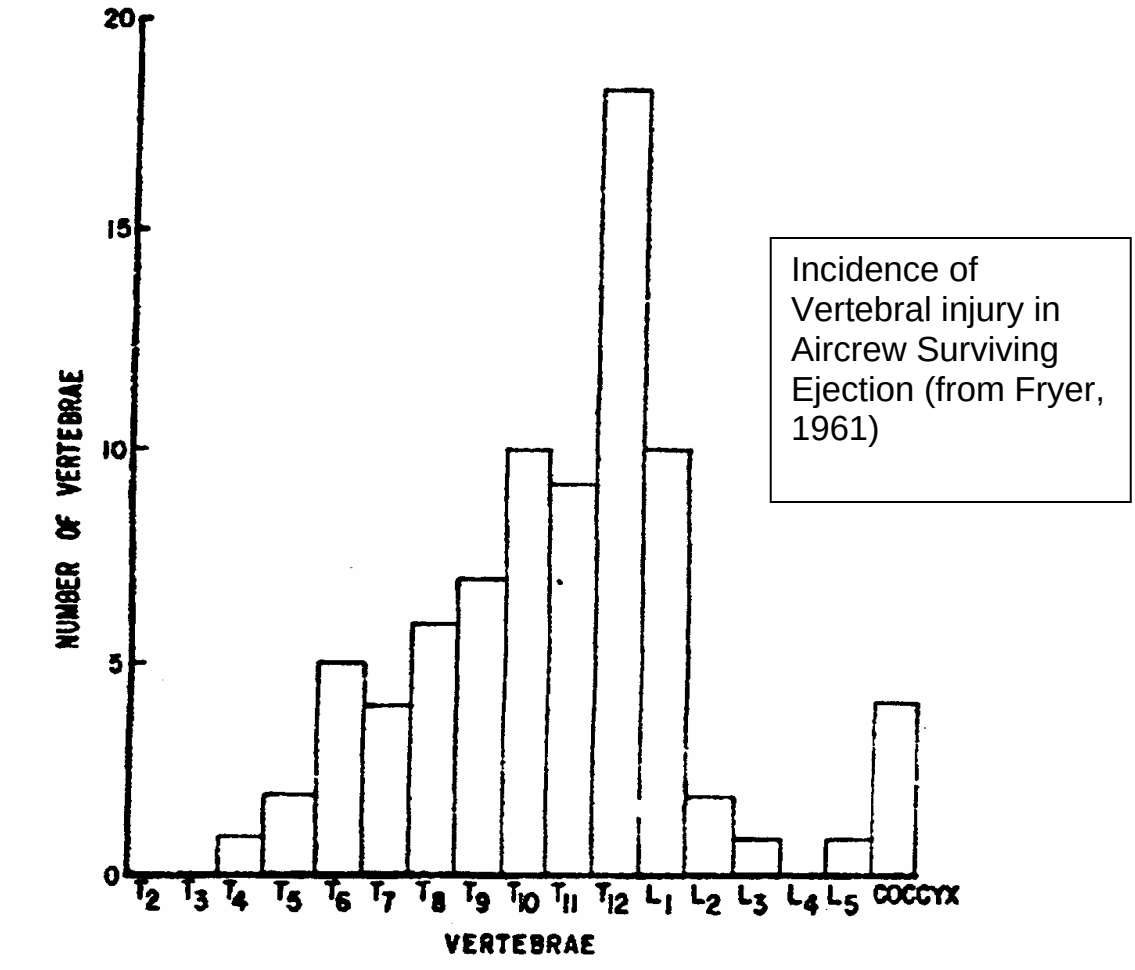


Fatigue life of animate tissues.
Straight lines represent the functions $N = (\sigma_u/\sigma)^X$

Impact Acceleration and Spinal Injury

Investigations of impact accelerations on the human body include horizontal seated impact occurring in vehicle collision, vertical impacts occurring in the seated posture (due to pilot ejection or lifeboat free-falls) and in the standing posture due to blast in ships. Studies of impact acceleration include both cadaver and epidemiological studies.

