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FOOTBALL INJURIES

Papers Presented at a Workshop

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Committee on the Skeletal System
Division of Medical Sciences
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FOREWORD

The committees of the Division of Medical Sciences of the National Research Council have traditionally served in the role of providing a forum for discussions by diverse disciplinary groups that share interests in particular problems. This volume, which consists of papers prepared for a workshop on football injuries, is the report of such a forum. The authors represented a cross-section of disciplines concerned with various aspects of the training, health, and well-being, including protection from injury, of football players, as well as other athletes.

The workshop was planned and conducted by the Subcommittee on Athletic Injuries of the Committee on the Skeletal System. The planning of the workshop was supported by funds from the Surgeons General of the Department of the Army, Department of the Air Force, and Department of the Navy and from the National Institutes of Health, Public Health Service. The holding of the workshop itself and the publication of this volume were made possible by a grant from the Office of the Commissioner, National Football League.

We extend our appreciation to the participants, to those who planned and carried out the workshop, to those who prepared the publication, and to the financial sponsors.

Charles L. Dunham, M.D.
Chairman
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National Research Council

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EPIDEMIOLOGY OF SPORTS INJURIES

JESS F. KRAUS

Epidemiology is the field of science concerned with study of the factors and conditions that determine the occurrence and distribution of a disease or injury in a population.[7]

The application of the epidemiologic method to the study of accidental injuries and deaths was first suggested by Gordon only 20 years ago. Gordon[2] demonstrated that injuries, like disease processes, conform to particular biologic laws. He showed that the biologic principles governing disease as a community problem hold equally well for injuries. He also suggested a pattern of epidemiologic analysis similar to the procedures used in the study of infectious and chronic disease for deriving a better understanding of accidental, injury-producing mechanisms.

It is the purpose of this paper to discuss the applicability of some of the more general epidemiologic processes to the investigation of sports injuries. Most examples given will be directed to the study of football injuries.

It can be shown that the distribution of injuries, like that of disease, has definable variations or patterns. The rate of occurrence is different for various groups and circumstances. Study of conditions accounting for variation among groups contributes much to the understanding of etiology. The approach most often found successful is the identification of variables associated with a susceptible host (a football player), the agent (the game), and the environment. Study of the interrelationships of host, agent, and environment often leads to identification of causal mechanisms.

The initial procedure in the epidemiologic approach involves study of the distribution of injuries or deaths in terms of who was injured when, where, and how. Often called "descriptive epidemiology," this procedure usually involves analysis of extant data for identifying high- and low-risk groups according to some common factor, such as age, date, or type of game. This step is the springboard in developing generalized hypotheses of causation and furthermore directs the researcher's attention to the more provocative aspects of the problem being studied.

MORTALITY AND MORBIDITY DATA

Injuries, like many other pathologic entities, have a spectrum of severity. The more severe injuries are generally known and reported, whereas minor injuries go unreported. Most available information on football injuries, for example, concerns the more severe types reported. That fact alone should serve as a warning against widespread inferences stemming from only mortality or severe-morbidity data.

Probably the most valuable expression of risk of injury is the frequency, or incidence. This rate is established by comparing the number of persons with injury or death to the number exposed to a chance of being so affected. An incidence is, therefore, a probability statement of risk.

The usual and most often attempted approach in the descriptive phase of the epidemiologic process is a study of the frequency distribution of deaths due to the event in question. If the ratio of injuries to deaths is extremely large (as it is with football), great care must be exercised in making broad assumptions about risk. The death rate in 1967, as reported by the American Football Coaches Association,[1] was 1.77 per 100,000 participants. Further analysis shows that the death rate for college football players is 2½ times higher than for high-school players. The cause of this excess risk to college football players is not understood. Some hold that "poor conditioning," "improper equipment," or "existent pathology" was responsible. There is no epidemiologic evidence to support these contentions fully.

One of the major assumptions in analyzing death rates is that all participants are exposed in the same manner, for the same length of time, and to the same degree of hazard. These assumptions (for statistical purposes) are not true. In fact, it is thought that, if it were possible to categorize players on the basis of exposure, there would be a drastic variation in the rate of death for the groups exposed for different lengths of time. For example, it would not seem reasonable to expect the risk of death or injury to be identical for a starting quarterback, who plays in the entire game, and a specialty player, such as an extra-point kicker, who could conceivably miss playing in many games.

Although a description of deaths according to such variables as time, academic level, and type of activity is intuitively interesting, little value can be extracted from it without indices of exposure with which to relate it.

Available data on morbidity are conspicuously fragmented or, in some cases, totally absent. What information is available has been derived from schools, colleges, and interested individuals doing special studies. The data are generally reported in the same manner as fatalities, i.e., as numeric or percentage frequencies but without regard to a population at risk. Special analytic epidemiologic studies will be necessary for correctly characterizing death and injury data in their proper perspective.

There is an abundance of literature pertaining to the description and treatment of athletic injuries. These descriptions are valuable for characterizing new or declining injury patterns. The increase in neck injuries with the introduction of the newer football helmet is an example of hypothesis generation on the evidence of change in injury pattern. Occasionally, causal factors may be identified by a simple comparison of frequency distributions of injury types. That was the case when excessive numbers of neck injuries were found to result from modifications in design and use of the helmet. That is, however, an exception to the rule. Most evidence can be generated only after exhaustive evaluation of all relevant factors, either singly or in combination.

HOST VARIABLES

One of the basic questions in the investigation of injuries, as well as other causes of morbidity, is how injured persons differ from uninjured persons. Some football players go through high school, college, or the professional ranks without suffering severe injury.* Why is there such a variation in injury experience? It could be accounted for on the basis of differences in host factors, such as age, pathologic states, and athletic condition.

One approach is to visualize the mechanisms of injury as partly (or entirely) the contribution of host susceptibility and/or an injurious event (game or practice) in a hazardous environment. This approach has been successful in other studies relating to accident causation. For example, Haddon *et al.*[3] showed that the excessive number of skiing injuries among women is attributable to variations in musculoskeletal injury threshold between the sexes, rather than to variations in ski bindings.

Inherent host factors include age, weight, height, body build, physiologic status, existing pathologic conditions, and psychologic or emotional traits. All these are intuitively of interest and are amenable to prospective or retrospective epidemiologic studies. Unfortunately, no known studies compare these factors between injured and uninjured populations or develop estimates of risk attributable to the presence or absence of these factors in tackle football. The value of investigating host factors can be demonstrated on the basis of the evidence

* The reference to "severe injury" (an injury necessitating medical attention) is prompted by my contention that the elimination of all injuries is not consistent with the nature of football itself. Minor cuts, bruises, strains, and sprains are part of the game, but severe injuries prohibit participation by players and, in effect, threaten the existence of football as an athletic contest.

collected at the University of Minnesota in a study of touch-football injuries.[5] Although the difference in mean age between injured and uninjured players was significant ($p < 0.0001$), the actual difference in age was only about 1 year (Table 1). The practical application of this finding is doubtful. Many of the players were between 18 and 23 years old. The injury-predictive quality of age was illustrated when the risk of injury was quantitated for each age interval. As can be seen in Table 2, players 23 years old or older had a risk of serious injury almost four times as high as that of any other age group.

TABLE 1
Comparison of mean ages of uninjured, injured, and seriously injured touch-football players

Injury status	No. players	Mean age, years	Variance	z^a	p^b
No injury	2311	20.3	5.25	-	-
Injury	283	21.3	10.08	6.62	<0.0001
Major injury	151	21.6	12.45	5.62	<0.0001

[a] Absolute value of variate of unit normal distribution.
[b] For two-tailed tests.

TABLE 2
Age-specific injury attack rates and risk ratios in touch-football players

		Injured			Seriously injured		
Age, years	No. players	No.	Rate, %	Risk ratio[b]	No.	Rate, %	Risk ratio[b]
<19	563	34	6.0	1.0	16	2.8	1.0
19-20	975	93	9.5	1.6	49	5.0	1.8
21-22	655	87	13.3	2.2	44	6.7	2.4
≥23	400	69	17.3	2.9	42	10.5	3.7
Total	2593	283	10.9	--	151	5.8	--

[a] "Seriously injured" players sustained major injuries.
[b] Attack rate of age group divided by attack rate of age group with lowest rate.

One of the more provocative findings of the Minnesota study was that participants who had been injured previously in high-school, college, or intramural football games had a risk of injury several times higher than players who had played in high-school, college, or intramural games and were not injured (Tables 3, 4, and 5).

It was postulated that the excessive risk was due to age. That is, older players in college would naturally have more opportunity for exposure to injury than younger players. A further analysis comparing age-specific attack rates according to history of previous injury showed, however, that the excessive numbers of injuries to players with a prior history of intramural football injury could not be accounted for on the basis of age (Table 6). Many other host factors were investigated and are summarized elsewhere.[6]

Many host factors can be investigated in an epidemiologic manner. Age, weight, height, body build, some physiologic or emotional conditions, body conditioning or strength, etc., are easily recognized as possible contributing factors to the production of injuries but remain as only potential areas of productive research.

AGENT VARIABLES

Time

Not all games result in injuries to their participants. The epidemiologic process is uniquely able to identify the factors of the game that seem to contribute to the production of injuries. The distribution of deaths according to time of the game is one of the more interesting variables. There appears to be, for example, an excessive risk of death in the later quarters of football games.[1] The often-quoted reason for this finding is fatigue, but no epidemiologic evidence supports this contention.

There are no reliable statistics on morbidity from tackle-football injuries including an entire football season. The study at the University of Minnesota[5] indicated that the distribution of injuries is significantly excessive in the latter part of football games (Table 7).

The peak incidence of deaths appears to occur in the month of October; however, the failure to relate these deaths to numbers of games of exposure hampers the derivation of any meaningful inferences. Some other variables--such as type of activity, nature of the play, score of the contest at time of injury, and number and types of personal penalties--should be carefully considered in assessing the role of game factors in injury production. The epidemiologic process could be most beneficial in evaluation of the role of game variables.

TABLE 3
Attack rates and risk ratios according to history of disabling injury in high-school football

History of high-school football injury	No. players	Injured					Seriously injured				
		No.	Rate, %	Risk ratio	z	p^a	No.	Rate, %	Risk ratio	z	p^a
Yes	385	72	18.7	2.0	5.34	<0.0001	36	9.4	1.8	3.2	<0.003
No	2208	210	9.5	1.0			115	5.2	1.0		

[a] For two-tailed tests.

TABLE 4
Attack rates according to history of high-school football and disabling high-school football injury

History of high-school football	No. players	Injured		Seriously injured	
		No.	Rate, %	No.	Rate, %
High-school football with injury	385	72	18.7	36	9.4
High-school football without injury	1059	111	10.5	60	5.7
No high-school football	1149	55	4.8	55	4.8

Students who played high-school football and were injured, compared with students who played high-school football and were not injured: all injured, $z=4.22$, $p<0.0001$; seriously injured, $z=2.54$, $p=0.001$.

Students who played high-school football and were not injured, compared with students who did not play high-school football: all injured, $z=5.20$, $p<0.0001$; seriously injured, $z=0.98$, $p=0.327$.

Students who played high-school football and were injured, compared with students who did not play high-school football: all injured, $z=8.80$, $p<0.0001$; seriously injured, $z=3.40$, $p=0.007$.

Equipment

Probably one of the most widely discussed aspects of football is protective equipment. The football helmet appears to have received the lion's share of attention. Many authorities in sports medicine believe that the cause of many head injuries is directly related to the design of the helmet. No epidemiologic studies, however, have compared the efficiency of the different types of helmets available. Such a study would compare the types of helmet in use and their frequency of use with the numbers of head and neck injuries occurring to players wearing the different types. A probability of head/neck injury per type of helmet could be derived once some confounding variables were controlled.

TABLE 5
Attack rates and risk ratios according to history of college intramural touch-football injury before fall quarter, 1966

Intramural touch-football injury history	No. players	Injured					Seriously injured				
		No. injured	Rate, %	Risk ratio	z	p^a	No. injured	Rate, %	Risk ratio	z	p^a
Yes	167	51	30.5	3.2	8.44	<<0.0001	34	20.4	4.2	8.31	<<0.0001
No	2426	231	9.5	1.0			117	4.8	1.0		

[a] For two-tailed tests.

TABLE 6
Age-specific attack rates according to history of intramural football and intramural football injury

	(A) Intramural football injury			(B) Intramural football but no injury			(C) No intramural football			Test comparison					
		Injured			Injured			Injured		A *vs.* B		B *vs.* C		A *vs.* C	
Age, years	No. players	No.	%	No. players	No.	%	No. players	No.	%	z	p	z	p	z	p
All injured															
<19	2	0	0.0	46	2	4.3	515	32	6.2	-0.30	0.764	-0.51	0.610	-0.36	0.719
19-20	50	14	28.0	484	46	9.5	441	33	7.5	3.95	0.0001	1.13	0.258	4.68	0.0001
21-22	65	18	27.7	384	45	11.7	206	23	11.2	3.44	0.0005	0.20	0.841	3.25	0.001
≥23	50	19	38.0	259	37	14.3	91	13	14.3	3.99	0.0001	0.00	1.000	3.22	0.001
Total	167	51	30.5	1173	130	11.1	1253	101	8.1	6.92	0.0001	2.65	0.008	8.89	0.0001
						Stratified (z', p')				5.54	0.0001	0.41	0.682	5.40	0.0001
Seriously injured															
<19	2	0	0.0	46	1	2.2	515	11	2.1	-0.21	0.833	0.02	0.984	-0.21	0.834
19-20	50	8	16.0	484	29	6.0	441	14	3.2	2.67	0.008	2.04	0.041	4.18	0.0001
21-22	65	14	21.5	384	25	6.5	206	8	3.9	3.99	0.0001	1.35	0.177	4.56	0.0001
≥23	50	12	24.0	259	21	8.1	91	9	9.9	3.34	0.0008	-0.52	0.603	2.25	0.024
Total	167	34	20.4	1173	76	6.5	1253	41	3.3	6.15	0.0001	4.14	<0.001	9.41	0.0001
						Stratified (z', p')				4.90	0.0001	1.45	0.147	5.39	0.0001

TABLE 7
Distribution of injuries and major injuries in the two halves of football games[a]

	Injuries			Serious injuries		
Half of game	No.	No. expected	% of total	No.	No. expected	% of total
1st (0-21 min)	110	136	40.4	57	72.5	39.3
2nd (22-42 min)	162	136	59.6	88	72.5	60.7
	$\chi^2_{(1)}=9.942$, $p<0.005$			$\chi^2_{(1)}=7.510$, $p<0.01$		

[a] Practice injuries and injuries of unknown time excluded from analysis.

Recently, the face mask was introduced into tackle football. The basis for addition of the face mask was the intuitive feeling that a bar of sorts would protect the player's face from direct blows. There was no experimental trial to evaluate the effectiveness of face masks. Some physicians are now challenging their effectiveness on the grounds of an increased incidence of head and face injuries directly or indirectly attributable to it.

The evidence to support the use of mouth protectors was gained largely through the epidemiologic process. Several studies have demonstrated the effectiveness of the mouth protectors in reducing or preventing mouth and dental injuries by comparing the numbers of injuries to players similarly exposed but wearing and not wearing mouth protectors. The methodologic approach should be equally applicable to other types of equipment.

Other equipment has received far less attention. Shoulder pads, hip pads, and thigh pads have been ignored by the researcher, as evidenced by the lack of data supporting the protection claimed for the various styles and types of equipment.

Football shoes have received considerable attention in recent years. Some advocate low-cut shoes for performance. Others feel that high-top shoes prevent ankle injuries. To some, the type of cleat appears to be related to the incidence of foot, ankle, knee, and leg injuries. Hanley[4] cites his study to support a cleatless heel. Other investigators recommend a conical, soccer, or ripple-type heel. This problem can be resolved only when information on risk of injury according to type of shoe or cleat has been gathered and evaluated. The results may be different from those expected, as shown by the findings at the University of Minnesota,[5] which indicated that the risk of foot, ankle, or knee injury for players with a standard touch-football shoe was twice as great as for players similarly exposed but wearing tennis shoes (Table 8).

TABLE 8
Attack rates and risk ratios of foot and leg injuries according to shoe type

Shoe type	No.	%	Foot and leg injuries			
			No.	No. expected	Rate, %	Risk ratio
Non-touch-football	8,909	72.5	71	91.4	0.8	1.0
Touch-football	3,376	27.5	55	34.6	1.6	2.0
Total	12,285	100.0	126	126.0	1.0	--

$\chi^2_{(1)}=19.507$, p <0.0005

ENVIRONMENT VARIABLES

There is ample evidence of the relationship of football fatalities to high temperature and humidity. There is no evidence, however, on the occurrence of football injuries at high temperature or humidity. It would help to complete the assessment of environmental factors if there were evidence on such variables as humidity, barometric pressure, lighting (night games or practice), wind direction and speed, and condition of turf.

The factors of uneven sod, holes, debris, and other foreign matter on the playing surface are mostly controlled. In the last 2 years, there has been some evidence, as reported by Whitehurst,[8] leading to the belief that artificial turf may drastically reduce the frequency and severity of foot and leg injuries. The number of man-games of exposure is far too low to offer conclusive evidence to support his finding, but with additional fields and hours of exposure, relationships may be drawn.

NATIONAL SURVEY

To assist in evaluating the entire problem of tackle-football injuries, an effort is underway through the cooperation of the American College of Sports Medicine's Committees on Safeguards and Medical Aspects of Sports and their member groups to compile basic morbidity data on injuries. The instrument under consideration is shown in Fig. 1. Through a tedious process of field trial and experimentation, it is hoped that an acceptable surveillance form will be introduced in about 100 colleges and 100 high schools for the 1969 fall football season.

INSTRUCTIONS FOR COMPLETING THIS FORM

A ALL QUESTIONS MUST BE ANSWERED EXCEPT THOSE MARKED WITH AN ASTERISK (*) WHICH MAY BE OMITTED IF NOT PERTINENT.

B **EXCEPT WHERE INDICATED, ONLY ONE RESPONSE MAY BE MARKED.**

C THIS FORM MUST BE COMPLETED WITHIN 48 HRS. AFTER THE INJURY HAS OCCURRED.

UNK=UNKNOWN.
NEC=NOT ELSEWHERE CLASSIFIED.

PLAYER IDENTIFICATION

1 Initials_____ F. M. L.

2 Place of Injury_____

3 Injury Number (Col 7) ( ) 1st ( ) 2nd ( ) 3rd ( ) 4th ( ) 5th+ — Col 7

INSTITUTIONAL IDENTIFICATION — Col 8

*4 IF HIGH SCHOOL PLAYER

a. **Type of High School** (Col 8)
1 ( ) Public 3 ( ) Private (non-rel)
2 ( ) Paroch 4 ( ) Other

b. **Size of High School** (Col 9) — Col 9
1 ( ) 500 3 ( ) 1000-1499
2 ( ) 500-999 4 ( ) 1500+

*5 IF JR. COLLEGE, COLLEGE OR UNIVERSITY — Col 10

a. **Type of School** (Col 10)
1 ( ) Primarily Tax Supported
2 ( ) Private/Religious

b. **Size of School** (Col 11) — Col 11
1 ( ) 1000 4 ( ) 10000-19999
2 ( ) 1000-4999 5 ( ) 20000+
3 ( ) 5000-9999

PLAYER DESCRIPTION — Col 12-13

6 AGE (Col 12-13)

01 ( ) <14	05 ( ) 17	09 ( ) 21
02 ( ) 14	06 ( ) 18	10 ( ) 22
03 ( ) 15	07 ( ) 19	11 ( ) 23
04 ( ) 16	08 ( ) 20	12 ( ) 24+
		99 ( ) UNK

7 YEAR OF ELIGIBILITY (Col 14) — Col 14

1 ( ) Freshman	3 ( ) Junior
2 ( ) Sophomore	4 ( ) Senior
	9 ( ) UNK

8 WEIGHT (Col 15) — Col 15

1 ( ) <140	5 ( ) 200-219
2 ( ) 140-159	6 ( ) 220-239
3 ( ) 160-179	7 ( ) 240-259
4 ( ) 180-199	8 ( ) 260+
	9 ( ) UNK

9 HEIGHT (ft/in) (Col 16) — Col 16

1 ( ) <5'6"	5 ( ) 6,3-6,5
2 ( ) 5,6-5,8	6 ( ) 6,6-6,8
3 ( ) 5,9-5,11	7 ( ) 6,9+
4 ( ) 6,0-6,2	9 ( ) UNK

*10 PLAYING POSITION WHEN INJURED IN GAME, SCRIMMAGE, OR DRILLS (check one box only) (Col 17-18) — Col 17-18

01 ( ) Split End	10 ( ) Flanker
02 ( ) Tight End	11 ( ) Halfback
03 ( ) Tackle	21 ( ) Safety
04 ( ) Guard	22 ( ) Line Backer
05 ( ) Ce ter	23 ( ) Halfback
06 ( ) QB	24 ( ) Guard
07 ( ) Fullback	25 ( ) Tackle
08 ( ) Wingback	26 ( ) End
09 ( ) Tailback	99 ( ) UNK

INJURY DESCRIPTION — Col 19-30

11 NATURE OF THE INJURY (primary) (Col 19-30)

01 ( ) Abrasion	11 ( ) Sprain Severe (tear)
02 ( ) Contusion	12 ( ) Strain (muscle) Mild (non-tear)
03 ( ) Concussion (minor)	13 ( ) Strain (muscle) Severe (tear)
04 ( ) Concussion (major)	14 ( ) Sun Stroke
05 ( ) Dental Injury	15 ( ) Heat Exhtn
06 ( ) Dislocation	16 ( ) Frost Bite
07 ( ) Fracture	17 ( ) Meniscus (tear)
08 ( ) Laceration	18 ( ) Other
09 ( ) Puncture Wd	99 ( ) UNK
10 ( ) Sprain Mild (non-tear)	

12A BODY PART INVOLVED (check one major injury only) (Col 21-22) — (Col 21-22)

Head and Face	
01 ( ) Jaw	08 ( ) Eyelid/Brow
02 ( ) Face	09 ( ) Lip
03 ( ) Nose	10 ( ) Tongue
04 ( ) Scalp	11 ( ) Teeth
05 ( ) Skull	12 ( ) Ear
06 ( ) Forehead	13 ( ) Head CNS
07 ( ) Eye	14 ( ) Other Head
Neck	
21 ( ) Vertebrae	23 ( ) Nerve
22 ( ) Soft Tissue	24 ( ) Other Neck
Chest	
31 ( ) Ribs	33 ( ) Heart
32 ( ) Lung	34 ( ) Other Chest
Abdomen	
41 ( ) Liver	43 ( ) GI Tract
42 ( ) Spleen	44 ( ) Other Abdomen
GU Tract	
51 ( ) Kidney	54 ( ) Penis
52 ( ) Bladder	55 ( ) Other GU
53 ( ) Testes	
Musculo-Skeletal (M-S)	
60 ( ) Vertebrae	70 ( ) Pelvis
61 ( ) Acromeoclavicular	71 ( ) Iliac Crest
62 ( ) Shoulder NEC	72 ( ) Hamstring
63 ( ) Clavicle	73 ( ) Hip
64 ( ) Upper Arm	74 ( ) Thigh
65 ( ) Elbow	75 ( ) Knee
66 ( ) Forearm	76 ( ) Lower Leg
67 ( ) Wrist	77 ( ) Ankle
68 ( ) Hand	78 ( ) Foot
69 ( ) Finger	79 ( ) Toe
	80 ( ) Other M-S

12B Multiple Injury (describe) (Col 23-30) — (Col 23-30)

99 ( ) UNK

13 BODY SIDE OF INJURY (Col 27) — (Col 27)

1 ( ) Right	3 ( ) Not Applicable
2 ( ) Left	9 ( ) UNK

14 SEVERITY OF INJURY (Col 28) — (Col 28)

1 ( ) Fatal
2 ( ) Hospitalized
3 ( ) Injury With No Participation (lost time)
4 ( ) Injury But With Partial Participation
5 ( ) Injury But With Active Participaiton
9 ( ) UNK

15 PRE-EXISTING PHYSICAL/SENSORY PROBLEMS (Col 29) — (Col 29)

Was there evidence of a pre-existing physical or sensory problem associated with this injury?
1 ( ) Yes 2 ( ) No 3 ( ) Uncertain 9 ( ) UNK

16 DID THE PLAYER CONTINUE TO PARTICIPATE IN SAME GAME OR SESSION AFTER INITIAL INJURY SITUATION? (Col 30) — (Col 30)

1 ( ) Yes 2 ( ) No

FIGURE 1
Form being considered for collecting injury data. (Continued on facing page.)