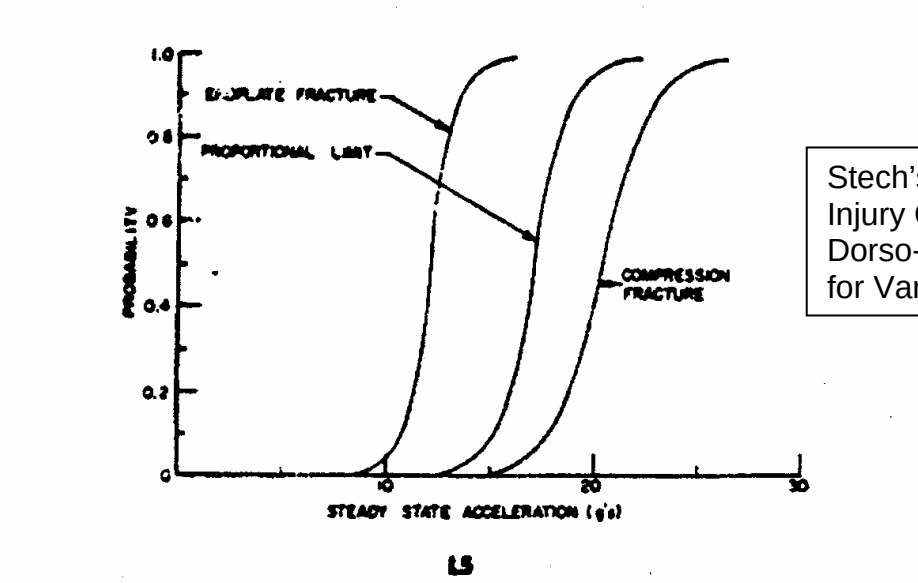
Vertebral fracture incident rates of 21% were reported in early pilot ejections from US naval aircraft. The ejection seat was specified to produce accelerations of 18 to 22 g (180 to 220 $m.s^{-2}$). Data obtained from British and Swedish aircraft using the same system showed a fracture rate of 19% in 220 ejections and 43% in 7 ejections respectively. Laurell and Nachemson (1963) reported no vertebral fractures in 23 cases of ejection with accelerations of 15 to 20 g, and a 41% fracture rate from ejections at 20 to 25 g. Although fractures were found at all levels of the thoracic and lumbar spine, the US and British data showed the highest rates occurred in the lower thoracic region from T8 to L1. Over 50% of pilots injured sustained more than one fracture.



Incidence of Vertebral injury in Aircrew Surviving Ejection (from Fryer, 1961)

Tolerance of the spine to G_z impact acceleration has been estimated by Stech (1963) from a combination of in vitro data of yield strengths of individual vertebrae, biomechanical analyses of the spine, and probability theory. Stech constructed injury probability curves as a function of z-axis acceleration level. Data included probability of

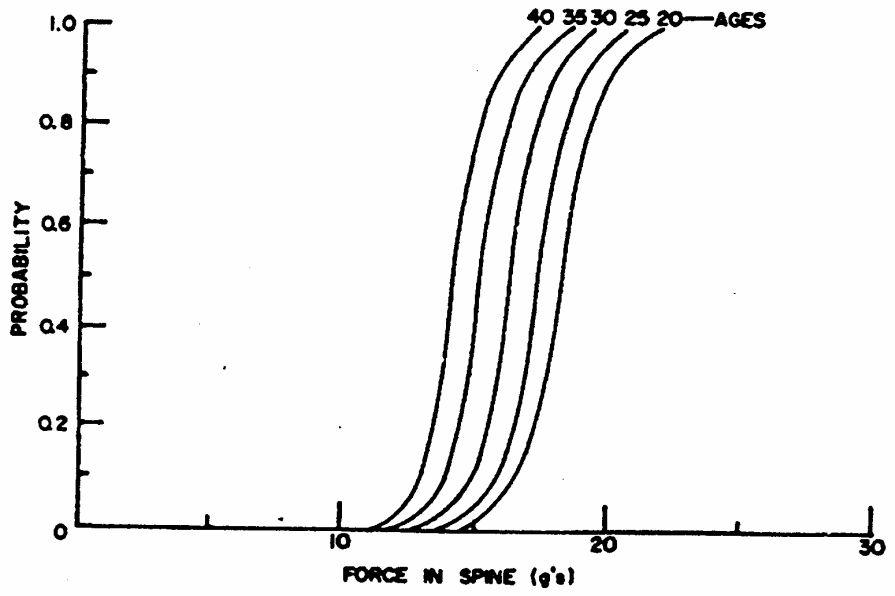
end plate fracture, proportional limit deformation, and compression fracture at different vertebral levels.



Stech's probability of Injury Curve for the Dorso-Lumbar Column for Various Age Groups

Figure 9. Probability of injury curves for L1-L5 vertebrae during steady state acceleration.

Stech and Payne (1963) also calculated the combined injury probability function of compressive fracture of a vertebra for the complete spinal column. Results indicated a 0.5 probability of fracture at an acceleration of 18 g at age 20 years, reducing to 13 g at age 40 yr. By comparison the 0.5 probability of end plate fracture in the lumbar region was calculated to occur at approximately 10 to 12 g, which represented about half the acceleration level for vertebral fracture.



The role of the articular facets in impact acceleration was studied using instrumented human cadavers. The facet joints are initially subjected to compression followed by a tensile phase. The latter results from a forward flexion of the head and torso, which applies a tension to the posterior structures of the spinal column. The forward flexion of the torso also causes increased loading of the anterior aspects of the vertebra, and anterior wedge fractures. This explains why injury predictions shown in the figures above occur at lower values of "g" than suggested in the data of Ruff (see Table). Ruff's analysis, based on cadaver specimens, only considers compression of the vertebra in the longitudinal axis. In real life, response to vertical impact loading in the seated position results in both axial compression of the vertebral column, plus a bending moment which causes added compression of the anterior aspect of the vertebrae, particularly in the lower thoracic region. This can be demonstrated (or simulated) in dynamic biomechanical models. This type of information can be used in the design of seating, posture, and restraint harnesses for workers at risk of impact loading.

Due to the dynamic characteristics of the human body, the peak force transmitted to the body tissues is dependent on:

- impact acceleration magnitude,
- pulse duration, and
- natural frequency of the body (f_n).

For a constant acceleration, or for accelerations with slow onset rates and durations greater than f_n, the initial response is equal and opposite to the applied force (i.e. equilibrium is established).

If the pulse length is much shorter than the natural period of the body, the elastic tissues will still be compressing when the acceleration ends. Under these circumstances, (in a simple mass-spring system), the force transmitted is then dependent on the imposed velocity change, or Δv (Glaister, 1978).

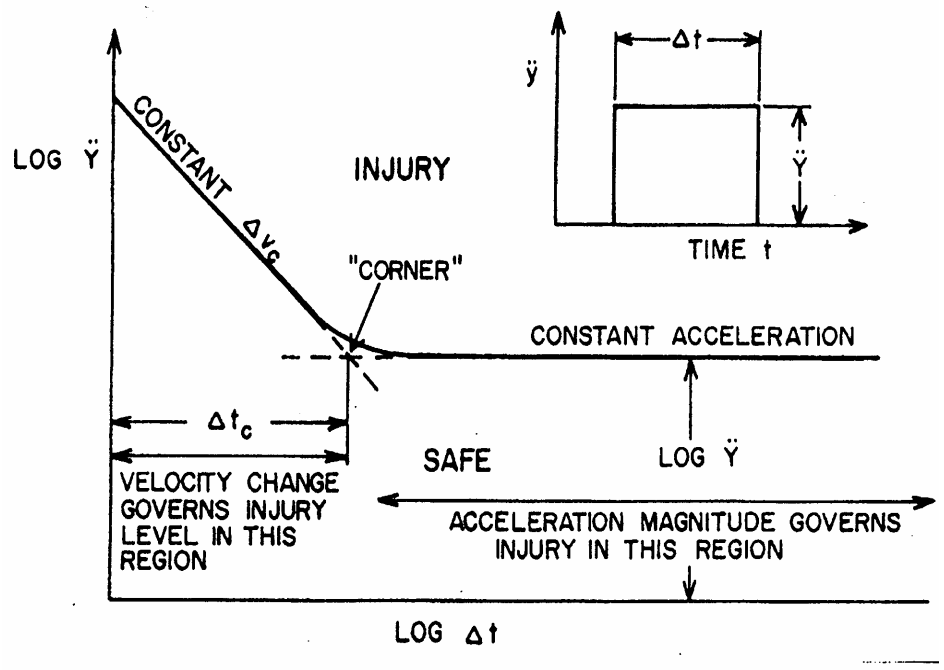


Figure 2. A tolerance curve, after Kornhauser and Payne.

As the velocity change due to an acceleration pulse can be represented as the integral of acceleration with respect to time, then the allowable pulse amplitude (i.e. acceleration) will increase linearly with the inverse of pulse length (i.e. frequency). The concept of Δv (acceleration x pulse length) is used in the analysis of the severity of vehicle collisions and their likely effect on humans.

Glaister suggested that for a seated posture, where the natural frequency of the body approximates 5 Hz, the critical pulse length for impacts is about 0.2 seconds (this value corresponds closely with the pulse length of early catapult ejection seats). Glaister extended his theoretical approach to produce curves of equal acceleration tolerance as a function of pulse duration, for the body in seated, standing and supine postures. These data are shown in the figure below. These curves should be treated with caution as they are only intended as an indication of the most probable level of tolerance.