*17A WAS THIS A REINJURY OF A PREVIOUS TRAUMATIC CONDITION? (Col 31) — (Col 31)
1 ( ) Yes 2 ( ) No 9 ( ) UNK

*17B IF A REINJURY, HOW DID IT OCCUR? (Col 32) — (Col 32)
1 ( ) Athletic Activity 9 ( ) UNK
2 ( ) Nonathletic Activity

INJURY DESCRIPTORS — (Col 33)

18 GENERAL ACTIVITY (Col 33)
1 ( ) Game 4 ( ) Drill
2 ( ) Scrimmage 5 ( ) Other
3 ( ) Exercise 9 ( ) UNK

*19 IF GAME OR SCRIMMAGE, INDICATE TYPE OF PLAY (Col 34) — (Col 34)
1 ( ) Pass 6 ( ) Fumble
2 ( ) Run 7 ( ) Other
3 ( ) Punt 8 ( ) NA
4 ( ) Kick Off 9 ( ) UNK
5 ( ) Field Goal

20A ACTIVITY AT TIME OF INJURY (Col 35) — (Col 35)
1 ( ) Blocking or Blocked
2 ( ) Kicking or Blocking Kick
3 ( ) Tackling (or being tackled)
4 ( ) Pile-up
5 ( ) Stepped-on
6 ( ) Running (noncontact)
7 ( ) Running (fixed object)
8 ( ) Other
9 ( ) UNK

20B STATUS (Col 36) — (Col 36)
1 ( ) Offensive
2 ( ) Defensive
9 ( ) UNK

21 FIELD SURFACE CONDITION (Col 37) — (Col 37)
1 ( ) Dry 3 ( ) Frozen
2 ( ) Wet/Mud 9 ( ) UNK

22 FIELD SURFACE TYPE (Col 38) — (Col 38)
1 ( ) Grass 3 ( ) Artificial
2 ( ) Dirt Soil 9 ( ) UNK

23 TIME OF INJURY (Col 39) — (Col 39)
1 ( ) Spring Prac 3 ( ) Game
2 ( ) Pre-season Prac 4 ( ) Between Game Prac
9 ( ) UNK

24 MONTH OF INJURY (Col 40-41) — (Col 40-41)
01 ( ) Jan 05 ( ) May 09 ( ) Sept
02 ( ) Feb 06 ( ) June 10 ( ) Oct
03 ( ) Mar 07 ( ) July 11 ( ) Nov
04 ( ) Apr 08 ( ) Aug 12 ( ) Dec

*25 IF INJURY OCCURRED IN REGULAR SEASON, INDICATE WEEK OF PLAY OF SEASON (Col 42-43) — (Col 42-43)
01 ( ) 07 ( ) 13 ( )
02 ( ) 08 ( ) 14 ( )
03 ( ) 09 ( ) 15 ( )
04 ( ) 10 ( ) 16 ( )
05 ( ) 11 ( ) 17+( )
06 ( ) 12 ( ) 99 ( ) UNK

26 DAY OF WEEK OF INJURY (Col 44) — (Col 44)
1 ( )Mon 4 ( ) Thurs 6 ( ) Sat
2 ( )Tues 5 ( ) Fri 7 ( ) Sun
3 ( ) Wed 9 ( ) UNK

27 TIME OF DAY OF INJURY (Col 45) — (Col 45)
1 ( ) 6-8:59 AM 5 ( ) 6-8:59 PM
2 ( ) 9-11:59 AM 6 ( ) 9-11:59 PM
3 ( ) 12 noon-2:59 PM 7 ( ) 12-5:59 AM
4 ( ) 3-5:59 PM 9 ( ) UNK

28 TEMPERATURE AT TIME OF INJURY — Col 46
0 ( ) 20 3 ( ) 40-49 6 ( ) 70-79
1 ( ) 20-29 4 ( ) 50-59 7 ( ) 80-90
2 ( ) 30-39 5 ( ) 60-69 8 ( ) 90-99
9 ( ) 100+

29 CLIMATE AT TIME OF INJURY (Col 47) — Col 47
1 ( ) Clear or Cloudy 3 ( ) Snow or Sleet
2 ( ) Rain or Drizzle 9 ( ) UNK.

*30 QUARTER OF INJURY (game injuries only) (Col 48) — Col 48
1 ( ) 1st 3 ( ) 3rd 9 ( ) UNK
2 ( ) 2nd 4 ( ) 4th

*31 IF EQUIPMENT WAS REQUIRED, WAS IT WORN, I.E., LEFT PADS OFF, ETC. (Col 49) — Col 49
1 ( ) Yes 2 ( ) No 9 ( ) UNK

*32 IF A HEAD INJURY, GIVE HELMET DESCRIPTION — Col 50
a. **Type Faceguard** (Col 50)
1 ( ) One Bar 2 ( ) Two Bars 3 ( ) Cage — Col 51
b. **Suspension Type** (Col 51)
1 ( ) Yes 2 ( ) No
Internal Padding (Col 52) — Col 52
1 ( ) Yes 2 ( ) No
2 **Combination Suspension & Padding** (Col 53) — Col 53
1 ( ) Yes. 2 ( ) No

*33 IF A DENTAL OR MOUTH INJURY, WAS MOUTHPEICE WORN (Col 54) — Col 54
1 ( ) Yes 2 ( ) No 7 ( ) UNK

34 IF ILIAC CREST, PELVIS, OR THIGH INJURY, DESCRIBE PADDING USED TO COVER AREA (Col 55) — Col 55
1 ( ) Saft 3 ( ) Combination 9 ( ) UNK
2 ( ) Hard 4 ( ) None

*35 IF ANKLE, KNEE, OR LEG INJURY — Col 56
a. **Describe Joint** (Col 56)
1 ( ) Taped 3 ( ) Braced 9 ( ) UNK
2 ( ) Wrapped 4 ( ) None
b. **Shoe Type** (Col 57) — Col 57
1 ( ) Lowcut 3 ( ) Hightop 5 ( ) Other
2 ( ) Medium 4 ( ) Soccer 9 ( ) UNK
c. **Cleat Type** (Col 58) — Col 58
1 ( ) None 3 ( ) Soccer 5 ( ) Ripple
2 ( ) Conical 4 ( ) Multiple 6 ( ) Other
d. **Heel** (Col 59) — Col 59
1 ( ) Ring 3 ( ) Shoe 9 ( ) UNK
2 ( ) Bar 4 ( ) Cleat

36 IS THERE ANY EVIDENCE WHICH INDICATES THAT THE INJURY WAS CAUSED OR THE SEVERITY AMPLIFIED BECAUSE OF INADEQUATE, IMPROPERLY WORN, OR FAILURE IN THE DESIGN OR MATERIALS IN THE EQUIPMENT WORN? (Col 60) — Col 60
1 ( ) Yes 2 ( ) No 4 ( ) Uncert 9 ( ) UNK

37 IS THERE ANY EVIDENCE WHICH INDICATES THAT THE SEVERITY OF THE INJURY WAS LESSENED BY THE PROTECTIVE EQUIPMENT WORN AT THE MOMENT OF INJURY? (Col 61) — Col 61
1 ( ) Yes 2 ( ) No 4 ( ) Uncert 9 ( ) UNK

38 RCORD COMPLETED BY (Col 62) — Col 62
1 ( ) Trainer (octive 5 ( ) Manager
2 ( ) Trainer (student) 6 ( ) Physician
3 ( ) Coach 7 ( ) Other
4 ( ) Ass't Coach

Many of the factors introduced in this paper will be evaluated from the national survey. There are, of course, many other variables that cannot be critically analyzed from a surveillance effort. Through the epidemiologic process, however, the factors that appear to be most provocative can be studied in greater detail. The most meaningful data that may be derived from the survey will be a reasonably reliable national picture of the problem of tackle-football injuries.

SUMMARY

The epidemiologic method has been shown to afford a very useful approach to the study of accidental injuries. Through the mechanism of mortality and morbidity studies, followed by well-designed investigation of contributing player, game, and environmental factors, evidence may be generated to answer some of the more confounding questions on the etiology of football injuries. The process of evaluating the factors that appear to differentiate injured from uninjured players would seem to be highly productive for characterizing the risks associated with the presence or absence of these factors.

An effort to gain the information needed to begin epidemiologic study is now underway through the implementation of a national surveillance system of tackle-football injuries, in the hope that adequate data on the predictive character of some host, game, and environmental variables may be collected that will, in turn, shed light on the complex etiology of football fatalities and injuries.

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METHODS OF ESTABLISHING HUMAN TOLERANCE LEVELS

LAWRENCE M. PATRICK

For obvious reasons, human tolerance cannot be established through direct exposure of people to an injury environment. Furthermore, tolerance depends on environment and is therefore different for different exposure conditions. For example, the energy in a bullet might be sufficient to cause injury, whereas the same quantity of energy in a large mass traveling at low speed might merely push a person out of the way without injuring him. Injury through a crushing force is essentially a static injury, and a bullet injury is the result of a dynamic impact; the protection required for each is different.

Consequently, human tolerance must be based on quantitative and qualitative data related to specific environments. Several methods of establishing the tolerance include: calculation based on known conditions, such as mass, velocity, hardness, and geometry, combined with velocity, direction, and impact site on the body; simulation of the environment; and measurement during activity in the normal environment. Unfortunately, not all the conditions are usually known, so it is often necessary to conduct experiments to establish them before calculations can be made. In such a case, it is probably as easy and more accurate to measure impact conditions directly by simulating the environment.

Simulation of an environment requires duplicating conditions accurately. For example, the crash environment for an automobile is duplicated by crashing it under the same conditions as those being studied. The sports environment can be simulated by ascertaining from observations and high-speed movies the exact conditions of each impact under study. These can then be duplicated with anthropomorphic dummies or human cadavers propelled with suitable mechanical devices and with the instrumentation necessary to measure injury-producing accelerations or forces.

The most obvious and, in some respects, the optimal method for establishing tolerance is to measure the appropriate conditions in the real environment. The forces on a knee striking a helmet, the ground, or any other object can be measured with a force-indicating covering (Met-net) or some other device. Unfortunately, the forces measured in this manner are seldom near the human tolerance level and would

therefore generally be too low, unless enough data were obtained to include cases of injury. The drawback of this type of damage is the required modification of a player's equipment or the addition of transducers, which in themselves might cause injury to the player.

An alternate to measuring the environment accurately is to set impact values well above those which can be encountered in the environment and provide protection for the higher values. This has the major disadvantage of requiring the levels to be excessively high, so that the protective devices must be larger than truly necessary.

A voluntary tolerance under simulated conditions indicates approximate injury tolerance level, but it is generally low; true injury tolerance level is likely to be two to four times the voluntary level.

A technique used extensively at Wayne State University involves the establishment of the voluntary tolerance levels followed by impacts of the same kind to human cadavers at severities that are increased until there is skeletal damage. It has been shown that bone does not change its physical properties substantially after death if it is kept moist. The intact cadavers are embalmed; consequently, the bone is moist and the data are realistic. The cadaver data are also conservative and must be corrected for age, because the cadavers used are generally those of persons who were over 60 years old at the time of death. The bones of such cadavers break more readily than would those of persons in the prime of life and in good physical condition. The use of cadavers is limited to tests of skeletal damage and, in some cases, internal-organ damage that can be deduced from skeletal damage. Brain-injury levels have been established by this method. A linear fracture that results from impact of the forehead against a hard, flat surface produces a moderate concussion. Therefore, an impact to the forehead from a hard, flat surface that produces a linear fracture is equated with a moderate concussion.

Many investigators have used anesthetized animals to study impact tolerance. The mechanism of injury can be accurately determined in this manner, but the correlation between animals and humans has not been found. Therefore, the use of animals is of questionable value in sports injuries.

Human tolerance of impact can be measured in terms of force, pressure (force per unit area), and/or acceleration. Important considerations include the part of the body; the direction, velocity, mass, and geometry of the impact; the hardness, rigidity, and roughness of the involved surfaces; and the player's physical condition. Many of these items are interrelated in such a way that protection against one automatically provides protection for some of the others. There are some intuitive approaches to protection that have been proved through trial and error. In general, it is known that a soft surface is less injurious than a hard surface, a large area less injurious than a small area, and low velocity less injurious than high velocity, if other conditions are the same.

Tolerance levels for impact to various parts of the body have been established that are applicable to the sports environment. These include impact to the head, knee, chest, and face.[3,5] If the tolerance level is known, then protection can be provided by designing a protective device that will attenuate the force or acceleration of an impact to the tolerable level.

KNEE INJURIES

Knee injuries have been reported as among the most prevalent injuries in football. Although they are not dangerous to life, they can end players' careers and can cause prolonged pain and discomfort. Some surgical procedures can repair many knee injuries satisfactorily but require rather long periods of convalescence.

A proposed method of determining knee-impact tolerance follows: The environment must first be established by subjecting volunteers to loads simulating those of impact from other players in various directions. Obviously, the loads must be well below the injury level. The same loads should then be applied to cadavers, so that the deflections and loads may be compared with those of the volunteers to establish equivalence. Thereafter, the loads should be increased until fracture or soft-tissue damage results. The types of loads to be applied should be decided on the basis of high-speed movies, with location, velocity, and force as important measures of injury. Some knee injuries have been reported to occur with no impact, when turf and cleat conditions combine to produce bending or torsion loads beyond the injury level. Forces and accelerations during voluntary and cadaver loading can be measured by a suitable transducer, such as hydraulic pressure in a bladder, strain in thigh pads, and the previously mentioned Met-net. If an impact involves a rigid or semirigid body, an indication of the force can be achieved by mounting an accelerometer on the impacting mass.

Protection of the knee requires limitation of angular excursion. A protective device to limit angular excursion must not be restrictive to the point of impairing the normal function of the knee, and it must not result in other injuries to the wearer or to other players. Splints and braces have been suggested as knee protectors but have not been acceptable and, in most cases, have not been effective. A pneumatic splint or some other new device is required. Of course, the best way to eliminate the injury is to avoid the condition that causes it. A breakaway cleat or optimization of the turf-cleat interface would minimize or eliminate injuries related to the turf-cleat interface, and more stringent refereeing could reduce exposure to impacts from unprotected directions.

Many knee injuries occur when players are blocked from a blind side. This can be avoided by providing adequate visibility in the helmet and by using a device to warn of impending impact. Perhaps some kind

of radar or other electronic gear can be developed to give a player a signal when a blocker is approaching from a blind side.

Another method, which appears ridiculous but might be possible, would use a pneumatic splint that would inflate immediately after impact but in sufficient time to prevent excessive angular motion of the knee in any direction. If the maximal velocity is assumed to be 25 ft/sec (7.6 m/sec) and an inflation time of 15 msec is possible, then it will require a trigger approximately 4.5 in. (11.4 cm) from the leg to initiate inflation in time to prevent an injury. A similar device has been proposed and is under development for use in automobiles to minimize occupant injury from impact. In that case, a sensing device triggers an inflatable airbag in front of the passenger when the acceleration caused by the impact reaches a predetermined level. Obviously, innovation will be required in the solution of the knee-injury problem.

A hydraulic bag around the knee is another possibility; the fluid pressure would increase with the impact, providing stiffness or bracing for the knee and attenuating the force.

HEAD INJURIES

Head-impact tolerance is probably better known than any other. It is the most important part of the body to protect and consequently has been given the greatest consideration. Transmitted acceleration to the head is the characteristic associated with tolerance by most investigators. For massive impacts where moderate but not fatal injury results, as is the case in automobile-racing collisions, a severity index of 1000* or more has been established using the equation established by Gadd.[2] A transmitted acceleration of up to 400 *g* for a few milliseconds at a time has also been established as a tolerance level.[1] Voluntary and cadaver neck reactions in the whiplash environment have been reported by Mertz.[4] Chest-impact forces of 1100-1200 lb (500-550 kg) on a padded target 6 in. (15 cm) in diameter have been established[6] on cadavers and from investigations of accidents that involved known forces of up to 1800 lb (815 kg) with the force distributed over the thorax and abdomen through a steering wheel.[7]

Many different types of helmets are in use in industry and sports. The well-known construction hard hat is used primarily to prevent concentrated loads from falling objects. They provide very little attenuation of impact and are not effective against side impacts. Automobile- and motorcycle-racing helmets are designed for one massive impact and are not suitable for football players. However, many of their design characteristics are relevant and will be useful in designing the optimal football helmet.

* Field data on impacts to windshields and instrument panels indicate the value of 1000 to be satisfactory or even conservative for single severe head impacts. However, it is probably too high for repeated blows such as those received by football players.

Current statistics, about which there seems to be some controversy, show very few fatalities in football, compared with the number of players involved. There are generally said to have been about 12 fatalities in 1968, compared with between 2 and 3 million participants. Considering that each participant is exposed to many impacts each year, it is obvious that the present helmet is doing a good job in providing protection and that modifications must be made with care to avoid increasing the number of fatalities.

The present trend is to apply the same standard to football helmets that is used for automobile racing and other high-hazard vehicular activities, as specified in Standard Z90.1 of the United States of America Standards Institute. I do not think that is a safe approach to the problem of head protection for football players. Standard Z90.1 is based on a design criterion of one severe impact with moderate injury. To achieve this protection, the available distance must be used efficiently by providing the maximal force that the head can stand over the total stopping distance. If this approach is used in the standard for football helmets, the transmitted accelerations will be comparatively high, and it is possible that more fatalities and injuries will result, rather than fewer, because of the cumulative effects of many impacts at too-high acceleration levels due to the stiff liner.

If a player will be subjected to many impacts, a lower transmitted acceleration is required for the less severe impacts. Consequently, the standard should be based on many impacts of a lower energy level, with a greatly reduced acceptable level of transmitted acceleration. This can be accomplished by a softer liner, which would "bottom out" if subjected to the higher energy specified in Standard Z90.1, but which would effectively attenuate the impact at the energy levels and conditions of football. The input energy will have to be determined from observations and measurements made on the playing field, with the design predicated on mitigating injuries from football impacts, rather than the more severe impacts encountered in other activities.

SUMMARY

It is essential that the protective equipment for sports be designed on the conditions associated with those sports. The environment for each sport must be determined through calculation, measurement, and/or simulation of playing conditions. Emphasis must be placed on the most severe conditions, but the cumulative effect of many less severe exposures should also be considered. Study of the performance of successful protective equipment will be valuable in determining the design criteria for future protective equipment.

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HUMAN TOLERANCE OF IMPACT

JOHN P. STAPP

Biodynamics deals with the effects of mechanical forces on living tissues. It is concerned with correlating mechanical-force factors with the physiologic and anatomic changes that occur in a living body or any of its parts during or after exposure to mechanical stresses. These changes include:

(1) Acute, immediately reversible anatomic and physiologic reactions that are not injurious.

(2) Acute, persistent anatomic and physiologic reactions with signs and symptoms of injury, such as:

(a) neurocirculatory shock, concussion, pain, and anxiety with associated changes in vital signs and physiologic reactions;

(b) abrasions, contusions, ecchymoses, hematomas, and ruptures of skin, membranes, and viscera;

(c) strains, sprains, and dislocations of joints;

(d) fractures of bones and cartilages; and

(e) associated reversible disabilities.

(3) Chronic, irreversible anatomic and physiologic effects with disabling injuries, such as:

(a) permanent impairment of function of an organ or structure of the body; and

(b) amputation or avulsion of or irreversible damage to an organ or structure.

(4) Fatal injury, including immediate and delayed effects on vital structures and functions due to:

(a) hemorrhage, hypoxia, obstruction, or direct trauma to respiratory or cardiac control centers;

(b) combined effects of multiple injuries and shock; and

(c) decapitation or evisceration.

All those conditions may result from abrupt velocity changes when a moving living body collides with another moving body or a stationary and resistant object or when a stationary body is struck by a moving object. Finding the quantitative relationship between mechanical-force characteristics and degrees of physiologic and anatomic effects provides criteria for survial without injury, survival with injury, and death. Such criteria are essential for determining limits of performance and specifications for protective design.

The quantitative stress analysis of man derives its upper limits from observations made on accidental or suicidal exposures of living humans to collision force, confirmed by experimental application of comparable dynamic force to cadavers and anesthetized animal subjects; in the range of reversible effects, anesthetized animals and human volunteers have been experimentally exposed to measured increments of dynamic force while responses were recorded. The quantitative relationship of collision force to its effects on the whole body and its parts determines the characteristics of human responses and vulnerabilities. The magnitude of difference between the threshold of injury to a vital organ and manifestations of irreversible injury constitutes a safety factor applicable to exposure limits and performance standards for protective equipment.

FALLS

In the United States in 1968,[29] falls caused more than 20,000 deaths, mainly from head injuries sustained after tripping or stumbling. An astronaut whose 170-lb body mass acquired 56 billion ft-lb of kinetic energy during a safe 5-min ascent into orbit,[29] more than 100 miles above the earth, returned safely to earth, only to be incapacitated a few weeks later by tripping and striking his head on the edge of a bathtub; the incident involved the dissipation of fewer than 560 in.-lb of energy in less than 0.01 sec. Terminal free-fall velocity of the human body is attained in dropping through air from 482 ft above sea level to a constant speed of 178 ft/sec, or about 120 mph, as air drag offsets the acceleration of gravity. The velocity is higher at altitudes of decreased air density, but retards to that at sea level before impact. In decreasing order from terminal velocity, a fairly complete spectrum of effects of free fall onto snow, water, earth, concrete, and steel landing surfaces can be constructed from observations of human falls from known heights.

Collision forces can be estimated from the height of free fall, the weight of the victim, the depth of plastic deformation of the impacted surface, the body orientation and contacting area at impact, and the location and extent of injuries, compared with those obtained in anesthetized animal and cadaver subjects in experiments in which forces were recorded.

In the case of survived falls soon after which death occurred from other causes, the nature and extent of nonfatal internal injuries observed at autopsy could be compared with those of anesthetized experimental animals after equivalent exposures to impact.

The pioneer in mechanical analysis of survived human falls was Hugh DeHaven, who founded the Cornell University Crash-Injury Research Program. DeHaven became involved in the problem of crash survival after a midair collision at 600 ft between World War I fighter planes; he sustained severe crash injuries, whereas the other pilot walked away from his demolished aircraft practically unhurt. During his convalescence, he began studying aircraft accidents, and thereafter he dedicated his life to crash-survival research, until his retirement in 1952. His analytic methods are illustrated in Table 1, based on data from two cases in a report on survived falls published in 1942.[5]

An estimate of compression forces can be made by assuming that half the body surface contacted the earth during indentation; therefore, dividing the product of body weight and impact *g* by half the body area will give compression, in pounds per square inch. According to Sendroy and Cecchini,[22] the body surface area for a 125-lb, 62-in. female would be 2635 in.^{2};

$$\frac{(125 \text{ lb})\ (135.8\ g)}{(2635 \text{ in.}^2)\ (2)} = 12.9 \text{ psi.}$$

Had the victim landed on her feet at the same impact velocity, assuming the area of the soles to be 50 in.^{2}, the compressive force would be 339.5 psi.

Corresponding values for the other fall victim would be 15.9 psi for half the body area, and 373.3 psi for the soles of the feet.

In either case, the same fall to a landing on the feet could have proved seriously disabling or fatal. Paradoxically, the smaller the area of initial contact in a fall or dive into water, the higher the plunge velocity that can be sustained. Snyder,[25] reporting on unimpeded falls of 39 males and 11 females ranging from 7 to 80 years of age, found that 116 ft/sec (79.1 mph) was the maximal velocity for surviving a feetfirst plunge with body perpendicular at water entry. Impacting the water with half the body area--whether front, back, or

TABLE 1
Analysis of survived human falls[a]

Sex	Age, years	Height in.	Height cm	Weight lb	Weight kg	Fall height ft	Fall height m	Impact speed ft/sec	Impact speed m/sec	Indent depth in.	Indent depth cm	Impact g[b]	Impact surface	Body position	Effects
F	42	62	157	125	56	55	17	54	16	4	10	135.8	Packed garden earth	Left side and back	Conscious, uninjured
F	21	67	170	115	52	93	28	73	22	6	15	165.5	Loose garden earth	Right side, back, and occiput; nearly supine	Fractured right wrist, conscious

[a] Derived from DeHaven.[5]
[b] Calculated from $\frac{V^2}{2gs}$

side first--presented approximately five times the contact area of a headfirst or feetfirst entry, correspondingly increasing the deceleration rate and the magnitude of impact against relatively incompressible water and lowering the velocity limit for survival. The highest survived velocities for specified body orientations at impact in this series were: prone, 88 ft/sec; supine, 93 ft/sec; lateral, 87 ft/sec; and buttocks first, 105 ft/sec. In one uninjured survival of feetfirst water entry at 97 ft/sec, absence of spinal injuries suggests a deceleration of less than 35 *g*. If it is assumed that maximal deceleration from 97 ft/sec was 35 *g*, then the deceleration distance, s, in water would be:

$$V^2/2gA = (97 \text{ ft/sec})^2/[(2)(32.2 \text{ ft/sec}^2)(35.0)] = 50 \text{ in.}$$

Thus, most of the kinetic energy would be dissipated in less than one body length from an entry velocity of 66 mph.

Snyder[24] describes 305 known free falls from the San Francisco Golden Gate Bridge from 1935 through August 1966, with only two survivors. Fall heights range from 239 ft (sea level at high tide from a low part of the span) to 261 ft (from the highest point on the bridge at low tide). Respective water-entry velocities from free fall would be 108 ft/sec (73.6 mph) and 111 ft/sec (75.7 mph). Between 1937 and 1966, the San Francisco Coroner's records include 169 autopsies of these fall victims.

Findings from these autopsies include: 129 cases of lung lacerations by fractured rib fragments, including 82 with lacerations of the heart and great vessels, and 91 cases of liver lacerations. There were 52 occurrences of rupture of one or more heart chambers and 38 of aortic rupture. These indicate water entry other than perpendicular to the long axis of the body. One 16-year-old boy plunged 244 ft from the Golden Gate Bride, hitting the water feetfirst at 72.8 mph. His only injuries were a midshaft fracture of the left clavicle, fractures of the fifth lumbar vertebral left transverse process, and minor retroperitoneal hemorrhages. No contusions of the body surface could be found at the time of rescue or during 3 days of hospitalization before he was sent home.

Tolerance of repeated exposures to water-collision impact has been demonstrated by professional exhibition divers of Acapulco, Mexico, who plunged headfirst 135 ft from cliffs into the ocean, hitting the water at an average speed of 58.5 mph. The most experienced of these divers performed this feat an estimated 26,000 times in 25 years. Schneider, Papo, and Alvarez[20] examined six of these divers and found x-ray evidence of old thoracic vertebral compression fractures in four of them. Three had involvement of the fifth thoracic vertebra, two had old fracture signs in the sixth, and one showed old healed fractures of the second, third, fourth, and seventh thoracic vertebrae. In addition,

healed fractures were found in metacarpals, radii, and ulnas. The only known death from attempting this feat was that of an American college amateur diver. This long record of repeated whole-body decelerations from 58.5 mph in a distance of less than one body length, with only occasional survivable and reversible injury, suggests an upper limit for exposure to whole-body impact at an injury threshold level, beyond which frequency and severity of injury would be expected to increase as a function of exposure increments.