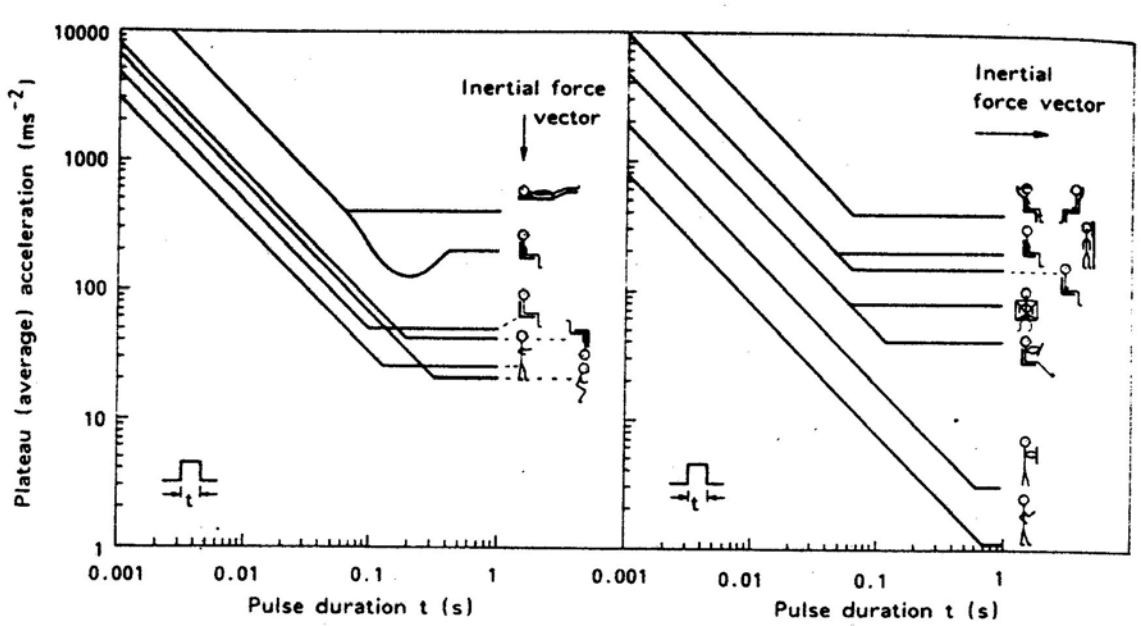


Fig. 5.9 Shock tolerance of the human body in various positions and postures and with various restraints. Adapted from Glaister (1978). The data generally apply to 'maximal voluntary exposures of keen experimental subjects'; data for horizontal motion of standing persons apply to general public in transportation systems.

Although some authors have concluded that the human response can be approximated by a linear system others suggests that the dominant natural frequency of the body increases from 5 Hz when exposed to whole-body vibrations to approximately 12 Hz when subject to severe impact. The shift of frequency is attributed to a pronounced non-linearity of human structure that results in increased stiffness and natural frequency at greater acceleration magnitude (Payne, 1991).

Conclusions

The spine is a complex structure consisting of a series of rigid elements (vertebrae) connected by flexible visco-elastic units (intervertebral discs). Compressive, bending and shear loading can be transmitted by a combination of forces in the intervertebral discs, apophyseal facet joints, ligamentous structures and active muscle contraction. The intervertebral disc is subject to stress whether a person is standing, walking, or seated. An important difference between a purely mechanical system and a living system is that the mechanical system does not change under constant stress provided the strain does not exceed the elastic limit. In a biological system the elastic properties of tissue are a time dependent function of the applied stress. Thus, loss of fluid takes place from the intervertebral disc space in response to static loading, (referred to as creep). This will affect the stability of the spinal unit and cause a redistribution of stresses in the surrounding tissue.

Investigations of spinal units in-vitro have shown non-linear load-deflection characteristics. Ultimate strength and stiffness increases with the rate of compression. Fracture of the end plate occurs within the elastic limit of the material. In general, failure occurs due to compressive fractures of the vertebra, while the intervertebral disc remains intact. In single impact studies, vertebral damage occurs most frequently in the lower thoracic and upper lumbar region in the form of anterior wedge fractures, at impact accelerations of 18 to 25 g. Some of this data is from pilot ejection data where the pilot would experience a very high acceleration. This is looked at more closely when we discussed shock and vibration later in the course.

Both nutritional and fatigue mechanisms have been postulated to explain chronic degenerative failure in response to whole body vibration and repeated mechanical shocks. Whether some of these models are applicable to repeated lifting scenarios remains to be investigated. Obviously, in industry we are more interested in tissue damage due to the effects of repeated lifting rather than form vibration or repeated shock. However, the mechanisms of injury may be similar.

Calculation of fatigue failure properties is based on in-vitro data. They do not include consideration of the ability of tissue to recover or repair through on-going nutritional mechanisms, or the process of ageing. Hence, these calculations may underestimate or overestimate the real fatigue life of tissues in-vivo due to the absence of regenerative or degenerative factors in the model.