Falls into snow from great heights have been survived. A celebrated example was RAF Flight Sergeant Nicholas Alkamade. About midnight on 23 March 1944, he jumped without a parachute from a burning bomber at 18,000 ft, free falling into pinetree branches in a German forest, which retarded his descent to a landing in 18 in. of snow. His only injuries were burns (incurred before the jump) and minor scratches and bruises. The incident was thoroughly investigated and documented by his German captors. Another case, in Alaska in February 1955, was reported by Kiel:[8]

> During one of the battalion drops, from 1,200 ft on a clear, relatively warm day, an observer noted what appeared to be an unsupported bundle falling from one of the C-119 airplanes; no chute deployed from the object. The impact looked like a mortar round exploding in the snow. When the aid men reached the spot, they found a young Negro paratrooper flat on his back at the bottom of a 3½ ft crater in the snow, which consisted of alternating layers of soft snow and frozen crust. He could talk and did not appear injured; nevertheless, he was air evacuated to a hospital. His only injuries were an incomplete fracture of a clavicle, a chip fracture of the second lumbar, and a few bruises. He was released from the hospital in time to return south with his unit.

During free fall, the human body attains a constant velocity--195 km/hr (121.3 mph) at 147 m (482 ft) above sea level--known as terminal velocity, when wind drag offsets gravitational acceleration. There is retardation from higher velocities attained in less dense air at considerable altitudes above this, so that final impact at sea level is at terminal free-fall velocity, whether from 18,000 or from 1200 ft. Thus, the paratrooper decelerated from 54.25 m/sec (178 ft/sec) in less than 1.065 m (3.5 ft) of crusty snow. His average deceleration can be calculated from:

$$V^2/2\ gs = (54.25\ \text{m/sec})^2/[(2)(9.8\ \text{m/sec}^2)(1.065\ \text{m})] = 141\ g.$$

There is a similarity between such whole-body free-fall impacts and the ground impacts at considerably lower velocities in football blocking and tackling.

MECHANICAL IMPEDANCE EFFECTS

The amplification of impact force by resonant response is a very important factor in the injurious deformation of viscous-elastic living tissues. Propagated deformation in compression and shear through a viscous-elastic mass proceeds at an interface with the remainder that is not yet in motion, storing energy that is expended in the restoration to the original shape of the mass. A pneumatic-elastic analogy would be the deformation of a football during a kick, as seen in high-speed motion pictures. Frame by frame, the foot can be seen indenting one side of the football while the other side stands still. As the indentation reaches a limit, for an instant all parts of the football move at the same rate as the foot; then expenditure of stored energy begins to return the ball to its original shape while reacting against the toe of the indenting foot. The other side of the ball accelerates faster than the foot by the added velocity of rebound. If the kick is delivered at a rate equal to or greater than the resonance of the football, the ball will spring away from the foot at a rate approaching twice the acceleration and four times the velocity of the kick. A partially inflated football that indents to a kick with no rebound would deform and thereafter travel no faster than the original peak acceleration of the foot permitted.

Relatively firm masses, such as the pelvis and the thorax, held together by the relatively elastic lumbar spine, can behave like two weights linked by a spring. A kick in the pants can telescope the lumbar spine and be followed by a rebound that amplifies the motion of the upper torso. The focal point of compression in the lumbar spine receives the pressure of the kick plus the reaction pressure of, in effect, a spring's being compressed. The combined loading at this point results in a higher probability of compression fracture at this location than at points above or below it. If the kick in the pants is delivered more slowly than the time required for elastic compression of the lumbar spine, the upper torso gets into motion before the lumbar "spring" can be significantly compressed, and the threshold for compression fracture is not exceeded. Amplification of impact force can be modulated in two ways: (1) by adjusting the rate of application of force, and (2) by interposing a deformable material that will change the rate of transmission of force to the body.

Ruff[18] investigated static and dynamic compression loading of the vertebral column in the longitudinal axis. Fresh cadaver vertebral-column segments were compressed in a static-test machine to find the breaking load on each of the lower five thoracic and all five lumbar vertebrae. He estimated a compression limit of 10 kg-m (about 870 in.-lb)

to fracture the first lumbar vertebra with loading applied to the whole spinal column. A stress-strain analysis of the vertebrae in the living spinal column was accomplished on a subject lying supine on an x-ray table with heels, hips, and shoulders supported on skateboards, with x-ray pictures taken at each compression increment of 10 kg (22 lb) applied between the head and feet, to record compression of intervertebral disks and reduction in space between vertebrae, from the fourth thoracic level to the fifth lumbar vertebra. In all, six such increments were applied, up to a total of 60 kg (132 lb), equivalent to the body weight of the subject. The reduction in intervertebral spaces with load increment as a function of strain was determined, and from it the percentage of body-weight loading in the upright position for each vertebra was calculated. The product of this percentage and gravities, equaling the static breaking load for each vertebra previously measured on fresh cadaver static tests, was supposed to equal the dynamic accelerative load range of tolerance for the vertebra. He obtained values ranging from 18.2 to 25.7 *g* from these calculations. Tests with living human subjects catapulted upward in ejection seats on a test tower resulted in lumbar spinal fractures in three of four subjects exposed to 26.0 *g* for 0.005 sec.

Latham[10] exposed human volunteers strapped tightly to a seat to sinusoidal oscillations of 2-20 Hz in the vertical axis. Body displacement at the iliac crest relative to that of the seat was amplified about twofold at 5 Hz, indicating whole-body resonance; shoulder displacement relative to the iliac crest was maximal at 8 Hz, indicating resonance between upper torso and pelvis-lower extremities through the lumbar "spring" coupling; and oscillations of the head relative to the shoulder attained resonance at 17-20 Hz. These frequencies correspond with impact pulse durations of 0.1, 0.063, and 0.025-0.029 sec, respectively. Dropping a subject in the seat 15.24 cm (6.0 in.) onto a concrete floor excited a fundamental resonance of 5 Hz, recorded by an accelerometer mounted on the crest of the ilium, adjacent to the center of gravity of the body. A sledgehammer striking upward against the bottom of the seat excited a resonant response of 8 Hz at the hip-to-shoulder linkage and 17-20 Hz in the shoulder-to-head coupling. Comparing the subjective sensations at each of the whole-body resonant frequencies, Latham considered the 5-Hz and 8-Hz exposures to be more severe than 17-20 Hz at similar input amplitudes.

Bierman[3] recorded standing waves in the abdominal and thoracic walls of 22 volunteers while they lay supine on a narrow table after a vest weighted at the corners was dropped from a height of 91.5 cm (3 ft) onto the chest and abdomen. The voluntary tolerance limit for this impact exposure was about 1500 kg (3300 lb) of force. No instrumentation other than high-speed motion-picture cameras and load cells (a type of strain gauge) was used. The subject, sandwiched between the table top and the weighted vest, sustained abrupt intra-abdominal and intrathoracic pressure increase, causing lateral deformation of the body cavities; the wave phenomena probably are confined to the body

wall, in contrast with the inertial excitation of both the internal masses and the body wall during abrupt velocity change of the whole body. Furthermore, the rigidity of bony articulations and the walls of the body cavities is determined by stiffening of opposing sets of muscles that surround the joints and body cavities. This can modulate the mechanical impedance characteristics of the whole body through a limited range of increase in resonant frequency, with stiffening, as demonstrated by Coermann[4] and Edwards and Lange.[6] They used animal and human subjects in various body postures and states of muscle tension on a shake table; increases of resonant frequency from 5 up to 8 Hz were correlated with muscle stiffening. Apart from the effect of muscle stiffening, Vogt *et al.*[34] found that volunteers seated on a shake table mounted to a centrifuge experienced an increase in resonant frequency and in subjective tolerance to low-frequency oscillations when head-to-foot centrifuge acceleration was applied simultaneously. A constant load of 2 *g* increased fundamental resonant frequency from 5 to 7 Hz while decreasing displacement of effective mass from 1.73 to 0.88 mm, for a decrease of 49%; likewise, at a constant load of 3 *g*, resonant frequency rose to 8 Hz and displacements diminished to 0.675 mm, with correspondingly lessened subjective discomfort. Vogt *et al.* found that up to 3 *g* of centrifuge accelerative force applying a steady load is a constraint to tissue deformation by the sinusoidal oscillations of the shake table, stiffening body response from a resonance of 5 Hz to 8 Hz, with a corresponding increase in tolerance to subjective sensations to the combined effect.

MUSCULAR TENSION EFFECTS

Morris *et al.*[15] describe 40 experiments with 10 healthy men in which the spinal column was supported with an "inflatable support" (pneumatic pressure with a closed glottis against the constriction of thoracic and abdominal-wall muscles) while each subject lifted a 200-lb weight from the floor with two hands and with his legs held straight. Intra-abdominal and intrathoracic pneumatic pressures were recorded, lever arms were measured for progressive displacements during the lift, and force vectors were calculated. The "inflatable support" contributed 600 lb, or 29%, of the 2071 lb exerted to lift the weight from the floor.

Under dynamic loading conditions of exposure to impact, the legs held straight and stiffened by muscle contraction with the feet against a 45-deg footrest supported up to 55.4% of the decelerative force, measured by a strain gauge in the footrest, while 25.6% of the load was applied to the seatbelt, and the remainder in friction against the seat, with the subject seated facing forward on a sled catapulted to a stop against a hydraulic piston brake. In 30 experiments on 15 volunteers at peak decelerations of 3.1-15.5 *g*, with strain gauges recording loads on all webbing restraints and against the footrest, Armstrong *et al.*[1] determined the distribution of forces with the legs held stiff and relaxed during an abrupt stop of the sled. The sum of

horizontal leg forces was as high as 364.8 kg (805 lb) during a sled deceleration peak of 12.4 *g*. At best, "propiotonic restraint" would be a fortuitous factor in an actual crash, depending on voluntary and reflex responses of a victim who was able to anticipate the collision.

Watt and Jones[35] report that organized leg-muscle antigravity activity during landing from a jump to the ground is normally brought about by a "packaged" or programed sequence of neuromuscular activity triggered well before the moment of impact. This denies reflex, or feedback, modulation of myotactic responses to the event. Human subjects whose electromyographic activity was recorded from electrodes over the body of the gastrocnemius before, during, and after a 38-cm (15-in.) jump onto a platform, instrumented to time the landing impact, demonstrated electromyographic activity about 180 msec before impact, increasing to a maximum and terminating well before myotactic reflex could have taken place. They postulate that constant-velocity parachute descent may cause difficulties in adjusting the phasing of neuromuscular organization that depends on constant acceleration of free fall.

Three modes of energy transmission to the whole body can produce effects ranging from subjective distress to injury, demonstrated in the previously described human accidental and experimental exposures to mechanical force: (1) abrupt pressure change and localized deformation resulting from inertial displacement of the body against restraints and from relative movement between supported and unsupported parts of the body; (2) excitation of interaction, by impact pulses matching or exceeding the body's resonant frequencies, between body masses through viscous-elastic linkages, and of organ masses through their ligamentous attachments; and (3) propagation of strain waves that, in compression, tension, or shear, can exceed the elastic limits of tissues at areas of maximal distortion.

These effects can be modulated by muscular stiffening and by constraint resulting from sustained accelerative force during exposure to impact or to oscillations in the range of whole-body resonances.

LOCALIZED IMPACT EFFECTS

The force applied per unit area is directly related to injury and survival limits. A 1000-lb blow applied to 1 $in.^2$ of the head can cause fracture and fatal concussion, whereas the same force applied to 200 $in.^2$ of the back would produce an innocuous 5-psi pressure rise. Anatomic and physiologic differences in vulnerability between particular body areas also determine injury thresholds and survivability. A crushing injury of the foot, requiring amputation, can be survived; a crushing injury of the head can be fatal. Decelerative forces that can be applied to the pelvic and shoulder girdles with impunity would deform the soft abdominal wall, with serious consequences. The more vulnerable ventral body areas can be guarded by flexing the trunk and neck and folding the limbs in the fetal position.

The design of protective equipment for football players--such as helmets, face guards, and shoulder, hip, and thigh pads--should take into account (1) frequency and severity of impact exposure and resulting injury rates at various anatomic areas, (2) methods of distributing and attenuating the force of a localized blow, and (3) avoiding injury to an opponent by impact against the device.

Neck

Simmons and Herting[23] calculated the forces incurred in American- and Japanese-style hanging executions. The traditional American 6-ft (1.83-m) drop is in excess of requirements for neck fractures, whereas the Japanese drop of 1.42 m (4 ft 8 in.) did not break the neck in two of three hangings witnessed by U. S. observers in the late 1940's. Assuming that, in these two cases of no neck fracture, the neck stretch was 2.54 cm (1 in.) at a body weight of about 68.2 kg (150 lb), the force at the end of a 1.42-m drop would be 944 kg (2080 lb). Estimated neck loadings for weights of 45.5 and 113.5 kg (100 and 250 lb) dropping 1.42 m in Japanese style would be 700 and 1430 kg (1540 and 3140 lb), respectively, and for the American-style 1.83-m drop, 980 and 1846 kg (2160 and 4060 lb), respectively. This would place their estimate of 944 kg for the two cases at a fracture threshold below the American minimum but within the overlap of the American-style and Japanese-style loading ranges.

Schneider *et al.*,[19] in analyzing head and neck motion and weight-bearing in the upright posture, describe the head and first two cervical vertebrae as a unit, designated as the "cervicocranium," bearing on the main neck column made up of replicating cervical vertebrae from the third to the seventh. According to Schneider *et al.*, the juncture of the cervicocranium with the third cervical vertebra is a site of mechanical weakness, where disruption occurs in judicial hanging and similar injuries. They describe normal weight-bearing in the upright posture of the head as symmetric loading by the two occipital condyles on either side of the lower skull opening against the bearing surfaces of the first cervical vertebra, along lines converging to a point where the body of the second cervical vertebra rests on that of the third. Shearing and bending forces applied in neck extension at this juncture of the cervicocranium with the third cervical vertebra tend to concentrate in the posterior arch of the second cervical vertebra at its weakest point, predisposing to rupture in a "hangman's fracture," however induced. Thus, this juncture becomes a fulcrum for disruption by simultaneous neck extension and head traction similar to hanging or to hooking the chin on the upper rim of a steering wheel in an automobile crash. Violent hyperextension and traction applied through the leverage of a football-helmet face guard are not without hazard in this regard.

Thorax and Abdomen

The effects of impact and deceleration on the thorax and abdomen have been described on the basis of accidental injury and within voluntary tolerance limits observed in experimental decelerations of the whole body, as well as by striking the chest and abdomen of the stationary body. Both types of experimental exposures have been applied incrementally to anesthetized animals through the range of injurious effects. High-speed motion-picture photography and cineradiography, as well as single and multiple-sequence still photography and x-rays, have been of great value in recording body displacements and relative internal-organ motion caused by impact and deceleration.

Of particular interest are the effects of impact on the heart and large arteries and veins in the thorax and abdomen. Several workers[2,16,21] have reported on lethal trauma produced when a nonpenetrating blunt object strikes the chest and abdomen and on the effects of transmitted force from whole-body deceleration in tearing and shearing the aorta and vena cava or rupturing atria and ventricles.

The relationship of the contractile state of the myocardium at the time of impact to the occurrence of cardiac injury was investigated by Life and Pince.[12] In a single cardiac cycle, the heart undergoes a sequence of profound changes in size, position, and configuration concurrently with electric, mechanical, and hydraulic changes associated with muscle contraction. To determine the relationship between applied impact to the chest and the contractile status of the heart in terms of the kind and severity of injury sustained, these authors dropped an 18.4-kg (40.5-lb) cyclindric weight 10.16 cm (4 in.) in diameter 1.98 m (78 in.) directly over the precordium of an anesthetized dog and measured the left ventricular pressure with a catheter introduced through the common carotid. Cyclic changes in left ventricular pressure, indicating the contractile status of the ventricular myocardium, provided a cue for phasing the drop of the weight. All 30 animals in this series were impacted at the end of the expiratory phase of the respiratory cycle as a control on pneumatic effects; the drop was timed with the selected stage of the cardiac cycle. The weight had a velocity at impact of 6.22 m/sec (20.4 ft/sec), and the kinetic energy transferred was 35.9 kg-m (about 260 ft-lb). Duration of the impact pulse was 50 msec, with a rise time of 20 msec at 1300 *g*/sec to a peak of 25.9 *g*. The equivalent loading was 473 kg (1040.7 lb), and the force:area ratio for the impacting end of the cylindric weight was 5.84 kg/cm^2 (83 psi). Regardless of whether impact occurred during systole or diastole, the left ventricular pressure rose to 427 $\pm$ 2.3 mm Hg.

The cardiac injury depended directly on the contractile state of the myocardium at impact. All animals impacted during either ventricular systole or diastole had ruptures of one or both atria; one or both ventricles were ruptured in animals impacted during ventricular systole, and none in animals impacted during ventricular diastole. Life and

Pince postulate that changes in the ventricle during systole predispose to damage when the heart is struck in this phase, and that rigidity of the myocardium during isometric contraction of the filled ventricle increases its frangibility relative to impact over the precordium. The relative incompressibility of the blood coupled with the relatively long opening response time of the pulmonary and aortic valves (compared with the brief duration of impact) may tend to produce an "anvil effect," in which the myocardium is caught between the "anvil" of the ventricular blood and the "hammer" of the inwardly deflected sternum. Whatever the mechanism of failure, impact during ventricular systole produces greater damage to the ventricular myocardium than impact during diastole.

It is postulated that aortic injuries in crash-injury deaths are due to pressure waves induced in vascular spaces, probably through the abdomen. Hansen[7] undertook to determine whether aortic injuries from impact deceleration were caused by tearing or shearing forces on the aortic arch due to abrupt displacement of the heart and diaphragm or were caused by increased intravascular pressure due to compression of thoracic vessels and the heart, raising hydrostatic pressures sufficiently to rupture the walls of the aorta and heart partially or completely. He exposed anesthetized beagle dogs to abrupt deceleration in the headfirst supine position to a peak of 20 *g* in a pulse of 300-msec duration. By means of a single-exposure flash x-ray, he filmed the heart once during a deceleration. By repeating the deceleration of the same subject at about 10-min intervals and phasing the x-ray exposure each time to a 25-msec increment in the approximately 300-msec pulse, he obtained a set of about 12 sequential single x-rays in as many impact exposures. From this composite of x-rays, the time-displacement curve of heart excursion during impact could be plotted approximately. The average displacement history suggested a sine-wave function with superimposed damping after one cycle, fully attenuated after rebound in 300 msec or less. The extent of displacements and the localization of tensions at the aortic arch near the origin of the left subclavian artery and insertion of the ligamentum arteriosum support the concept of shearing and tearing action. Two anesthetized chimpanzees decelerated supine and headfirst to more than 100 *g* sustained transverse rupture of the aorta, characteristic of tears.[31] Klotz and Simpson[9] produced ruptures of the aorta by static rise of hydraulic pressure to 3.16-4.57 kg/cm^2 (45-65 psi). These ruptures caused by static pressure were lengthwise of the vessel, whereas traumatic ruptures associated with deceleration and impact are transverse. Roberts *et al.*[17] compared aortic rupture in dogs produced by blunt impact against the sternum with blood circulating in eight subjects and following abrupt exsanguination through catheters implanted in the abdominal aorta and vena cava in six others. All animals were autopsied immediately after exposure. In both series, transverse tears occurred in the aorta with no difference in pathology whether blood was present in the great vessels or not. These authors believe that tears of the intima and media of the aorta and great vessels result from displacement of the heart during impact or deceleration, because:

(1) tears were localized anatomically, uniformly posterior or posterior and right lateral (pressure damage would have random distribution, in accordance with Pascal's law);

(2) they occurred in the absence of intravascular pressure, where blood had been drained abruptly before exposure to impact;

(3) aortic displacements were large enough to result in tears, and these experimental injuries were identical with those in human deceleration fatalities; and

(4) longitudinal tears characteristic of hydrostatic-pressure damage, such as were produced in experimental pressure elevation, were absent in the aorta and arteries and found only occasionally in veins.

Teare[33] described ruptured descending aortas in eight of 26 victims of a jet-aircraft crash; he attributed these ruptures to hyperflexion over the seatbelt. I reported a survived vertical crash from a height of 24.4 m (80 ft) by an Air Force free-balloon pilot, who sustained severe internal injuries; a year after the crash, the pilot underwent surgery for an aortic aneurysm, with replacement of the distal arch of the descending aorta by a Dacron prosthesis.[29]

Some areas and organs of the abdomen are anatomically vulnerable to direct impact. Widmann,[36] reporting on 119 cases of blunt abdominal trauma, of which 39 went to surgery, claims that the liver and spleen are less able to absorb energy under abrupt compression than are the hollow viscera, owing to limited mobility with respect to pedicles and proximity to the lower rib margins, against which they impinge during impact or deceleration. The spleen has an entirely arterial blood supply, so that hemorrhage tends to continue and recur, in contrast with the low-pressure portal venous supply to the liver, which favors rapid clotting. In his experience, Widmann estimates that one of every 450 trauma admissions has been for splenic rupture. Schneider *et al.*[21] warn that injuries resulting from seatbelt impingement in a car crash showing contusions of the abdominal wall may also include contusions of intra-abdominal viscera, leading to delayed perforation, scarring, hemorrhage, or intramural hematoma formation in the bowel. They cite case histories of five automobile-accident survivors in support of this observation, including one with a small bowel performation.

WHOLE-BODY IMPACT TOLERANCE

Abrupt-deceleration effects in whole-body exposure were observed and recorded on human volunteers and on anesthetized primates, bears, and swine relative to increments of velocity change in crash-simulation sleds and snubbed swings. Different body orientations and a variety of protective restraint systems were investigated in more than 3000 experiments (to date).

Lewis and I[11] determined tolerance limits for volunteers exposed to abrupt impact while seated and restrained in three different crash-force simulators:

(1) An aircraft seat suspended by cables to form a swing could be released from a predetermined height and arrested by an anchored steel cable in less than 5 in. (12.7 cm) after descending to the vertical position.

(2) A tubular steel sled was slipper-mounted between cylindric rails 250 ft (76 m) long on concrete piers 3 ft (91.5 cm) high; the center of gravity of the sled lay between the rails. Early experiments used gas-actuated ejection-seat catapults for propulsion; these were later replaced by a pneumatic-hydraulic catapult with a 43-ft (13-m) stroke piston capable of velocities up to 175 ft/sec (53.4 m/sec). A solid steel piston 6 in. (15.2 cm) in diameter and 4½ ft (1.4 m) long extending from the prow of the sled provided water-inertia braking by entering a corresponding cylinder in a steel block mounted between the rails at a chosen distance from the start of the run. The cylinder contained water held in by a plastic diaphragm, which was ruptured by entry of the piston, ramming water out of 135 holes through the upper half of the cylinder wall. Threaded chokes or plugs determined the cylinder leak rate and programed sled retardation by water inertia. This device was named the "Daisy track" (after the well-known toy air-guns). Several sleds were developed for the Daisy track, of one- to three-seat capacity, with provisions for a variety of pitch, roll, and yaw orientations.

(3) A portable 24-in.-gauge track 25 ft (7.6 m) long had a slipper-mounted platform that held a seat adjustable to face forward or rearward. It could be propelled by a rubber bungee slingshot to speeds of 25 ft/sec (7.6 m/sec) in 18 ft (5.5 m) to enter spring-loaded clasp-type friction brakes pinching the 2-ft (7.3-m) central keel attached lengthwise beneath the platform. These brakes could stop the sled in less than 6 in. (15.2 cm) with deceleration up to 28 *g* at onset as high as 1200 *g*/sec. This device was suitable for both experiments and demonstrations with human and dummy subjects. It was named the "Bopper."

Typical results with the three devices are shown in Table 2.

I found that, for a constant magnitude of decelerative force, the physiologic effect correlated with rate of onset for durations not exceeding 0.4 sec.[31] When a volunteer, seated and restrained with webbing straps on a rocket sled on rails, was exposed to 38.6 *g* in 117 msec, facing forward, recordings of the chest-mounted accelerometer and the reference accelerometer under the seat corresponded closely in onset and magnitude. The dynamic load of 7980 lb (3627 kg) against 280 in.^{2} (1806 cm^2) of webbing left a slight reddening of local pressure areas on the shoulders and hips. Physiologic reactions corresponded to those after moderate exercise (run 214, Table 3). An exposure to the same 38.6 *g* with onset in 28 msec against the same

TABLE 2
Comparison of swing, Daisy, and Bopper performance (all human subjects, facing forward, restrained by seatbelt only)

Simulator	Speed		Stopping distance		Deceleration			Body weight		Force on belt		Load area		Pressure		Injurious effects
	Duration, sec	Onset, g/sec	Peak, g	lb	kg	lb	kg	$in.^2$	cm^2	psi	kg/cm^2					
	ft/sec	m/sec	in.	cm												
Swing, man	15.4	4.7	5	12.7	0.08	570	23.0	156	71	3588	1631	48.0	309.6	74.7	5.3	None
Swing, seat	15.4	4.7	5	12.7	0.04	1500	30.0	--	--	--	--	--	--	--	--	--
Daisy, man	29.0	8.8	14¼	36.2	0.07	500	17.0	130	59	2210	1005	42.0	270.9	52.6	3.7	None
Daisy, seat	29.0	8.8	14¼	36.2	0.07	544	18.7	--	--	--	--	--	--	--	--	--
Bopper, man	19.0	5.8	5½	14.0	0.62	900	28.0	165	75	4290	1950	48.0	309.6	89.5	6.3	Back strain
Bopper, seat	19.0	5.8	5½	14.0	0.54	740	20.0	--	--	--	--	--	--	--	--	--

type of restraints resulted in neurocirculatory shock with blood pressure below 50 mm Hg and loss of consciousness for about 60 sec, shortly after the sled stopped. Two days of partial bedrest were needed for recuperation, during which black and blue contusions with local muscle soreness developed at strap impingement areas on shoulders and hips (run 133, Table 3). Even more severe reactions occurred after exposure in the backward-facing seated position to a peak of 40.4 *g* attained in 19 msec, with resulting lag followed by amplification to 82.6 *g* at 3826 *g*/sec onset, recorded on the chest-mounted accelerometer. The subject had severe back pain and cardiovascular shock with less than measurable blood pressure and loss of consciousness of 1 min with brief recovery and relapse of about the same duration as he was being evacuated from the sled to a hospital, where he was in bed sedated and under traction for the first day of a 3-day admission. Recuperation required a total of 5 days. Even in the less vulnerable, backward-facing seated position, the correlation between increased abruptness of onset and greater severity of physiologic stress is evident. The twofold amplification of recording from the chest-mounted accelerometer compared with that of the seat reference accelerometer indicates excitation of resonant response in the ribcage and sternum, deforming inward and rebounding, with unmeasured, corresponding displacements and deformations of viscous-elastic and pneumatic tissues of the thoracic contents.

The results of the human experiments described are applicable primarily to aircraft and automotive crash exposure and evaluation of protective restraint systems, including various configurations of webbing straps for seated subjects facing forward and bulkhead or seatback restraint for seated subjects facing backward. Nevertheless, these experimental impacts and their physiologic effects resemble the more severe types of body contact and ground collision effects encountered in football blocking and tackling.

Another approach to protection from the effects of whole-body exposure to impact is described by Lombard *et al.*[13,14] as isovolumetric containment. It consists of enclosing the torso in a semirigid, padded plastic container that, in addition to distributing forces uniformly against presenting surfaces, maintains posture, restricts deformation of the body wall, and confines displacements of internal organs and tissues. Survival limits of anesthetized small primates and of guinea pigs markedly increased by isovolumetric containment, compared with harness systems restraining the torso by holding the pelvic and pectoral girdles. Devices for isovolumetric containment ranged from a "sleeping bag" of webbing or fabric, tightened against a back support with head and neck fixed, to a rigid mold of plaster of Paris lined with energy-absorbing padding in a snug cocoon around the animal subject. The optimal orientation and containment permitted survival up to 400 *g* at 200,000 *g*/sec produced by impact of a table-top slingshot-propelled miniature sled on rails into a barrier of aluminum honeycomb metal, which was partially crushed to stop the sled in 0.50 in. (1.3 cm).

TABLE 3
Human exposures to whole-body crash impact

Run	Subject	Age, years	Velocity change ft/sec	Velocity change m/sec	Deceleration distance ft	Deceleration distance m	Duration, sec	Onset,[a] g/sec	Peak g	Weight lb	Weight kg	Force (f=ma) lb	Force (f=ma) kg	Restraint area loading in.^{2}	Restraint area loading psi
(1)	Subject seated faci g forward Restrai ts: Nylon webbing shoulder straps, belt, tie down straps from belt to seat pan.														
133	RL	30	213 - 87 = 126.0	65.0 - 26.5 = 38.5	24.6	7.5	0.155	1370	38.6	177	80	6832	3105	238	28.7
135	JPS	39	220 - 105.5 = 114.5	67.1 - 32.2 = 34.9	24.6	7.5	0.160	1344	38.1	172	78	6553	2979	217.5	30.2
214	PWS	39	222.5 - 41.0= 181.5	67.9 - 12.5 = 55.4	36.4	11.1	0.283	331	38.6	206	94	7952	3615	280	28.4
215	JPS	39	226 - 50.2 = 175.8	68.9 - 15.3 = 53.6	31.2	9.5	0.228	493.5	45.4	175	80	7945	3611	219.5	36.5
(2)	Subject seated facing backward Restraints: Seatbelt only														
113	WAR	29	206 - 90 = 116	62.8 - 27.5 = 35.3	24.6	7.5	0.160	1156	35.0	152	69	5320	2418	260	20.5
114	JFF	30	208 - 96 = 112	63.4 - 29.3 = 34.1	24.6	7.5	0.160	1160	34.8	153	70	5324	2420	252	21.1
335	ELB	32	44.3 to 0	13.5 to 0	1.24	0.38	0.065	2136	40.4	130	59	5252	2387	205	25.6
Measured on subject ELB's chest:								3826	82.6						

[a] Onset and acceleration calculated from brake-performance data.

Effectiveness of isovolumetric restraints for larger primates was demonstrated by Sonntag *et al.*,[28] with results shown in Table 4. Two experiments were done with anesthetized chimpanzees seated facing forward with legs extended, held in a contoured mold of energy-absorbing foam plastic between an aluminum shield with a facial opening and a plywood backboard, mounted on the Daisy sled. The sled was decelerated from a little less than 90 mph in 28 in. (about 40 m/sec in about 71 cm), attaining just under 150 *g* at a little less than 13,000 *g*/sec in approximately 16.0 msec. Other than muscular bruising and small abrasions to the malar eminences, neither of the chimpanzees sustained any significant injury. The calculated impact forces were 8100 and 7460 lb (about 3700 and 3400 kg) at peak *g*, sufficient to buckle the 1/8-in. (0.3-cm) aluminum shield about 3 in. (7.6 cm) outward. Factors contributing to uninjured survival were: (1) rigid, sandwich-board alignment of head, neck, and trunk; (2) containment of the front body wall and restriction of displacement of internal organs by compression against the 7-in. (17.8 cm) of foam plastic; and (3) impact-load distribution over most of the available presenting body area. Body-cast containment and damping by energy-absorbing padding offer the ultimate achievement to date in crash-impact protection.

DISCUSSION

Biodynamics research observations and experiments to date relate primarily to aircraft and vehicle crash survival. Military priorities have justified hazardous experiments with human volunteers and anesthetized animal subjects to accomplish the medical corps mission of conserving the fighting strength. No similar experiments have been carried out with complete optical and electronic instrumentation to determine impact forces and tolerance limits by exposure to measured increments of impact in football body and ground contact collision situations, although miniaturized telemetry in football helmets has been used to record forces sustained in actual football games, as described by Dr. Reid elsewhere in these proceedings.

The following estimate of forces encountered in a collision between two football players is submitted to suggest orders of magnitude being dealt with: Assume that two football players, each weighing 200 lb and running on a collision course at 30 ft/sec (100 yard dash in 10 sec), ram together at midfield, mutually indenting about 3 in. before falling apart. Each player then sustains:

$$\text{acceleration} = V^2/2gs = (30 \text{ ft/sec})^2/[(2)(32.2)(\tfrac{1}{4} \text{ ft})] = 56\ g.$$

$$\text{Force} = ma = (200 \text{ lb})\ \ (56\ g) = 11{,}200 \text{ lb}$$

Assuming a collision contact area of 400 in.^{2} for each player, the average impact load would be (11,200 lb)/(400 in.^{2}) = 28 psi. Two

TABLE 4
Isovolumetric crash restraints
(anesthetized chimpanzees)

Run	Weight lb	Weight kg	Velocity change ft/sec	Velocity change mph	Velocity change m/sec	Stopping distance in.	Stopping distance cm	Peak *g*	Onset, *g*/sec	Duration, msec	Force (f=ma) lb	Force (f=ma) kg	Restraint
3849	55	25	131.5	89.7	40.1	28	71.1	147.5	12,900	16.0	8100	3682	Polyurethane
3850	50	22.3	130.0	88.6	39.7	28	71.1	148.8	12,200	15.3	7460	3391	Polyvinyl

players in good training would perhaps be momentarily stunned by this encounter, but would probably pick themselves up and recover in time to take part in the next play. Such collisions and buffetings are repeatedly endured and recuperated from during a game, with some help from helmets, face plates, shoulder pads, hip pads, and thigh pads. Quantitative data on human tolerance, on evaluation of whole-body crash protection, and on the correlation of physiologic response to mechanical stress, even though primarily related to aviation and automotive crash requirements, can nevertheless be useful in criteria for design and performance standards of football protective equipment until more specific data can be obtained from controlled, adequately instrumented experiments with football players reproducing in simulation the impacts they experience while playing football. Projecting from the improvement that has been achieved in helmets through design and performance standards based on quantitative head-impact research, as reported by other writers in these proceedings, there could be justification for more exact research on whole-body impact protection in this violent body-contact sport, in which injury is a serious occupational and health hazard.

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ENERGY-ABSORPTION TOLERANCE OF THE EXTREMITIES AND PELVIS

F. GAYNOR EVANS

Anyone who has ever seen an American football game, either in person or on television, knows that it is a rough contact game in which the chances of being injured are greater than in most other sports. Football fans are also aware that the extremities, in general, and the knees, in particular, are commonly involved in football injuries. However, the type, the frequency, and the magnitude of injuries to the extremities may not be generally known.

An analysis of injuries occurring in 465 Air Force football players during 34 games in the 1965-1966 season[1] revealed a total of 290 injuries, of which 111 (38%) were classified as major. Of the 290 injuries, 38 (13%) resulted in hospitalization, 22 (8%) resulted in surgery, and 65 (22%) kept the victim out for the season.

Sprains were the most common injuries, accounting for 45 (41%) of the major injuries and 80 (28%) of the total injuries. Sprains resulted in 17 (45%) of the hospitalizations, 13 (59%) of the operations, and 33 (51%) players' being out for the season. The knee and ankle were involved in 87% of the major sprains and 35% of the major injuries.

Fractures accounted for 18 (6%) of the total and 15 (14%) of the major injuries. The ankle and the forearm-wrist-hand area were the most common sites of fractures. Dislocations accounted for 10 (3%) of the total and nine (8%) of the major injuries. The most common sites of the dislocations were the shoulder joint (glenohumeral), the forearm-wrist-hand area, and the ankle.

Contusions, which were found in all parts of the body, accounted for 86 (30%) of the total injuries and 11 (10%) of the major injuries. Strains of the musculotendinous unit occurred in 42 (14%) of the total and in eight (7%) of the major injuries. Lacerations and abrasions accounted for 16 (6%) of the total injuries, but none of the major.

Injuries not included in the above categories accounted for 38 (13%) of the total and 23 (21%) of the major injuries. These consisted of traumatic arthritis, boutonniere deformity, bursitis, brachial plexus stretching, tendon dislocations, and pneumothorax.

According to Thorndike,[25] from records between 1932 and 1959 in the medical room of the Dillon Field House at Soldiers Field, Chicago, the injuries severe enough to require an athlete to miss one or more sessions of practice or games were, in order of decreasing frequency, sprains, muscular contusions, strains, fractures and dislocations, simple contusions, inflammations and infections, joint contusions, lacerations and abrasions, and internal injuries. Of the 8293 injuries recorded during that period, 543 were classed as "miscellaneous."

Thorndike also pointed out that the greatest injury expectancy is with the fall sports, the most highly organized and supervised of any seasonal sports program. The Eastwood report on football fatalities is cited to the effect that, during the 29-year period covered, football fatalities averaged 17.24 per year. However, the incidence per 100,000 players was only 1.52 for high schools and 2.20 for colleges.

A statistical analysis, cited by Ferguson and Bender,[8] of injuries of 920 athletes at a prominent college from 1950 to 1955 revealed that 48.7% of them were severe enough to disqualify the athlete for one or more participations in all sports. Ten of the injuries and one illness prevented all further participation in athletics. Of the injuries, nine occurred in football. Analysis of the injuries with respect to the part of the body involved revealed that the knees (44 cases) were the most frequently injured, followed by the ankles (34 cases), the head (10 cases), and the neck (eight cases).

An analysis made by Hibbert[11] in four selected areas--one in the Pacific Northwest, one in the Rocky Mountains, one in the Southwest, and one in the South--of 46,824 high-school athletes during the 1948-1949 school year indicated that about one of every five participants was injured, with fatalities occurring in approximately one of every 25,000 participants. Football accounted for 86.3% of the injuries. Of the injuries, 32% were bruises, 20.6% were sprains, and 18.1% were fractures. In the 1947-1948 season, sprains constituted 34%, fractures 30%, and bruises 19% of the injuries. The sprains occurred, in descending order of frequency, in ankles, knees, back, shoulders, and wrists. The corresponding order of frequency for fractures was clavicles, legs, fingers, forearms, and nose. The highest incidence of injury (38.5%) occurred at 17 years of age, followed by 28.7% at 16 and 25.5% at less than 16 years of age. From 18 to 20 years of age, the incidence of injury was less than 6%. These data indicate that the frequency of injury decreases with the age (and experience) of the athlete.

When considering skeletal injuries of the extremities in athletes of high-school age or younger, it should be remembered that the epiphyses of some of the long bones may not fuse with the shaft of the bone until the age of 20 or later (Fig. 1). This may account for epiphyseal injuries in young people, although Hale, Research Director of Little League Baseball, reported no injuries of this type in 771,810 boys,[8] probably because baseball is not primarily a contact sport (the greatest number of injuries were caused by getting hit with a pitched ball).

Furthermore, Little League players are usually small and therefore collisions between them are less likely to cause severe injuries than are collisions among high-school, college, or professional football players.

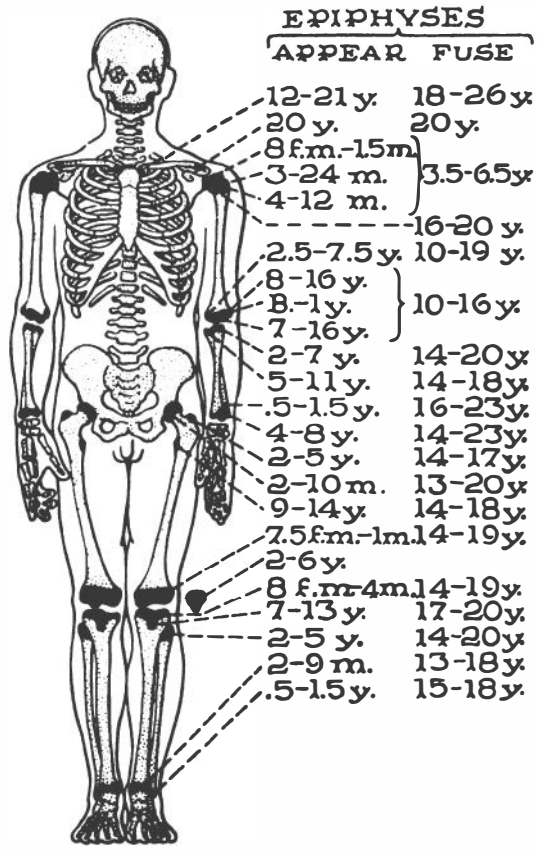

FIGURE 1
Time of appearance of epiphyses and their fusion with the shaft of human long bones. (Reprinted with permission from Morris.[21])

The response of a body, including the human one, to the application of a force is a function of several factors, both mechanical and biologic. Among the mechanical factors to be considered are the type and the magnitude of the force; the point, direction, speed, and duration of application of the force to the body; and the size and shape of the body and mechanical properties of the material composing the body.

Under the same loading conditions, a large body can support a greater force before failing than can a small body of the same shape and composed of the same kind of material because of differences in the amount of material in the two bodies. However, a steel body tolerates a greater force before breaking than does a wooden body of exactly the same shape and dimensions because of differences in the mechanical properties of steel and of wood. Furthermore, a body composed of more than one kind of material may tolerate a greater force before failing than a body composed of either one of the materials alone.

The human extremities and pelvis are bodies consisting of a variety of different materials (tissues), and their tolerance to the application of a force is influenced by differences in the mechanical properties of these materials. Such biologic factors as age, sex, physical condition, and race also affect the tolerance of the extremities and pelvis.

Of the various tissues composing the adult human body, muscle forms the largest proportion (43%) of the total body weight (Fig. 2). The same is probably true for the extremities and the pelvis in particular, although no actual figures pertaining to this were found in the available literature. The extremities form a larger proportion of the total adult body mass than is generally realized. Dempster[2] found that the upper extremities of eight adult human male cadavers constituted 9.7%, the lower extremities 31.4%, and the shoulders 10.3% of the total body weight (the proportional weight of the pelvis was not determined). Thus, the extremities, including the shoulders, account for about half (51.4%) the total adult body weight.

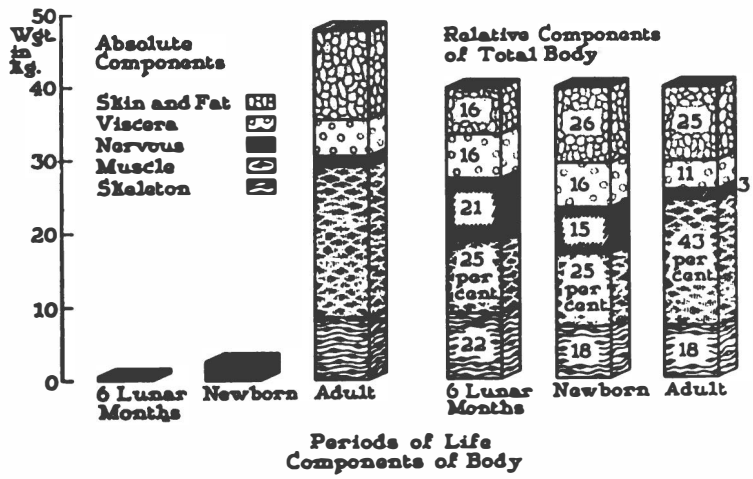

FIGURE 2
Age differences in the absolute and relative components of the human body. (Reprinted with permission from Wilmer.[26])

Lissner and I[15] have pointed out that all injuries result from the absorption of energy that occurs when a part of the body comes into contact with another body or object moving at a different velocity. Such a situation can occur under three conditions: (1) a part of the body at rest but free to move, such as the head or an extremity, can be struck by a moving object and accelerated; (2) a moving part of the body can strike a resting object and be decelerated; or (3) a moving part of the body can strike or be struck by a moving object, in which case the part of the body is accelerated or decelerated, depending on which is moving faster (the part of the body or the object) and on the relative masses and direction of each. The injury to the body part will be identical in all three conditions if the velocity difference between the body part and the other object is the same and if the location of the blow on the body part is the same.

Because most football injuries arise from blows or impacts, the capacity of the various components of the extremities and other parts of the body to absorb energy is one of the most important mechanical properties to be considered. It is therefore surprising to find little information in the literature on the energy-absorbing capacity of various parts and tissues of the body.

The first investigation, as far as I am aware, of the relationship between trauma and energy was by Gurdjian and Lissner,[9] who produced deformation and fracture of dry human skulls by the application of various amounts of energy to different parts of the skull. Most of the tests were made with 14.3 in.-lb of energy, although in other tests the energy level varied from 8 to 18 in.-lb. In a later study,[10] intact heads of adult human cadavers were tested with energies varying from 400 to 907 in.-lb and impact velocities of 13.5-22.8 ft/sec (4.1-7 m/sec). The lowest amount of energy required to produce a single linear fracture was about 400 in.-lb. Great variation in the energy required to produce a single linear fracture was seen from one head to another, owing to differences in scalp and skull thickness, variations in skull shape, and slight changes in the site of the blow to the head. The fact that a single linear fracture could be produced in a dry skull with as little as 40 in.-lb, compared with the 400 in.-lb required for the intact head, suggests that the scalp and soft tissues covering the skull form an excellent energy-absorbing material.

Concerning the relationships of energy, velocity, and acceleration to head injury, it has been emphasized[6] that not only the magnitude of the energy involved is important, but also its rate of absorption. Thus, the longer the time during which the energy is absorbed, the greater the magnitude of the energy that can be safely tolerated. The magnitude of the energy and the total duration for it to be absorbed were accurately measured. The energy varied from 286 to 581 ft-lb, and the time for absorption of the energy, from 0.0019 to 0.0117 sec.

The energy-absorbing capacity, under bending, of fresh humeri, femora, and tibias has been investigated by Mather.[16] Paired femora of 28 subjects, the humerus and the femur of 12, and the tibia and the femur of 16 were tested. The results of the study (Fig. 3) showed that the femur absorbed more energy than the humerus, the tibia more than the femur, and the left femur more than the right. Correlations between the energy-absorbing capacities of specimens in each group of subjects were significant at the 5% level or better, except for the tibia and the femur of the same subject, which showed no significant differences in their energy-absorbing capacity.

In a second study, Mather[17] determined the energy required to fracture the tibia of an intact human leg when loaded like a simple beam. Legs from 107 men and 52 women, ranging in age from the teens to the 90's, were tested. None had died from physical violence. Mather found, for the legs of males matched with an equal number of randomly selected females, that 7-8 kg-m of energy had to be applied to fracture half the tibias (Fig. 4). The difference between the load-carring

capacity of the tibia of a younger subject (less than 65 years old) was significantly greater, at the 1% level, than that of the tibia of an older subject.

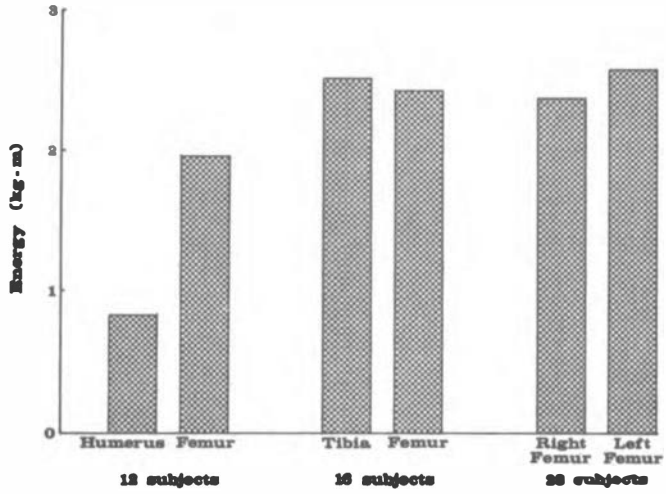

FIGURE 3
Comparison of the energy-absorbing capacity in bending of fresh human long bones. Based on data from Mather.[16]

Mather[18] has also investigated the energy-absorbing capacity of intact unembalmed adult human femora subjected to static and dynamic bending. One member of each of 32 pairs of femora was tested under static bending in a Mohr and Federhall materials-testing machine, using a capacity range scale of 500-10,000 kg. The deflection of the center of the bone was measured with a dial gauge, calibrated in units of 0.025 mm, attached to the test bed of the machine. The energy-absorbing capacity of the other member of each pair of femora was determined under impact loading in drop-weight tests in which a striker was dropped from a known height onto the center of the shaft of the bone. The velocity of the tuppet was 32 ft/sec (22 mph or 35 km/hr). The mean energy required to fracture the femur by static loading was 20.5 ft-lb, compared with a mean of 31.3 ft-lb in the impact tests. Mather took care to point out that impact tests, from the engineering viewpoint, are less reliable than static tests for determining the mechanical properties of a material, because structures are more sensitive to the stress-raising effects of flaws in the material under impact loading.

Tolerance of the extremities and pelvis to forces must also be considered in terms of the energy-absorbing capacity of their various tissue components. Although there is an extensive literature on the stress-strain characteristics and the modulus of elasticity of various tissues, a search of the available literature uncovered only one publication in which the energy-absorbing capacity of a tissue is mentioned: Lebow and I[4] compared the energy absorbed by wet- and dry-tested specimens of cortical bone from the embalmed femur of a 78-year-old white

man. The dry specimens absorbed an average of 69.0 in.-lb/in.^{3} of energy, compared with an average of 83.6 in.-lb/in.^{3} for the same number of wet specimens.

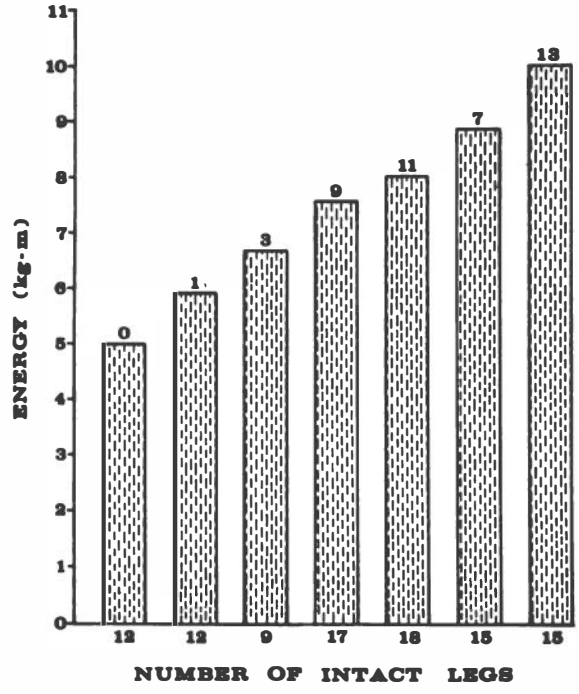

FIGURE 4
Energy required to fracture tibias of intact human legs when loaded like simple beams. Based on data from Mather.[17]

The average energy (kg-cm/cm^3) absorbed to failure by different human tissues was determined by measuring the area under the stress-strain curves for various tissues. The stress-strain curves, from which the energy was calculated, were for tension tests of standard specimens of bone and skin, strands of fibers of tendon and nerve, small pieces of tissue from the walls of arteries and veins, and a few muscle bundles. None of the test specimens was a cross section of an intact bone, tendon, nerve, blood vessel, or muscle. All the energy-absorbing data were calculated from stress-strain curves published by researchers working in the laboratory of Professor Hiroshi Yamada, Chairman of the Department of Anatomy at the Kyoto Prefectural University of Medicine.[12-14,20,22,24,28] From Fig. 5 it is evident that tendinous tissue has the greatest and skeletal-muscle tissue the lowest average energy-absorbing capacity. However, the energy absorbed by all the soft tissues combined (such absorption occurs in many parts of the body when a blow is applied to it) is more than twice that absorbed by bone.

Whenever a blow is applied to a part of the body, the skin and other soft tissues covering a bone absorb some of the energy of the blow before it reaches the bone. Even such a relatively thin layer of soft tissue as the scalp, as demonstrated by Gurdjian *et al.*,[10] is a safety factor, as far as skull fracture is concerned.

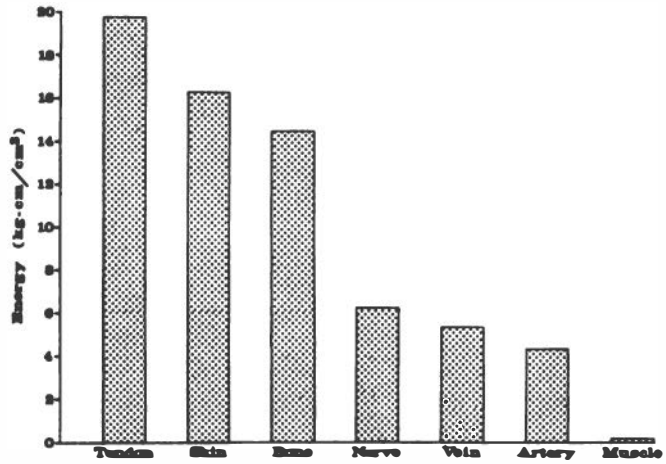

FIGURE 5
Average energy absorbed to failure by various human tissues. Based on measurements of the area under stress-strain curves published by various investigators.

The relative amounts of soft *vs*. hard tissues vary continuously along the extremities, as can easily be seen from examination of serial cross sections. In the proximal segments of the extremities (thigh and upper arm), the bone is surrounded by large muscles that, with other soft tissues, absorb much of the energy of a blow. In the proximal half of the middle segment (leg and forearm), the muscular tissue still predominates, although the muscular tissue is gradually replaced by tendons in the distal half of the segment. At the level of the wrist, there is more hard tissue (bone) than soft tissue. A somewhat similar change occurs in the leg as the ankle region is approached, although the relative reduction in the amount of muscle tissue is less than in the wrist region.

The most common injuries of the hand and foot are ankle and wrist sprains and digital fractures. In all three regions (especially the wrist), there is little soft tissue, compared with the amount of bone. However, the wrist and ankle bones consist primarily of spongy bone surrounded by a thin shell of compact bone. According to Policard and Roche,[23] the human ankle and heel bones contain 80% nonosseous tissues.

Spongy bone is structurally a foam, somewhat similar to styrofoam, and is thus a good energy-absorbing material.[7] The energy-absorbing capacity of spongy bone was experimentally demonstrated by Dr. Physick (cited by Wistar[27] in 1827) with a row of ivory balls. If the first ball in the row was allowed to impact the others, the concussive force between the balls was sufficient to impel the last ball of the row a distance approximately the same as that through which the first ball traveled before striking the others. However, when the first ball in the struck row was replaced by a ball of spongy bone of the same size and the experiment repeated, the momentum of the striking ball was entirely absorbed by the spongy ball, and the last ball of the row was

not moved. Dr. Physick also pointed out that the energy-absorbing capacity of the ball of spongy bone was greatly enhanced if it was wet.

Although muscle tissue itself has relatively little energy-absorbing capacity, the muscles of the proximal parts of the extremities, because of their size, absorb much of the energy of a blow to these regions. The low energy-absorbing capacity of muscle tissue may explain the frequency of the charley horse type of injury seen in different sports.

Let us consider next dislocations, sprains, and other types of joint injuries, especially those concerning the knee and the ankle. The stability of a joint depends on three anatomic factors: (1) configuration of the joint surfaces of the bones, (2) joint capsule and ligaments surrounding the joint, and (3) tendons of muscles crossing the joint. The relative importance of these structures varies with the joint and with the degree of freedom of movements at the joint. No joint can have maximal stability and maximal mobility simultaneously; there must always be some compromise.

The shoulder has the greatest freedom of movement of any joint, but it is also the most often dislocated. The anatomic stabilizing factors of the shoulder joint are, in order of importance, the muscle tendons, the configuration of the joint surfaces (humerus head in glenoid fossa), and the joint capsule and ligaments. In contrast, the knee is more stable but has less freedom of motion. The structures responsible for its integrity are, in order of importance, the tendons of the large muscles crossing the joint, the joint ligaments, and the articular surfaces of the bones forming the joint. Regardless of the anatomic stability of the knee, it is often injured in football and responsible for incapacitating players. In spite of the frequency of joint injuries, no data on the energy-absorbing capacity or other mechanical properties of human joint ligaments and capsules were found in the available literature.

We have studied pelvic deformations and fractures with the Stresscoat* method.[5] The Stresscoat pattern consists of parallel cracks in a special

* "Stresscoat" is the trade name for a strain-sensitive lacquer that is used in industry to determine areas of high tensile strain, where failure is likely to occur in machine parts. The structure to be tested is first given a white or black undercoating, over which the Stresscoat is sprayed. On the next day, the specimen is covered with a wetting agent and then tested under static or dynamic conditions. After the test, the specimen is sprayed with Statiflux powder, which fills the cracks in the Stresscoat, so that they can be seen. Because the powder does not change the sensitivity of the lacquer, the test can be repeated if no Stresscoat pattern is obtained.

lacquer. The cracks arise only from tensile strain in the underlying material and are oriented transversely to the direction of the strain. The cracks appear first at the site of highest tensile strain, where failure will occur if sufficient force is applied. The lacquer sensitivity is calibrated in inches/inch. For example, a sensitivity of 0.0007 means that every time the underlying material stretches (tensile strain) 0.0007 in., the overlying lacquer will crack.

A pelvis was weighed and Stresscoated. It was then suspended over a 140-lb steel block with a string and dropped onto the block by burning the string. The specimen was dropped so that the ischial tuberosities struck the block simultaneously; it was caught on the rebound, so that it hit the block only once. The weight of the specimen multiplied by the distance through which it was dropped yielded the energy (in inch-pounds) dynamically applied to the ischial tuberosities.

We tested 22 specimens of lumbar spines and pelves.[5] Sixteen embalmed specimens were tested with 33-112 in.-lb, and six unembalmed specimens, with 39-90 in.-lb of energy. No fractures were produced.

In a second series of cadavers, the lower extremities and pelvic musculature were removed, the bony pelvis Stresscoated, and 200-450 in.-lb dynamically applied to the ischial tuberosities. A fracture of the right ischium of one pelvis was produced with 240 in.-lb of energy. One specimen with about 0.5 in. (1.3 cm) of soft tissue under each ischial tuberosity showed only a threshold tensile strain (Stresscoat) pattern after application of 450 in.-lb of energy, the maximum used in any test. This demonstrates again the good energy-absorbing capacity of soft tissue.

An example of an extensive Stresscoat pattern obtained by dynamically loading the ischial tuberosities is seen in Figs. 6 and 7. The cracks constituting the Stresscoat pattern arose from tensile strain created in the underlying bone by displacements and oscillations of various parts of the pelvis (Fig. 8). Lateral and medial oscillations of the region of the anterior-superior iliac spine caused bending of the iliac ala, so that opposite sides of the same region were alternately subjected to tensile and compressive strain. The Stresscoat cracks arose when the side was convex and undergoing tensile strain.

Of particular interest in the general field of trauma is the demonstration by McElhaney and Byars[19] that the energy-absorbing capacity of cortical bone (embalmed human femur) decreases steadily as the strain rate of loading changes from 1 to 1500 in./in.-sec. This is of special significance, because most traumatic injuries arise from impact.

The energy absorbed to failure under static loading by small specimens of spongy bone from different parts of embalmed human femora has been determined.[3] However, the energy-absorbing values obtained would probably be different with impact loads.

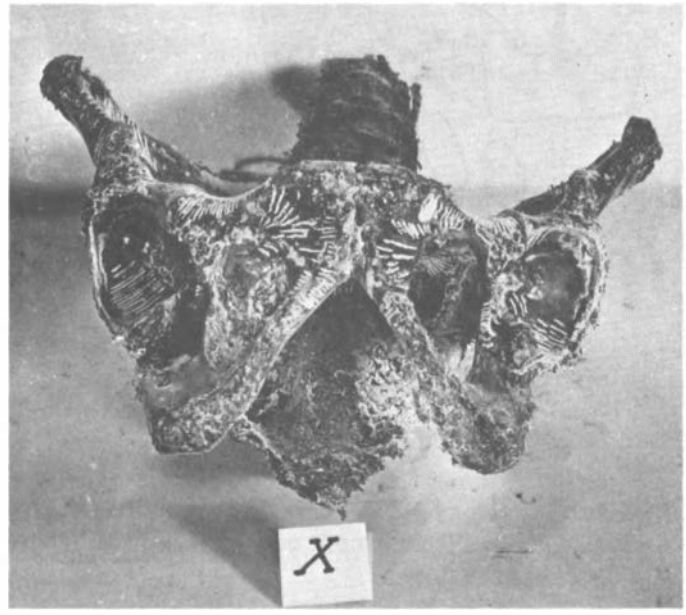

FIGURE 6
An extensive tensile-strain pattern produced in the embalmed Stress-coated pelvis of a white man by application of 67 in.-lb of energy to the ischial tuberosities.

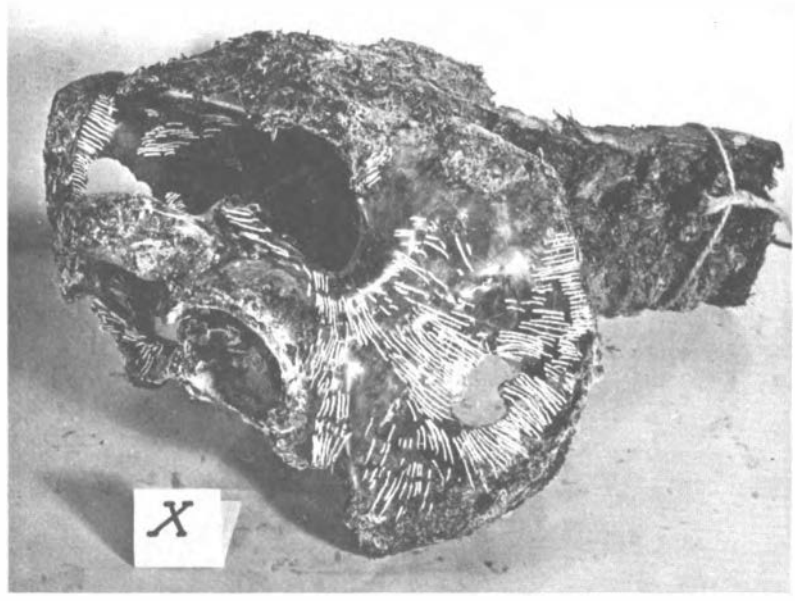

FIGURE 7
View from the right side of specimen seen in Fig. 6.

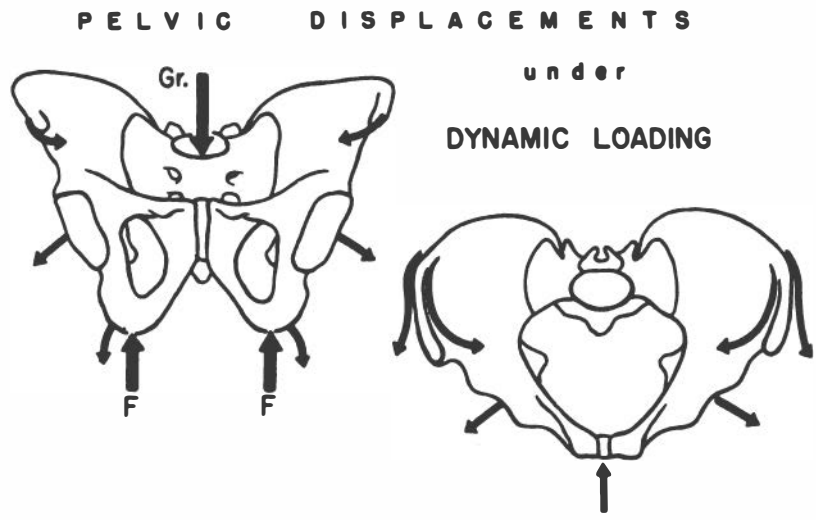

FIGURE 8
Displacements of various parts of the pelvis produced by dynamic loading of the ischial tuberosities. (Reprinted with permission from Evans and Lissner.[5])

It is hoped that the foregoing brief review of the present state of knowledge of the energy-absorbing capacity of adult human extremities and pelves will stimulate the much-needed research in this area. Data on the energy-absorbing capacity of joint structures (capsules, ligaments, and interarticular disks) would be particularly valuable and would aid in the design of protective clothing and gear to prevent, or at least reduce, the incidence of joint injuries. Anything that can contribute to the prevention or reduction of knee injuries would, I believe, be welcomed by all associated with football.

The work reported here was supported in part by Public Health Service research grant AM-03865-10 from the National Institutes of Health.

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MECHANICAL AND PHYSIOLOGIC FACTORS RELATED TO HEAD IMPACT

V. R. HODGSON, E. S. GURDJIAN, and L. M. THOMAS

Considering the number of participants in all levels of football competition, ranging from Little League through college to professional, and the numbers of injuries to other parts of the body, head injury ranks low. However, despite advances in protective gear,[8] serious head and neck injuries still occur, and, in terms of the threat of such injuries to life or normal activity, the head and neck rank as the most important parts of the body to protect. In addition, less severe head injuries, such as a subconcussive blow, can be a contributing factor to other injuries, because they reduce alertness. Because of the increase in size, speed, and specialized tactics of players, the propensity for injury will require new advances in protective headgear. It is the purpose of this paper to present our experiences in experimental and clinical head injury, with regard to types of injury and physical factors associated with them, in the hope of providing assistance in the design of head protection.

TYPES OF HEAD INJURY

The varieties of head injury may be listed as follows:[9]

Scalp:
- Laceration
- Subgaleal hematoma

Skull fracture:
- Linear
- Depressed
- Perforating

Intracranial clot:
- Epidural
- Subdural

Brain injury:
- Concussion
- Contusion
- Laceration
- Intracerebral clot

These injuries are defined below.

Scalp

Laceration. A scalp laceration is a cut or tear of the scalp due to direct contact of the head with another body.

Subgaleal Hematoma. Blood vessels connecting the pericranium and scalp may be torn as the result of force components tangential to the head. Bleeding may be diffuse, and blood may collect in the space between these structures.

Skull Fracture

Linear. A linear fracture due to a blunt blow is formed as follows:[3] At impact, the area around the point of application of the blow is bent in. Simultaneously, there is an outbending peripheral to the inbent area. This outbending is selective and may be localized to the parts of the skull where a linear fracture is initiated. Owing to the resulting tensile forces, the fracture then extends toward the point of impact and in the opposite direction. Extension is directly toward the region of impact, rather than to one side or the other, because, although this area is initially in compression (bent in), it rebounds immediately after the energy of the blow has been absorbed and becomes an area of tensile stress. If insufficient energy is expended in the blow, the fracture may remain limited and not reach the point of impact.

Depressed. A depressed skull fracture is an inward displacement of a part of the calvarium that, if other conditions were the same, can be produced by a blow of higher energy than required to cause a linear fracture.[3] The more rapid the blow, the less distant outbending there will be and the more localized the area of depression. The area of depression is fragmented by radial fracture extending from the center of impact. These radial fracture lines in a depression are due to tensile stress from inbending and are initiated on the internal surface of the skull.

Perforating. Perforating skull fractures are caused by the penetration of high-energy missiles or rigid objects of small cross-sectional area into the intracranial space.

Intracranial Clot

Epidural. Rupture of interconnecting and dural blood vessels can form a clot between the skull and the dura. This is usually produced by fracture but can occur if deformation of the skull is sufficient to cause a separation of the dura from bone.

Subdural. Blood vessels between the dura and the brain may be torn owing to relative motion between the brain and the skull caused by angular acceleration of the head or possibly by intracranial pressure due to linear acceleration of the head (Fig. 1).

Brain Injury

Concussion. Concussion has been defined[9] as the immediate posttraumatic unconscious state, not associated with macroscopic lesions of the brain, frequently reversible but potentially fatal, and associated (in the human) with amnesia. Cerebral concussion is probably due to trauma to the diencephalic and midbrain portions of the brain stem (Fig. 2).

Contusion. A brain contusion is a bruise on the surface of the brain. It is frequently seen in the frontal and temporal regions and is due to contrecoup pressure, direct slapping of the skull on the brain caused by a blow, or abrasion of the brain against the rough edges of the skull caused by the relative movements of angular acceleration (Fig. 3).

Laceration. A brain laceration is a cut or tear in the brain surface. It is usually caused by penetration of an object into the intracranial space.

Intracerebral Clot. Intracerebral clots are caused by tearing of blood vessels within the brain owing to relative movements in the brain tissue and attributed to shearing stresses involved in the pressure gradients of head accelerations and skull deformations (Fig. 4).

INVESTIGATIVE METHODS AND RESULTS

We will concentrate our attention on intracranial clots and brain injuries, because these are most prevalent among head injuries associated with football.

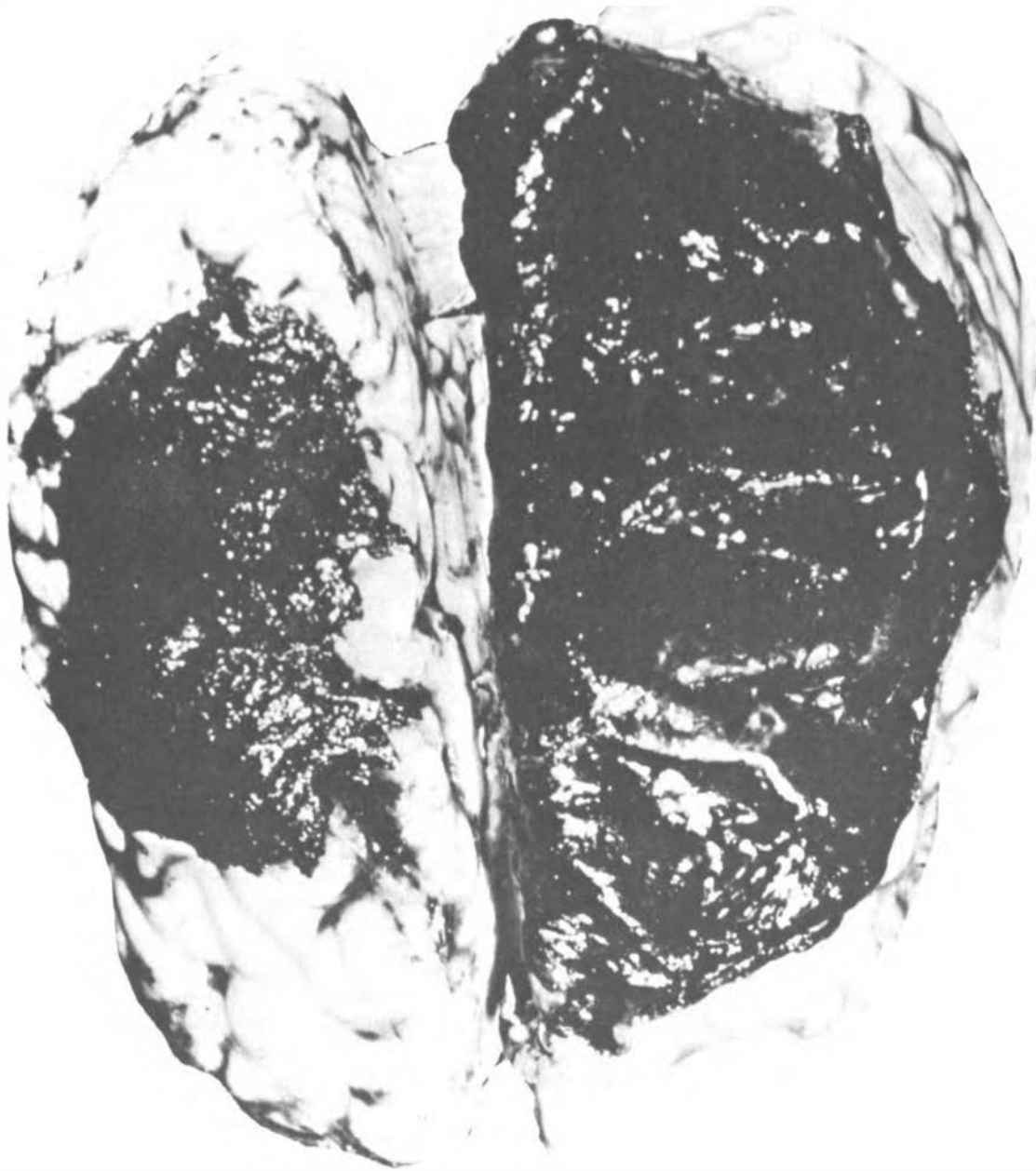

FIGURE 1
Acute bilateral subdural hematoma due to laceration of vessels from relative movements of the brain and veins connecting with the sagittal sinus.

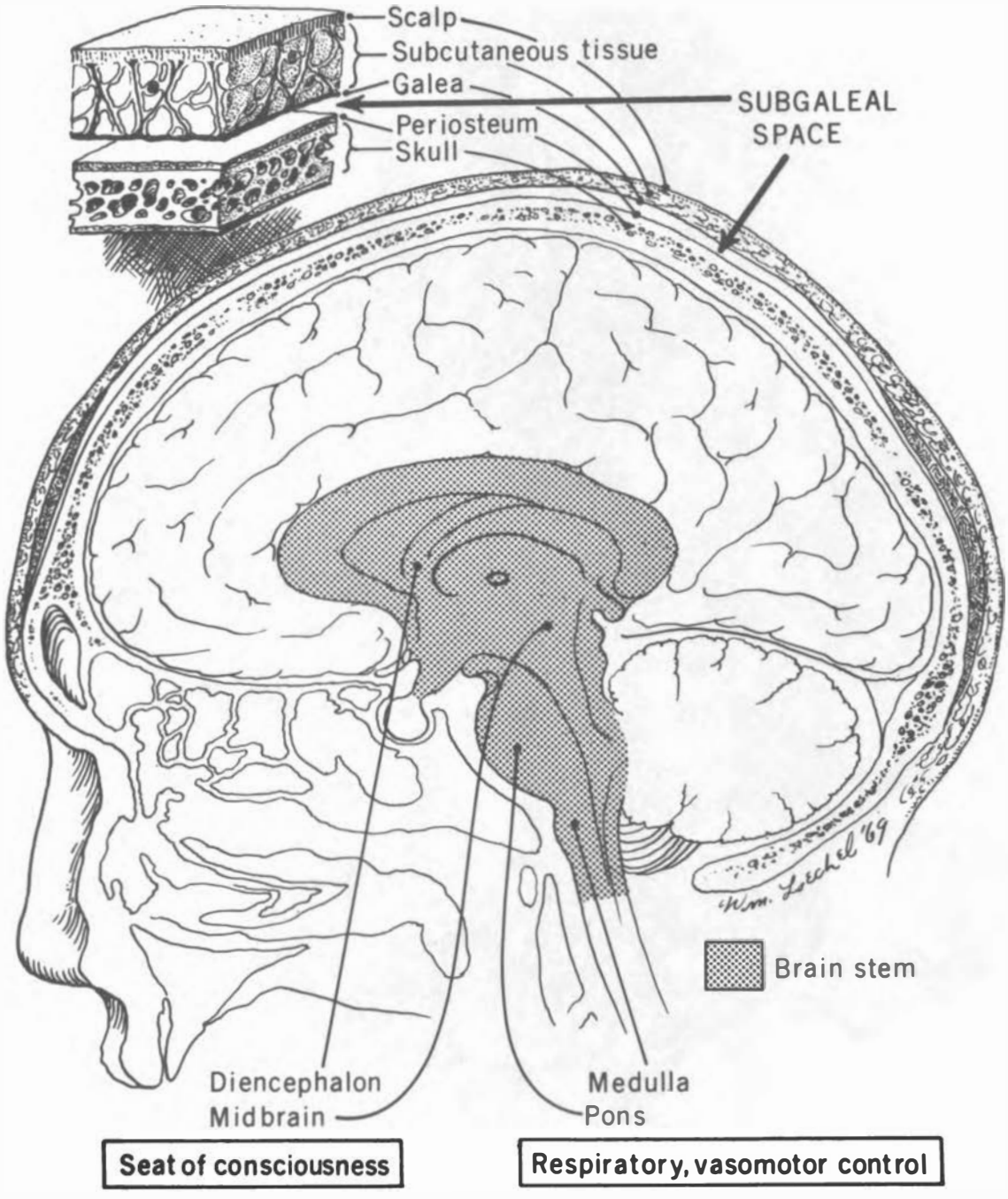

FIGURE 2
Sagittal section of the human head, showing some of the vital areas involved in head injury. (Reprinted with permission from *Tomograms of the Head*, by Marvin L. Daves and William E. Loechel. Published by Charles C Thomas, Springfield, Ill., 1962.)

FIGURE 3
Impact on dog head without fracture of the skull but with a contusion of the brain from the elastic inbending of the bone.

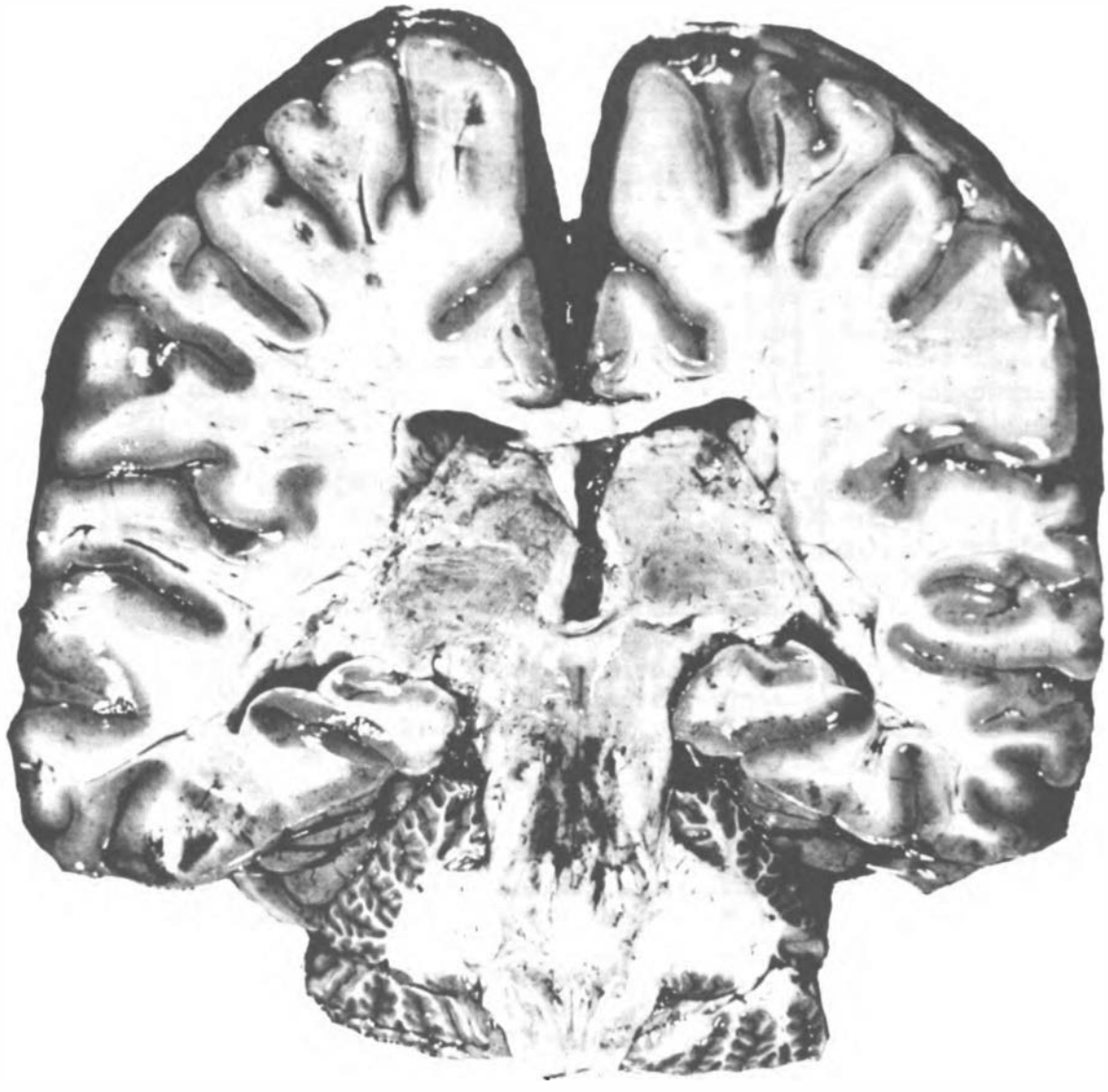

FIGURE 4
Extensive brain stem hemorrhages due to pressure gradients at the cranial spinal junction.

In our laboratory, we use anesthetized animals, human cadavers, and experimental and mathematical models to study head and neck injuries. We have studied the various indices of injury of the scalp, skull, and brain. We have measured deformation of the skull, which may result in various types of fractures and brain injuries. We have measured the motion of the heads of anesthetized animals after impact; intracranial structures are probably injured by relative movement of tissues as a result of translational and angular accelerations. We have observed responses to low-level sinusoidal forces at points on the skulls of human cadavers to try to learn about the mechanical impedance characteristics of the head, which are related to specific types of injury. The results of our studies are summarized in the following paragraphs.

The degree of injury suffered by an experimental animal after an impact to the head is determined by physiologic monitoring and by ascertaining response to mechanical and noxious stimuli. Figure 5 includes a graph of physiologic changes in a 13.5-lb (6.1-kg) mature female stumptail monkey after it received a blow that rendered it unresponsive to any kind of stimuli for several minutes and groggy for an hour. It sustained no fracture of the skull. This is an example of a moderate experimental concussive effect, which is characterized by a rise in blood pressure, decrease in heart rate (bradycardia), cessation of respiration (apnea), electrocardiographic abnormalities, and loss of response to mechanical stimuli (see Fig. 5). The results are similar in dogs and cats.

In a series of experiments with the stumptail monkey, the single index most closely associated with degree of concussion was apparent head acceleration (impact force divided by head weight). A plot of degree of concussion *vs.* apparent head acceleration is shown in Fig. 6. The degree of concussion was based on a relative study of the typical physiologic manifestations (shown in Fig. 5), as well as a careful electrocardiographic and electroencephalographic study of the animals. The animals in this series weighed 10.5-23 lb (4.8-10.4 kg) and the tests revealed that the larger the head, the harder it was to produce experimental concussion. In this species, head weight was proportional to body weight. In the human, of course, that is not always the case; in fact, smaller individuals may have larger heads than larger individuals. At present, this work and the studies of others indicate that linear head acceleration and associated pulse duration are the best bases for evaluating head-protective equipment.

It has been observed that, in an acute experiment, a series of subconcussive blows do not produce an observable cumulative effect. Koch and Filene,[5] however, have concluded that there is cumulative effect in the experimental animal with more severe blows. These opposing views are difficult to reconcile, but intensity of the blow and the condition of the animal are undoubtedly important in any cumulative effect. Age, sex, weight, state of health, and anatomic and physiologic differences may influence the results. The data summarized in Fig. 7 were gathered from multiple head blows to the same animal. It shows the variations

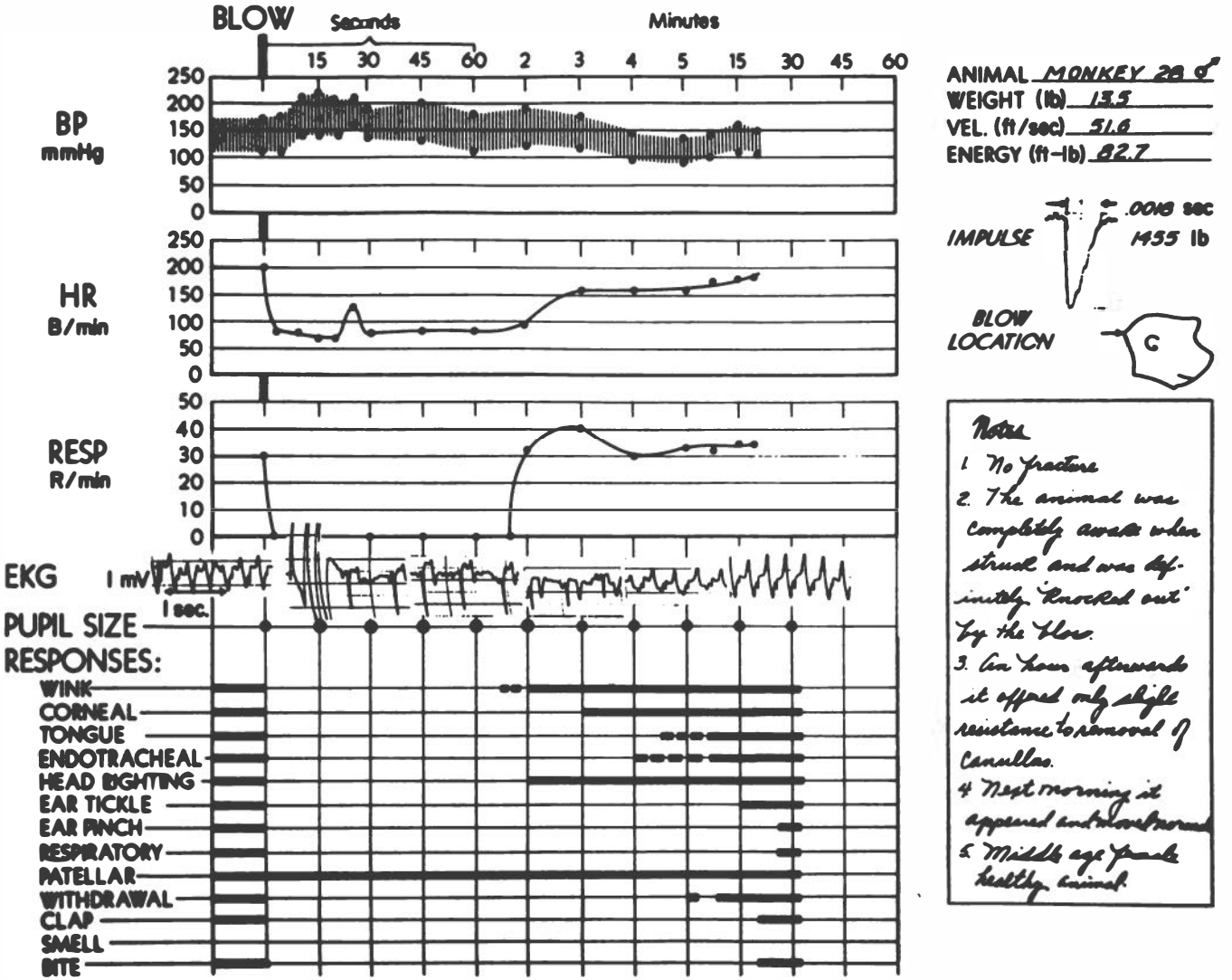

FIGURE 5
Typical physiologic changes seen in the lightly anesthetized stumptail monkey after a concussive blow to the occiput.

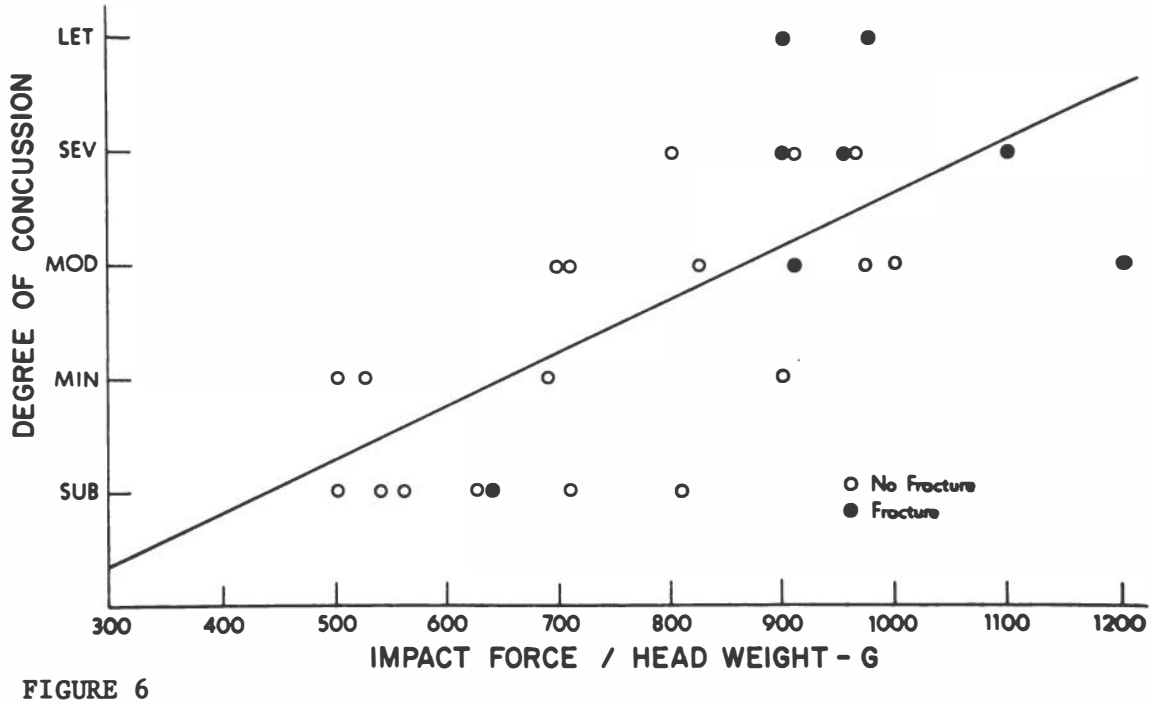

FIGURE 6
Relationship between degree of concussion and impact force/head weight (rigid-body acceleration).

in force, intracranial pressure, head acceleration, and pulse duration as the velocity of impact is increased from a low level to a lethal level. It can be observed that the pulse duration remains essentially constant throughout the range of velocity from 8 to 50 ft/sec (2.4 to 15.2 m/sec). The acceleration measured on the top of the head in line with the force increases nonlinearly from a low level to 500 *g* at 20 ft/sec (6.1 m/sec). At this point, the pressure opposite the blow (pickup 5) approaches -1 atm, which is near the vapor pressure of the spinal fluid in which the pressure pickup is bathed. Thus, a minimal pressure of nearly -1 atm was obtained for all subsequent higher-velocity blows. The maximal positive pressure on the side of the impact (pickup 6) increases positively in a nonlinear manner to about 95 psi (6.7 kg/cm^2) at the lethal velocity. The force of impact increases at a nonlinear rate to about 1600 lb (725 kg) at lethal velocity. The head acceleration increases even faster than the force, indicating the presence of some angular acceleration in the head motion. Undoubtedly, angular accelerations play a large role in the production of head injury, particularly hemorrhagic effects on the surface of the brain. However, up to now, it has been impossible to find any correlation between extent of head injury and angular acceleration, because flexural and vibration effects make it difficult to record skull acceleration at high impact velocities.

The effects of head acceleration in the human may be visualized by observing a transparent two-dimensional model of the sagittal section of the human head in a sequence of high-speed photographs taken during a

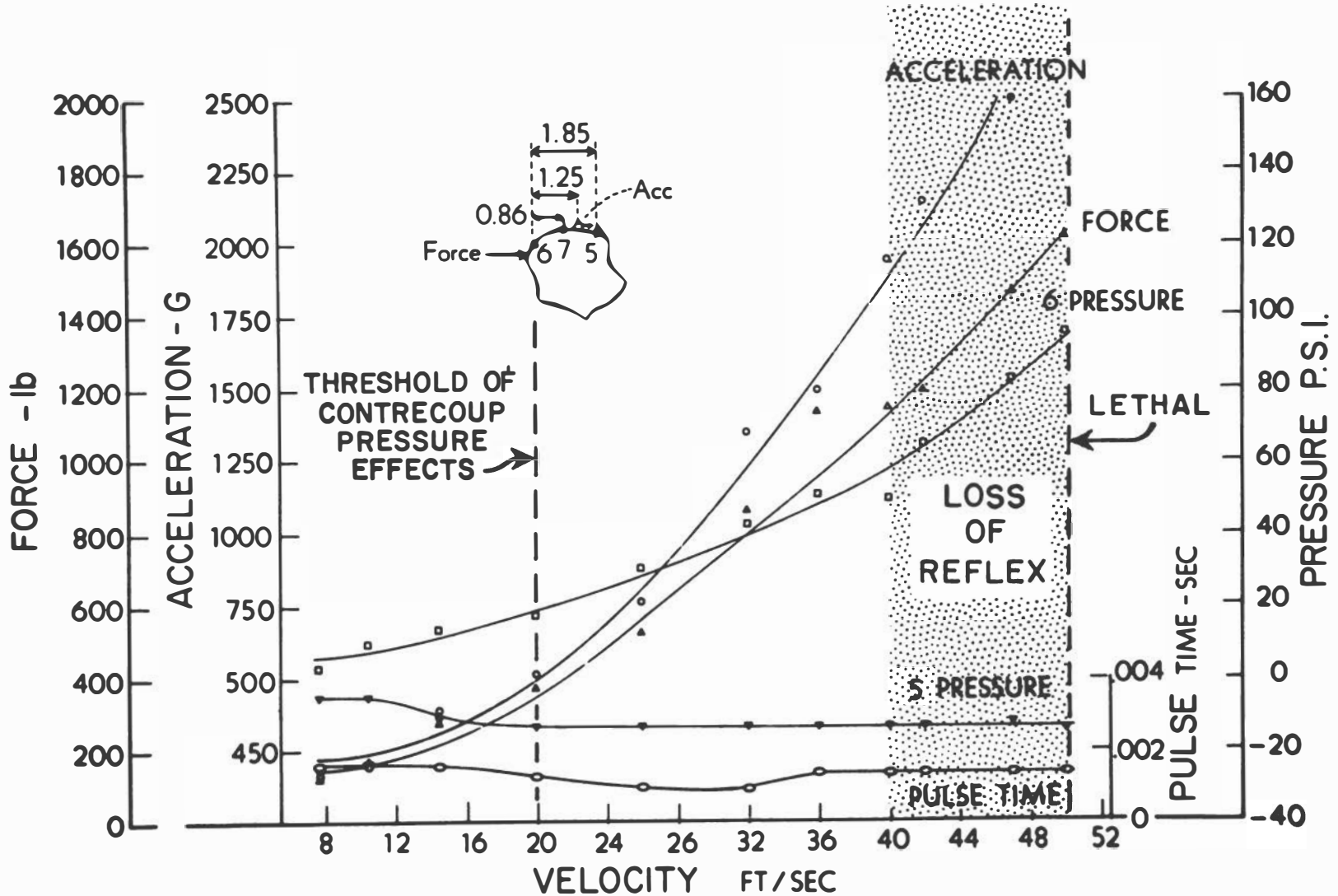

FIGURE 7
Variations in force, head acceleration, intracranial pressure, and pulse time as functions of impact velocity of a 2-lb (0.9-kg) striker against the occiput of a stumptail monkey.

0.003-sec impact to the model (Fig. 8). As with the blows to the anesthetized monkey, a positive pressure is recorded in the model on the side of the blow; opposite the blow, a negative pressure is indicated by the growth of tiny air bubbles. They attain a maximal size in frame 7 (after 0.0015 sec) and then disappear as the striker and head separate. Pressure records indicate that the phenomenon of reaching a pressure of -1 atm followed by a sharp positive-pressure spike occurs at a threshold level of 170 *g* in a model or cadaver preparation with a 6-in. (15.2 cm) major diameter and increases in severity as acceleration increases. These pressure effects may be interpreted as being due to acceleration of the skull and inertia of the contents, which produces a positive-pressure buildup on the blow side and tends to produce separation, and therefore below-atmospheric pressures, on the side opposite the blow. The negative gauge pressure extends toward the blow side into the brain a distance depending on the intensity of the impact. Blood-vessel hemorrhages (like the petechial hemorrhage in Fig. 4) detected opposite the impact locations have been attributed to this phenomenon by Unterharnscheidt and Sellier[11] and Gross.[1]

The physical properties of the head, including weight, structural geometry of the components, elastic and viscoelastic characteristics, and natural frequencies, as well as the size and shape of the striking object, relative velocity of impact, line of action, blow location, intervening absorbing material, area of impact, and body orientation with respect to the head during impact, are primary factors in determining the extent and nature of head injury. We have shown in the human cadaver, with the predominantly oval head, that deflections and volume changes under a static anteroposterior or posteroanterior load are very much smaller than those under a side-to-side loading.[10] This indicates that forces along the major a-p axis would produce predominantly acceleration (or deceleration), whereas transverse forces would tend to produce deformation with acceleration of the head. We showed that in the dog a blow to the occipital protuberance with an upward component was much more effective than transverse blows to the side or top of the head in producing experimental concussion and mortality.[2] Some of the signs of experimental concussions--such as loss of corneal reflex, drop in blood pressure, bradycardia, and apnea--are manifestations of neural dysfunction in the medullary region of the lower brain stem and upper cervical region of the cord due to shear stress during movement caused by pressure gradients.[9] It is probable that a blow to the occipital protuberance of the dog has more effect on these regions of the brain than a transverse blow. An occipital collision or uppercut in the human may have the same effect.

Figure 9 shows a flash x-ray sequence of the head of a monkey before impact (left) and 0.0005 sec after initial impact to the occipital protuberance. Lines of lead tags have been inserted into the brain of this anesthetized animal in positions calculated to indicate relative movements of the brain stem and brain with respect to the skull. It is evident from these x-rays that the blow has produced both linear

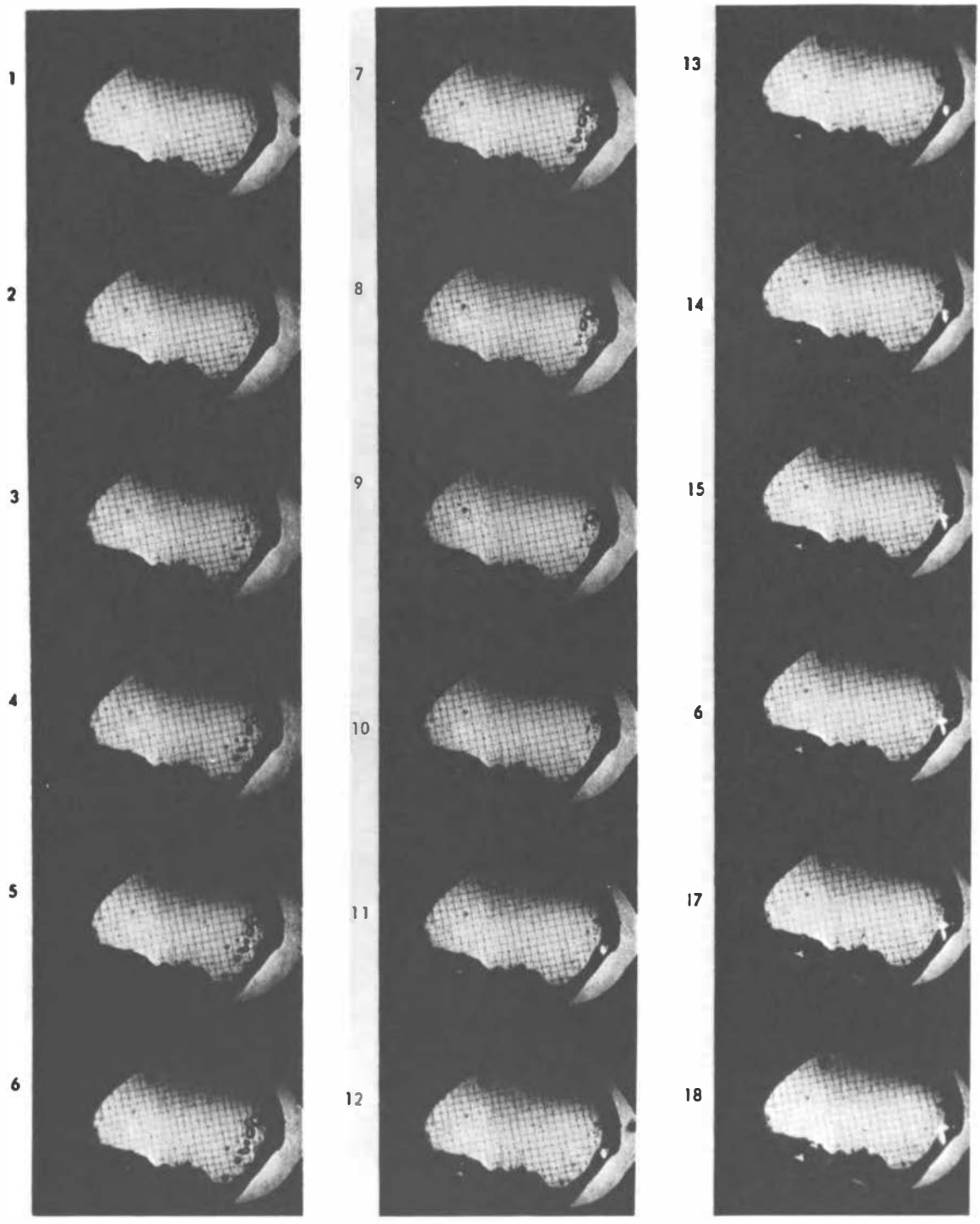

FIGURE 8
High-speed photographs of a two-dimensional clear plastic model of the human head taken at 7000 frames/sec. Frames 3-7 show the growth of bubbles in the occipital region due to below-atmospheric pressure caused by impact to the forehead on the left. The bubbles have practically disappeared by frame 12. Total elapsed time of bubble formation, 0.0015 sec.

and angular acceleration, which in this experimental situation were controlled to be plane motion. Unless by coincidence the line of action of the resultant force acting on the head passes through its center of gravity, the resultant motion will in general be translatory, as well as rotatory about the orthogonal axes through an arbitrary reference point. In the monkey subject shown in Fig. 9, there is no macroscopic evidence of relative movement in the brain tissue indicated by the lines of lead resin core solder across the brain. However, at the craniospinal junction, at least 2 mm of tissue displacement downward can be observed, indicating stretch and shear in the area of the medulla of the brain stem where the respiratory and vasomotor centers are. Pudenz and Shelden[6] and Hirsch and Ommaya[4] have shown that much larger relative movements between the brain and skull occur, owing to much longer-acting forces, indicating that relative movement of brain tissue is a function of time and diminishes as pulse time decreases. The impact depicted in Fig. 9 lasted only 0.0017 sec, because only skin and a hard rubber pad were between the striker and the skull. In a typical football collision involving a helmet, the pulse duration could be expected to be 0.007-0.030 sec, depending on the conditions of the impact.

More insight into the response of the human head to an impact may be obtained by studying Fig. 10, which compares the frequency response of a point on the front of the skull of the human cadaver (based on a silicon gel preparation, of the same consistency as brain tissue) with the frequency spectrum of pulses delivered to the head with and without a helmet. The top portion is a plot of mechanical impedance, dynamic stiffness, and apparent mass of the head against frequency of the vibrating force. The body is situated in an erect seated position suspended about the center of gravity with the head free to move (see Fig. 11). Owing to inertial, elastic, and damping properties of the head, the mechanical impedance, which is resistance to motion of a point on a body (force/velocity), varies with frequency, as do dynamic stiffness (force/displacement) and apparent weight (force/acceleration). The force and acceleration are measured at the driven point on the forehead in discrete frequency steps from 0 to 5000 Hz. The ratio of force to acceleration is then plotted as the apparent weight *vs.* frequency on the nomogram, and the mathematical relationship between apparent mass, dynamic stiffness, and mechanical impedance is governed by the nomograph construction. Mechanical impedance is plotted on the horizontal lines, the dynamic stiffness on the negative-slope lines, and the apparent mass on the positive-slope lines. In summary, the plot shows that the point on the forehead appears to the driving force as a mass of 10-11 lb (4.5-5.0 kg) up to 130 Hz, rising to a maximum of 25 lb (11.3 kg) at 313 Hz, falling steadily to 0.1 lb (0.05 kg) near 900 Hz, and leveling off near 0.5 lb (0.2 kg) above 1000 Hz. The mechanical impedance varies from low values below 10 lb/in.-sec (1.8 kg/cm-sec) to a maximum near 150 lb/in.-sec (20.6 kg/cm-sec) at 313 Hz, and then to a minimum value of 2 lb/in.-sec (0.4 kg/cm-sec) at 900 Hz. The dynamic stiffness varies from around 1000 lb/in. (180 kg/cm) at 30 Hz to a maximum of 250,000 lb/in. (45,000 kg/cm) at 313 Hz. It then drops to 10,000 lb/in. (1800 kg/cm) at 900 Hz and increases steadily thereafter.

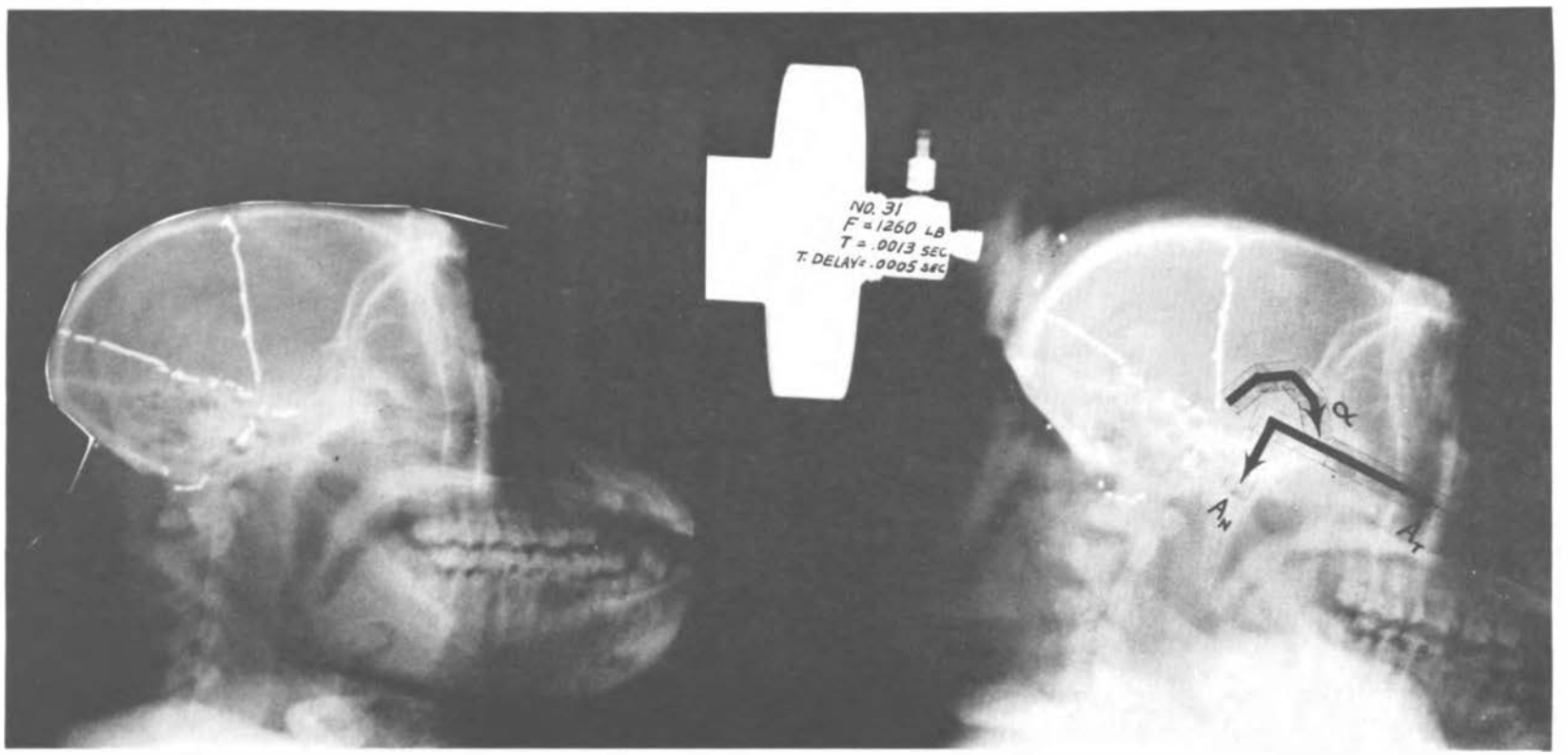

FIGURE 9
Flash x-ray of anesthetized stumptail monkey taken before impact (left) and 0.0005 sec after contact of a striker with the occiput. The x-rays indicate that both translation and rotation of the head occur. Lead solder tags at the craniospinal junction indicate several millimeters of superior-inferior movement of the brain stem relative to the skull at this location.

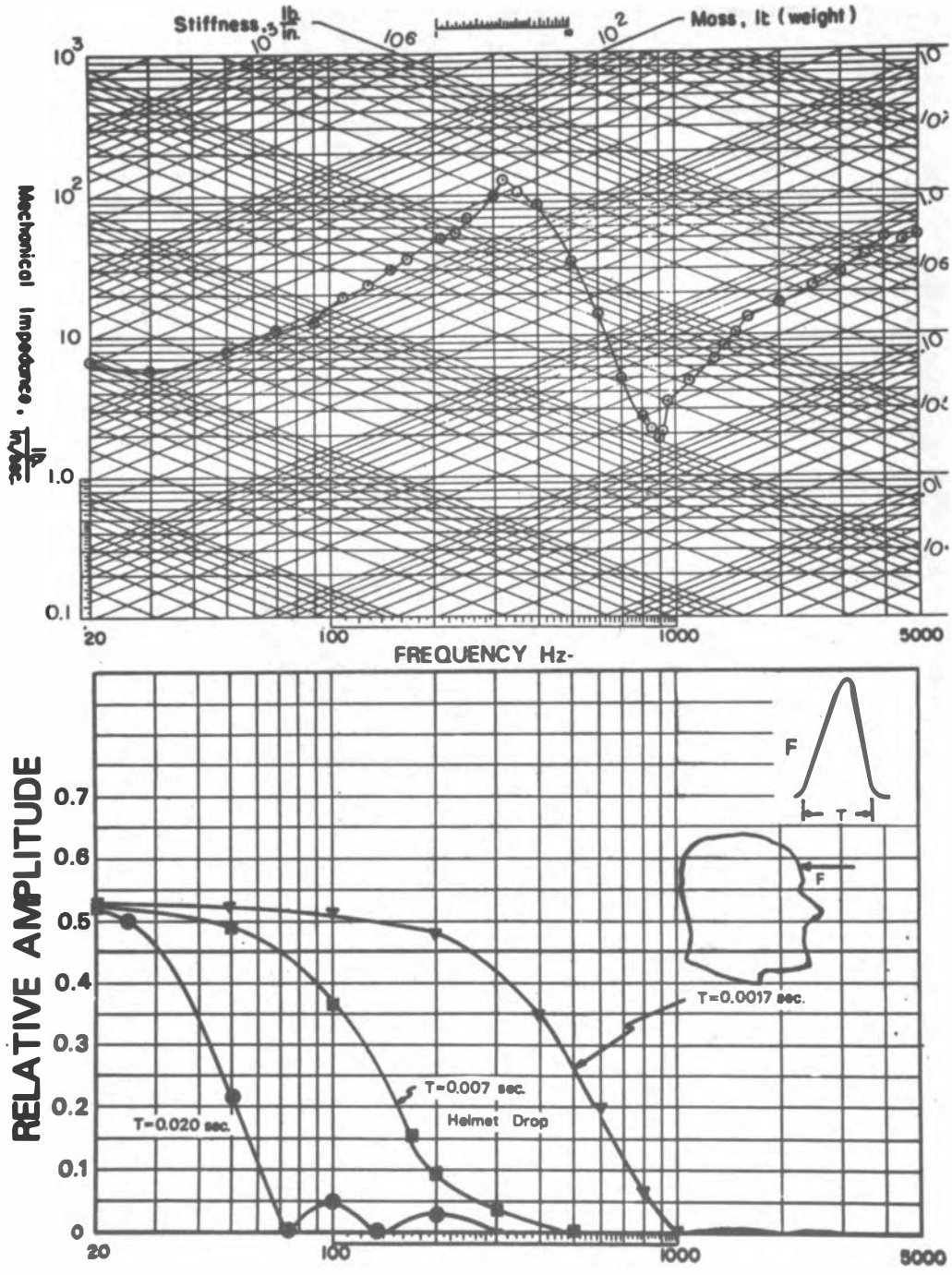

FIGURE 10
Top, plot of mechanical impedance data *vs.* frequency for 1-lb (0.45-kg) sinusoidal input to the frontal bone of the skull of the human cadaver. Bottom, frequency spectrum of three pulses delivered to the forehead of the same cadaver used to obtain the mechanical impedance data above. These spectra are for a short pulse (t = 0.0017 sec, no padding), for a helmet drop onto a rigid surface, and for a long pulse (t = 0.020 sec).

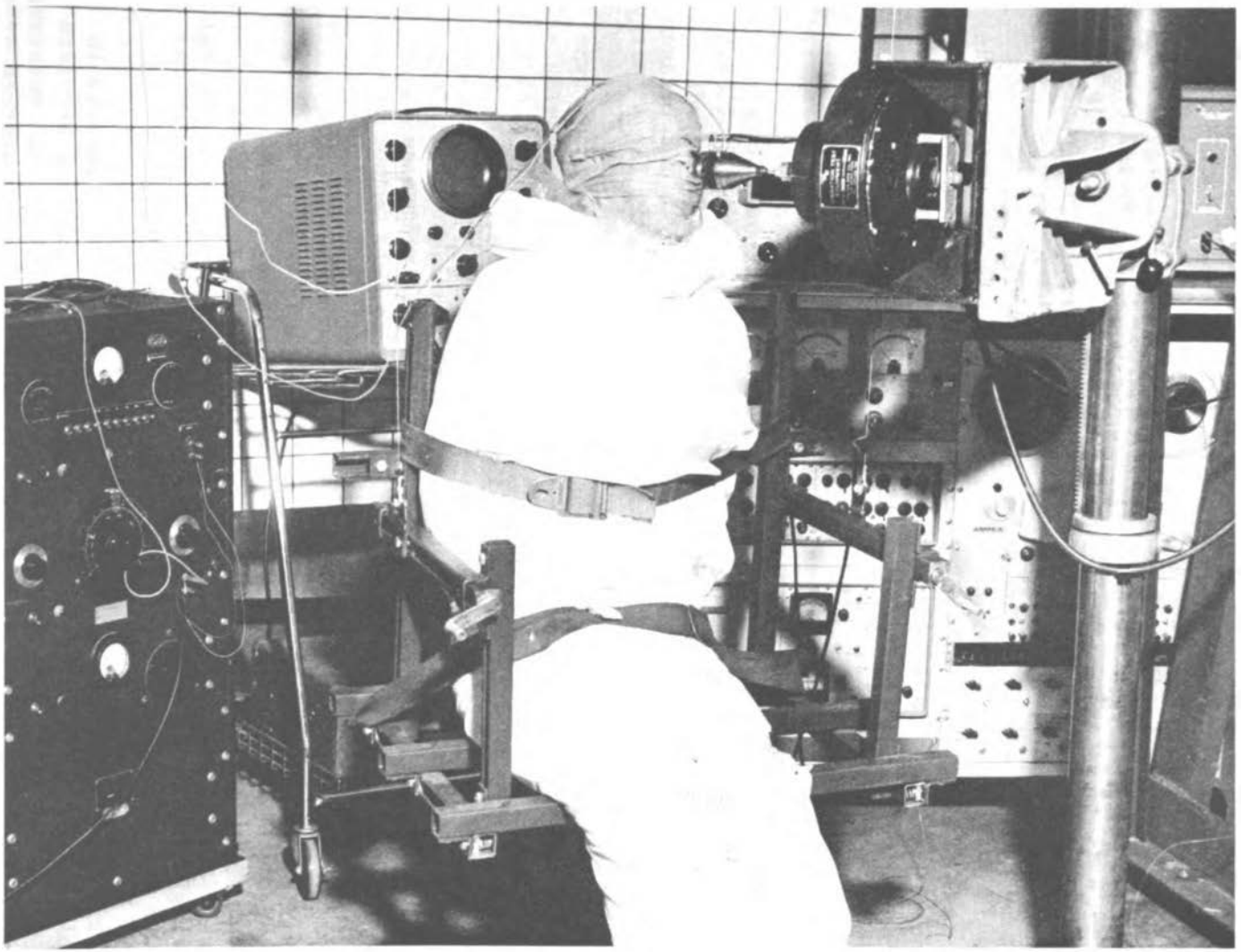

FIGURE 11
Setup for determining the mechanical impedance of a point on the human cadaver head.

The interpretation of this chart is as follows: Below 150 Hz is the frequency range in which the skull behaves essentially as a rigid body. The phenomenon at 313 Hz is called "antiresonance" and represents the first mode of bending, in which the skull flexes in a symmetric manner such that the rear of the head deflects while the front of the head presents maximal resistance to being moved. At 900 Hz is the third mode of the head, in which the front of the head deflects with minimal energy input. An unsymmetric second mode near 600 Hz could not be investigated thoroughly by our methods. Above 1000 Hz, the sinusoidal force is driving only the portion of the skull that immediately surrounds the point of application.

The bottom portion of Fig. 10 shows the frequency spectrum of pulses delivered to the forehead of the same cadaver on which the mechanical impedance data were taken. The spectrum shows the distribution of energy in a pulse as a function of frequency range to the left of the first touchdown of the curves; the natural frequency of the struck object determines the nature of the injury, as well as important characteristics of the pulse. The spectrum width varies inversely as the duration of the pulse; i.e., a long pulse will have a frequency spectrum with a narrow width, such as the 0.020-sec pulse spectrum shown on the left. The spectrum of this pulse is too narrow to excite bending modes in the head (below 150 Hz); consequently, all its energy will be transmitted as kinetic energy, producing rigid-body acceleration of the head and therefore accelerative types of brain injuries, as discussed previously. However, the energy in an extremely short pulse, such as the 0.0017-sec pulse recorded for impact to the cadaver forehead with no padding, is more diffuse, with relatively large energy components over a broader frequency range. A short pulse has a propensity to excite bending modes in the head and therefore is the type of pulse that, if it contains sufficient energy, produces fracture.

Between the plots of the 0.020-sec and 0.0017-sec pulse spectra is the plot of the spectrum of a 0.007-sec pulse obtained from an impact of a cadaver wearing a crash helmet (fiberglass shell with nonresilient liner on a rigid surface). This is probably the minimal pulse duration to be expected in a football contest--e.g., a fall onto a frozen turf. Most football head impacts would be of much longer duration using any of the modern headgear and therefore would produce predominantly acceleration of the head. Experience bears this out. For example, a professional football team might experience a maximum of six or seven concussions (players rendered unconscious) in a season, with perhaps the same number of subconcussive injuries (players dazed) in a single contest, but fractures are practically unheard of (on the basis of conversations with professional football trainers).

Reid *et al.*,[7] at Northwestern, were the first to study head impacts in football by means of radiotelemetry. They mounted a triaxial accelerometer to the right of the vertex of the shell of the helmet and measured accelerations along the x, y, and z axes. Their work was concerned primarily with the study of the feasibility of recording by radiotelemetry,

and it is uncertain what their recorded accelerations mean in terms of force input to the head or actual head accelerations and duration of impact. Accordingly, a preliminary study has been instituted in the Wayne State University Department of Neurosurgery laboratory designed: (1) to determine the relationship between helmet accelerations and head accelerations on the human cadaver for several of the types of football helmets commonly in use; (2) to determine the force-time input to the helmet corresponding with the measured accelerations; (3) to compare impact response of the head, including linear and angular acceleration, with the several types of helmets and with the body and head in various common attitudes with respect to each other during impacts to the front, side, and rear of the helmet; (4) to compare impact response of the helmets against a rigid barrier and soft turf; (5) to compare head-impact acceleration response with and without helmets; (6) to develop a harness or transducer mount that, when worn by the cadaver with miniature triaxial accelerometers attached, will produce accelerations similar to those measured near the same point on the skull; and (7) to record head-impact accelerations in football situations by radiotelemetry data transmitted from the heads of volunteers.

Preliminary results of drop tests (as illustrated in Fig. 12) indicate that helmet accelerations are much higher in amplitude (by a factor of about 4) and high-frequency components and have no relationship to skull-acceleration pulse shape directly under the helmet pickup. Figure 13 illustrates a typical oscillographic record of a drop test. Channels are referred to location in the insert diagram.

SUMMARY

The primary types of head injuries to be expected in football are related to intracranial pressure changes and relative movements caused by linear and angular accelerations (or decelerations). These are concussions, subdural hematomas, and petechial hemorrhages. It has been found that, for blows delivered to the occiput of a group of stumptail monkeys, the ratio of force to head weight (rigid-body acceleration of the head) was the best single index of degree of concussion. Head-impact tests involving various energy levels and body attitudes of human cadavers wearing several current and proposed helmets have begun in the laboratory. The general purpose of the experiments is to correlate head and helmet accelerations, input force, energy, and attitude in the laboratory with radiotelemetry data and high-speed photographs from scrimmage and college varsity action.

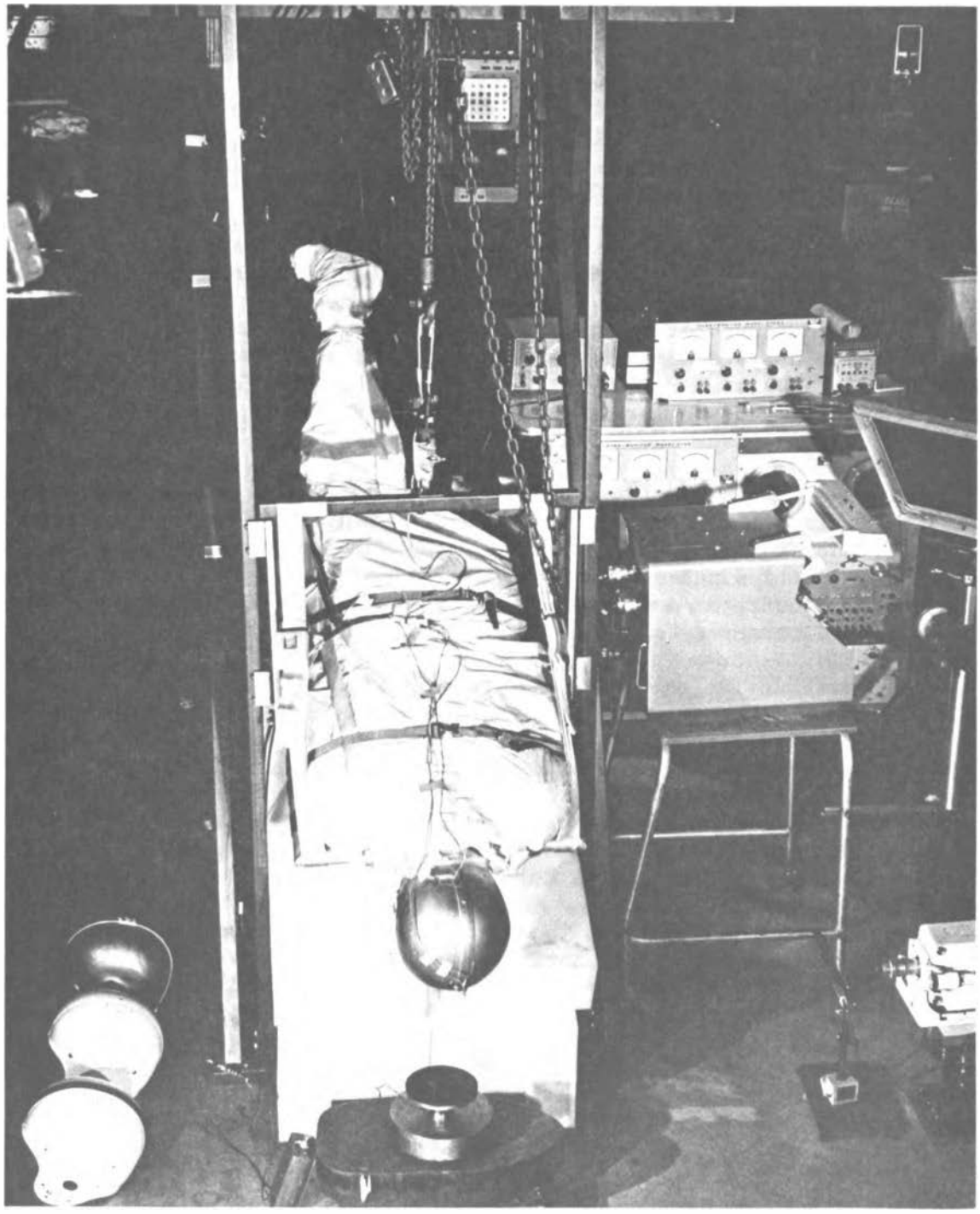

FIGURE 12
Typical setup for determining forces, head accelerations, and energies involved in impacts against a rigid surface with the body in various attitudes.

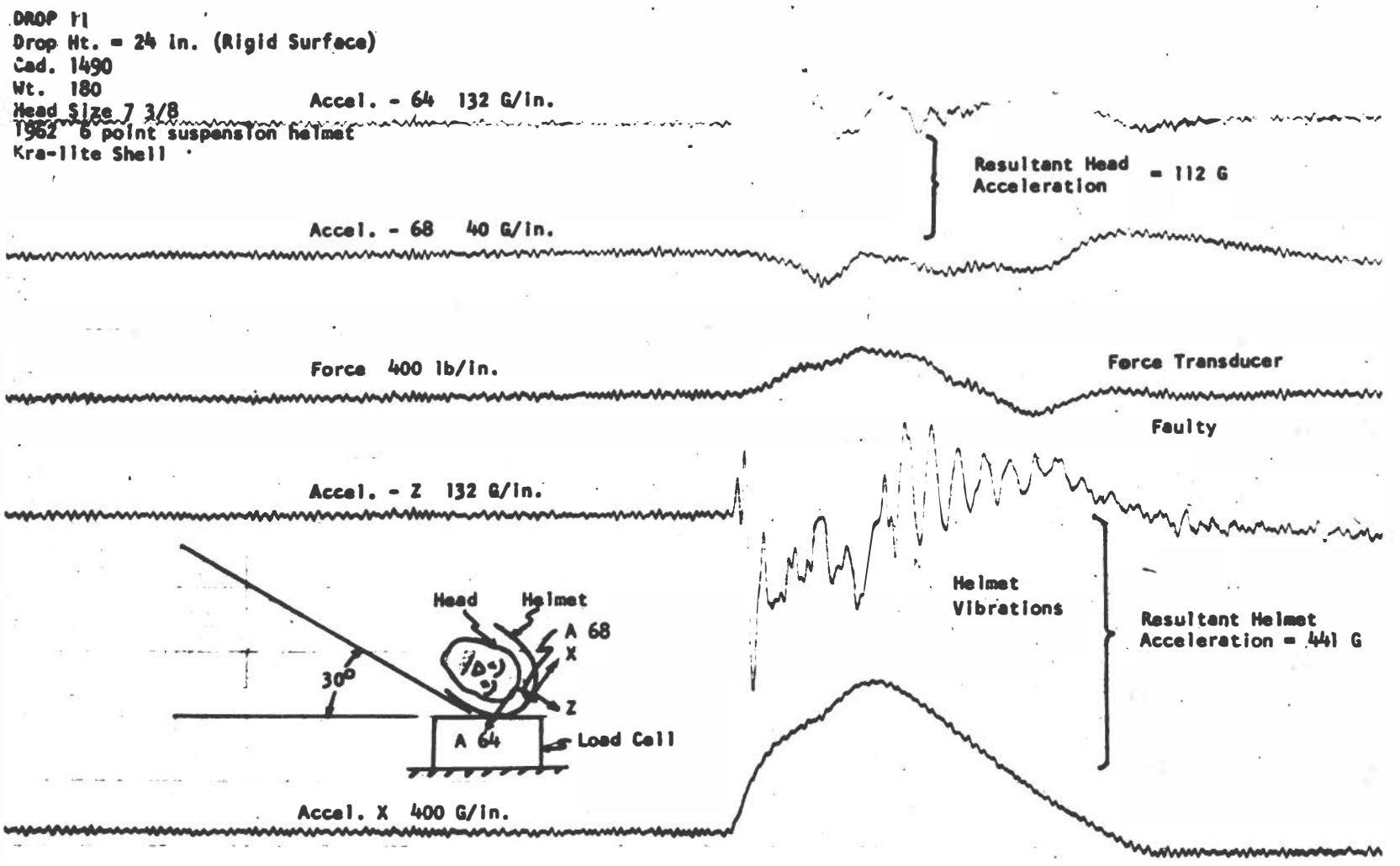

FIGURE 13
Typical oscillogram recorded to compare cadaver-head accelerations with accelerations measured at an adjacent location on the helmet.

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RADIOTELEMETRY STUDY OF HEAD INJURIES IN FOOTBALL

STEPHEN E. REID, JOSEPH A. TARKINGTON, and MATTHEW PETROVICK

Over 1 million young men participate in the game of football in the United States. During the 1967 season, 24 deaths directly related to football were recorded: 16 in high-school, five in sandlot, and three in college football. Of the 24 deaths, 21 (88%) resulted from injuries to the head and neck. This fatality incidence of fewer than three per 100,000 participants has remained fairly constant from year to year and implies that nothing has been done to improve the situation. Actually, with the changing game and today's bigger and faster athletes, the potential danger has increased and the fact that the fatality incidence has remained the same is a tribute to those responsible for the safety of the players.

The purpose of our research study, begun in 1961 at the suggestion of members of the Committee on the Medical Aspects of Sports of the American Medical Association, is to gather impact data to help reduce the incidence of head injuries in football players. This progress report covers the three phases of development of a perfected telemetry system, preliminary data, and plans for future work. The study has developed a reliable method for telemetering any type of scientific or physiologic data from the football field and could be valuable in developing or proving the reliability of any protective device, including those for the knee.

PHASE I: TELEMETERING OF HELMET IMPACT

All previous studies on head injuries in football had been done in the laboratory, recording accelerations in g's as a weight struck a head form. Forces were determined from calculations of conditions simulating those observed in slow-motion pictures of football games. In our study, the method of radiotelemetry and microelectronics was used to instrument a player in such a way that the actual impact to his helmet during regulation football games could be measured. The player participated in the game without being conscious that he was a "research laboratory." Slow-motion pictures were taken of the instrumented player in action.

To avoid commercialism with regard to any particular football helmet, we did not attempt to test the helmet on the field, but recorded the impacts encountered by the helmet, rather than by the head. All manufacturers then could be told of the forces encountered on the field and use the information to improve their products.

The first electronic helmet was worn by Joe Schmidt, the Detroit Lions linebacker, in the Pro Bowl game in January 1962. The method was perfected and a triaxial accelerometer was fixed to the shell of the helmet. The accelerometer measured impacts in three mutually perpendicular planes, located the point of contact on the shell of the helmet, and measured the time during which the force was acting. This information was then telemetered to the press box, where it was recorded.

Impact data of blows to the helmet (not to the head) were measured during the 1963, 1964, and 1965 football seasons of Northwestern University, and 893 impacts on one player's helmet were recorded (Table 1). During 50% of the time that the player was in the game, he received no contact. It was interesting to learn that 725 (81.2%) of the blows were encountered by the sides of the helmet, 96 (10.8%) by the front, 44 (4.9%) by the back, and only 28 (3.1%) by the top. Forces were recorded in g's, and the time during which the force was acting was measured in milliseconds. The recorded g's ranged from zero to 5780, but 87% of the blows registered 400 g or less, 7% from 400 to 1000 g, and 6% over 1000 g. The time during which the force was acting ranged from 1 to 150 msec, with the higher g ranges associated with the shorter intervals.

PHASE II: TELEMETERING OF BRAIN WAVES

In 1965, when the method of telemetering impact data from the football field was established, the decision was made to proceed to the next phase of the study: telemetry of the brain waves of the football player during a game. Electroencephalography provides a visual record of the spontaneous electric activity emanating from the brain through electrodes placed on the scalp. The principles are physiologically similar to recording the heart waves in electrocardiography. A tracing is recorded from each side of the brain by two pairs of electrodes placed at the parietal and occipital regions. A fifth electrode in the midline of the occiput acts as a ground wire (Figs. 1 and 2).

The method of telemetering electroencephalographic (EEG) tracings was perfected during the 1965, 1966, 1967, and 1968 football seasons of the Northwestern University football team. The normal background rhythm of adults during the resting state is 9-12 cycles/sec (alpha waves) when the subject has his eyes closed (Fig. 3). It was necessary to establish the instrumented player's background rhythm during the excitement of a football game with his eyes open, because this pattern had never before been established. Other activities of the player

produce different patterns, which were tabulated and found to be consistent for specific activities. The EEG tracings recorded at the very moment of impact may be of little value, because the scalp and the electrodes on it usually move with reference to the generator potential from the brain and, therefore, produce an artifact (Fig. 4). However, the EEG can be very significant immediately after impact, in that it normally reverts to its basic background rhythm. The frequency of the background rhythm tends to rise consistently with the commonly accepted relationship between high-frequency alpha activity and tension or extreme alertness; at other times, the frequency may fall below the value recorded under conditions of relative rest. It is this slowing of the background rhythm after impact that could indicate brain damage. This has not happened during this study, but the means for recording such data has been accomplished. There has been no previous knowledge of the character of the EEG pattern immediately after impact; such knowledge and a correlation with impact can now be obtained.

TABLE 1
Distribution of blows to football helmet during 1963, 1964, and 1965 seasons

Impact, *g*	No. blows	Duration of force, msec
0-400	781	1-150
401-1000	61	1-30
>1000	51	1-4

Recording force in *g*'s proved unsatisfactory. Although these were impacts encountered by the shell of the helmet, and not by the head, 1000 *g* seemed unrealistic. Force equals the product of mass and acceleration (F = MA). In the laboratory, the use of a head form allows the mass to remain constant; force is then directly proportional to acceleration and has some real meaning. On the football field, the player is a "coiled spring," and the mass varies from practically nothing, as in a near miss, to probably several pounds. What was actually being recorded, in most instances, were the vibrations of the shell of the helmet; even with recorded accelerations of over 5000 *g*, the player rarely experienced ill effects. This study has demonstrated that conditions that prevail on the football field cannot be simulated in the laboratory, nor are they identical on the field even under similar sets of circumstances.

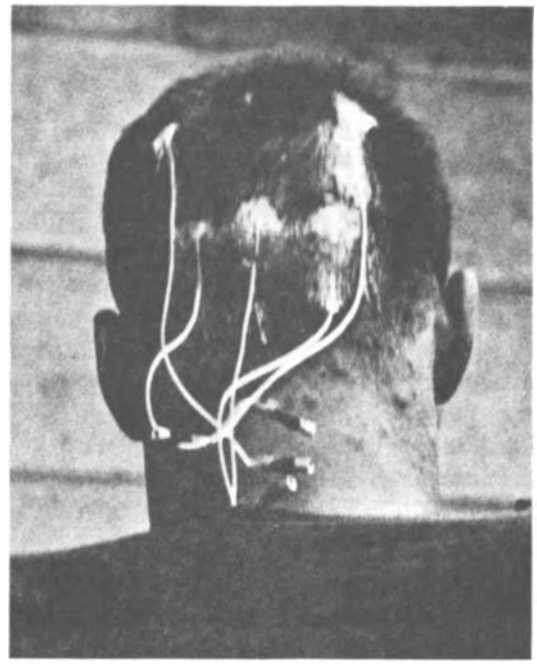

FIGURE 1
Bob Olson after a football game, with electrodes in place.

FIGURE 2
Special compartment on Bob Olson's helmet for EEG transmitter.

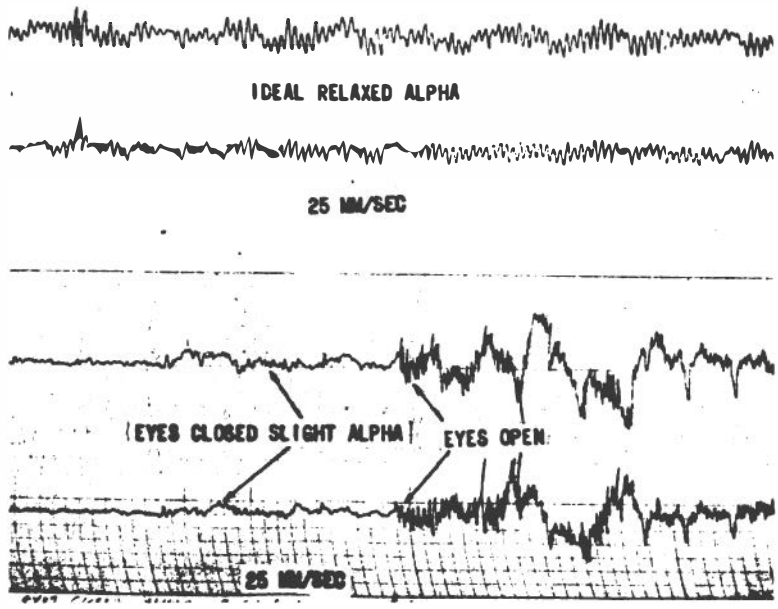

FIGURE 3
Top, telemetered EEG tracing showing alpha wave (eyes closed) from each side of brain. Bottom, sudden change due to stimulus of opening eyes, followed by settling down to normal background rhythm.

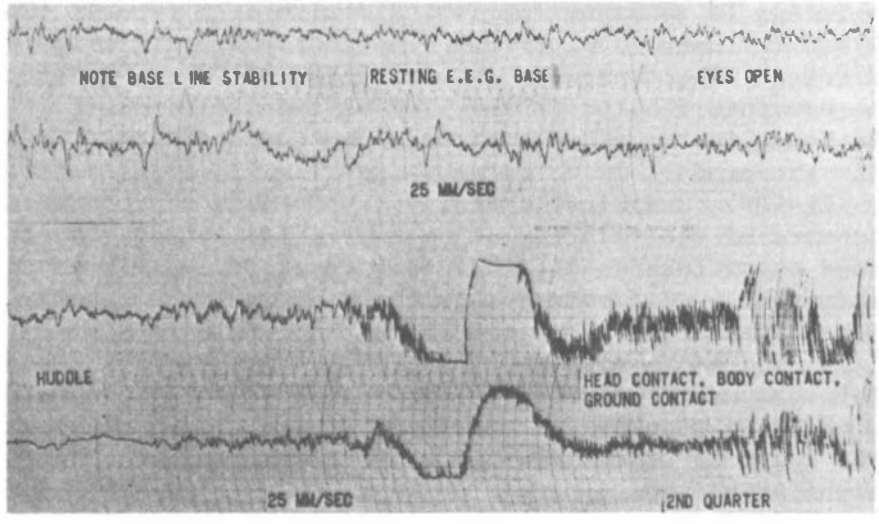

FIGURE 4
Top, normal background rhythm of EEG. Bottom, artifact of EEG at moment of impact.

PHASE III: TELEMETERING OF COMPLEX DATA

This undertaking, fraught with many difficulties since its inception in 1961, has of necessity developed in sequential stages to its final phase. Two separate telemetry systems, one for impact and the other for EEG, would interfere with each other's signals, causing "cross-talk" artifacts. In addition, with the data recorded on two separate galvanometers, with paper speeds of 250 mm/sec for impact and 25 mm/sec for EEG, it would be impossible to synchronize the two recordings accurately. The high paper speed needed to record impact required a very sensitive galvanometer, which used a light beam to register on photographic paper. This recorder, however, produced a tracing that could not be photographed.

Our basic-research project has resulted in the accumulation of a great deal of technical data from the very vigorous environment of the football field that had never before been available. The opportunity to collect these data is afforded only during a short season each year from a single instrumented player, and the harvest is subject to many unpredictable variables.

With the experiences gained in earlier phases, the project was now ready for its final stage of development. A single telemetering system was required that would transmit six channels of information to a special galvanometer in the press box capable of producing records suitable for photographing.

What force data should be measured, and from what part of the player's head? Accelerations are certainly important measurements of impact, but, when the mass involved is unknown, force has no real meaning. Force may be used to record impact, but the time during which the force is acting is an important variable with each impact. The velocity and the mass involved add important characteristics to recorded force. The three fundamental units of force--length, mass, and time--are interrelated in the various types of impacts. For instance, force is the product of mass and acceleration ($F = MA$), impulse is the product of force and time ($I = FT$) or of mass and velocity ($I = MV$), and kinetic energy is one-half the product of mass and the square of the velocity ($E_k = \frac{1}{2} MV^2$). All these components of impact have significance; the different types of impacts on the football field require different combinations of the fundamental units of force for more accurate understanding.

The helmet that was used consists of a smooth, rigid plastic shell and a system of strap webbings that suspends the shell on the wearer's head. The space of about 1 in. between the head and the shell allows for dissipation of the impact force. The characteristics of the suspension mechanism must permit some stretch of the webbing, to increase the time for dissipation, but must not allow the head to strike the shell. A small two-channel telemetry system was designed

and built to test the efficiency of various transducers in different locations on the player's head. The dynamic structure of the suspension system, its anchoring within the shell of the helmet, and its elasticity and force-absorbing qualities were monitored with a force transducer or strain gauge.

Preliminary engineering tests showed a significant difference between the type of helmet impacts and the forces that are reflected to the straps of the suspension system. The shell of the helmet has a high-frequency characteristic with considerable acoustics and mechanical ringing during impact. In contrast, the webbing within the helmet, when fitted against the player's scalp, acquires a low-frequency, damped oscillation with extended duration of forces.

We decided to cement a force transducer to the suspension system of the helmet at each of the three popular target areas of the head--right frontal and left and right temporal. The force that reaches the head is recorded by measuring the force components at the absorbing mechanism of the helmet. Toward the close of the 1968 season, a completed six-channel telemetry system was tested. It measured the more realistic forces of impact encountered by the football player's head--three channels of impact forces, two channels of EEG, and a spare channel for further development (Fig. 5). Recordings of the telemetered data are synchronized with the isolated action on video tape. The telemetered data are stored on electromagnetic tape and can be reproduced at any time for study.

COMPUTER ANALYSIS OF HEAD IMPACTS

To fulfill our criteria, it was necessary to conceive and design an analog computer that would automatically calculate the force, velocity, and acceleration of impacts that are delivered to the press box in graphic form. The force is calibrated and converted into a current instantaneously proportional to the force. This signal is examined by the first derivative of the computer. As the force is incrementally created, the changing magnitude or increasing force is converted into a velocity measurement. The velocity then corresponds to the rate of change or stretch of the webbing suspension while it is in the process of absorbing the impact. The velocity signal then is examined by the second derivative of the computer and is converted into acceleration. The scientific evaluation of head impact must measure the three fundamental units of force--length, mass, and time. The EEG alone does not provide the necessary reference point, as determined by these earlier studies. The final phase of the study included:

(1) measurement of direct force on the straps of the suspension system;

(2) measurement of time during which the force is acting;

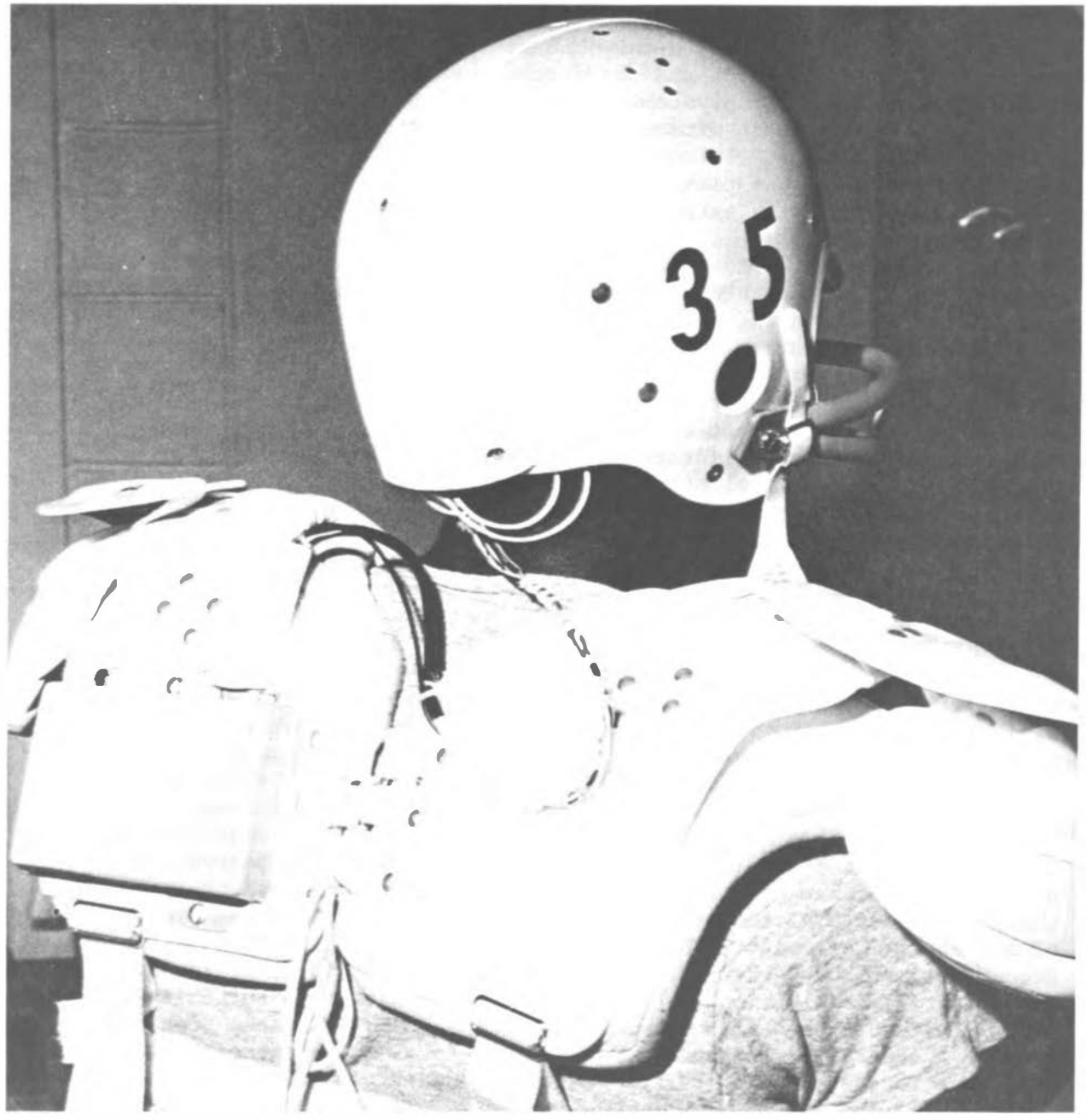

FIGURE 5
Woody Campbell with EEG transmitter compartment on helmet and radio station on shoulder pads for telemetering impact.

(3) measurement of the rate of change of the webbing as it stretches and absorbs the impact in reference to the player's head (the rate of change is therefore the velocity of the webbing as it moves within the helmet);

(4) measurement of the changing force magnitude or acceleration imparted to the webbing, with reference to the player's head and the anchorings of the webbing; and

(5) computation of involved mass from the formula, F = MA.

Those measurements constitute the minimum that will permit an adequate assessment of the relationships of football impacts. With them, one may determine the source of damaging impacts recorded by the EEG (Figs. 6, 7, and 8).

SUMMARY

This progress report covers the developmental stages of a six-channel telemetry system that records impact data from a player engaged in regulation football games. The recorded data include three channels of force received at the player's head and two channels of EEG. The three telemetered values of force are calibrated and converted into a current instantaneously proportional to the force. Following the laws of differential calculus, these signals are examined by the first and second derivatives of a specially designed computer in the press box and are converted to velocity and acceleration measurements of the absorbing suspension system of the helmet. The force values of impacts and synchronized EEG recordings are stored on electromagnetic tape and the data of any single football action can be reproduced and examined with the isolated action of the player recorded on video tape. Because every impact on the field results in a different combination of force components, the opportunity to examine these components in any significant head injury by EEG recordings will be a milestone in solving the problem of head injuries in football.

We wish to acknowledge the assistance of Dr. Frederic A. Gibbs for his interpretation of the EEG recordings, the Wilson Sporting Goods Company for its technical assistance, and the cooperation of the Northwestern University Athletic Department, and especially to Woody Campbell and Bob Olson, the recently instrumented football players. Funds for this project were provided by the Evanston Hospital and the Sports Foundation, Inc.

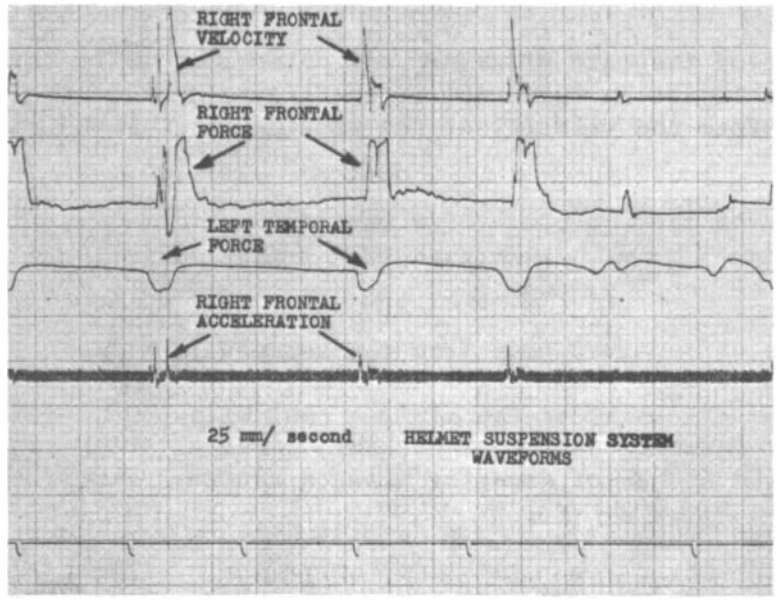

FIGURE 6
Telemetered signals of forces received by suspension mechanism of helmet. Each force produces a different type of graph. Right frontal force has been put through computer to find velocity and acceleration. Paper speed, 25 mm/sec.

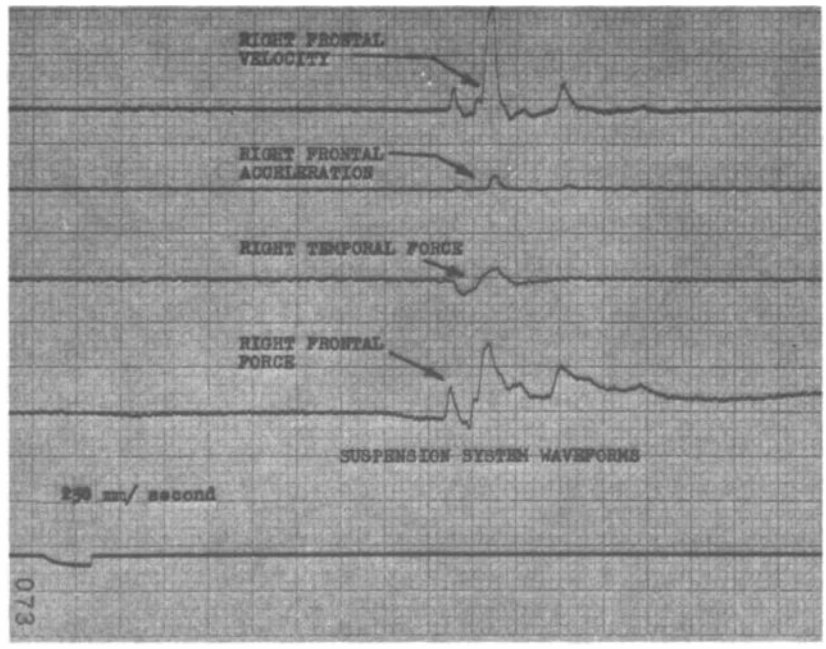

FIGURE 7
Telemetered signals of forces received by mechanism of helmet. Right frontal force has been put through computer to find velocity and acceleration. Paper speed of 250 mm/sec shows more detail than the 25 mm/sec of Fig. 6.

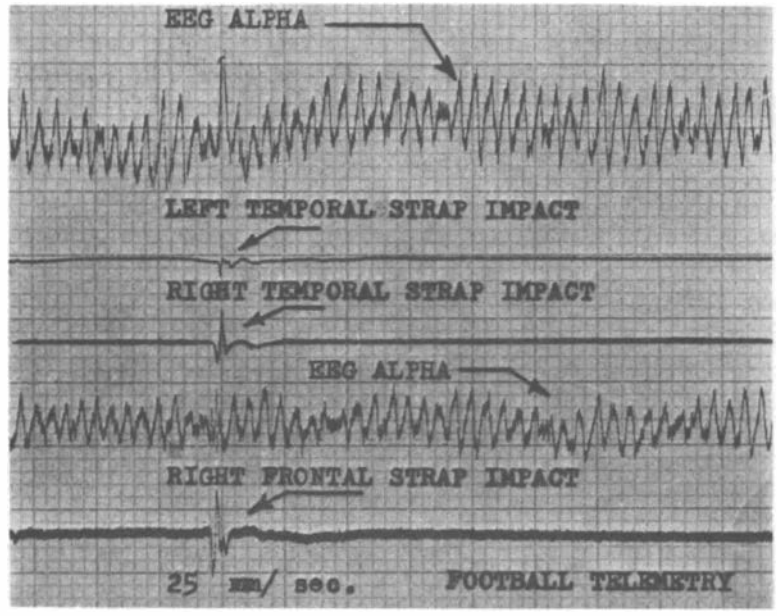

FIGURE 8
Simultaneous telemetering of two channels of EEG and three channels of impact, showing impact waves and their effect on EEG tracings.

INFLUENCE OF PHYSIQUE AND BODY SIZE ON PERFORMANCE

ROBERT M. GRUENINGER

The topic of size, physique (i.e., body build or shape), and performance is very broad. Hence, I shall be able to mention only a few highlights, with some new, interesting, and, I believe, important results that have practical applications, not only in professional football, but also in football as played by high-school boys and college men. Some of the recent results should ultimately redound to the benefit of the general public's health, especially in regard to real or imagined "weight problems."

It can be shown that body size and shape do have a significant influence on individual performance. The three-dimensional "mound diagram"[3] in Fig. 1 illustrates how performance--when it can be directly measured--is quantitatively related to Wetzel grid ratings of body size and shape, as determined from a contestant's height and weight.

Although this procedure was originally worked out by 1940 to evaluate the growth of babies and children,[7-9] it is particularly useful in our field as a classifier of the largest athletes, because it furnishes standards for body volume, density, and composition and energy requirements of adults, as well.[11] It yields values for body volume and density that agree, for practical purposes, extremely well ($r = 0.996+$) with results obtained by chemical analysis, underwater weighing, or K^{40} counting of embryos, infants, and adults.[10] Of particular interest is the fact that the latest (seventh) revision of the Food and Nutrition Board's manual, *Recommended Dietary Allowances*,[6] published by the National Academy of Sciences, tabulates values for daily caloric intake that are statistically identical, for the heights and weights specified over a 20-year postnatal age range, with those first proposed in the form of Wetzel's grid some 30 years ago; the mean difference, in fact, amounts to only 13 kcal/day.

For these basic reasons, but also because of my own long experience, I have full confidence in this objective approach to classifying athletes of all ages and to appraising their performance in terms of grid ratings.[4]

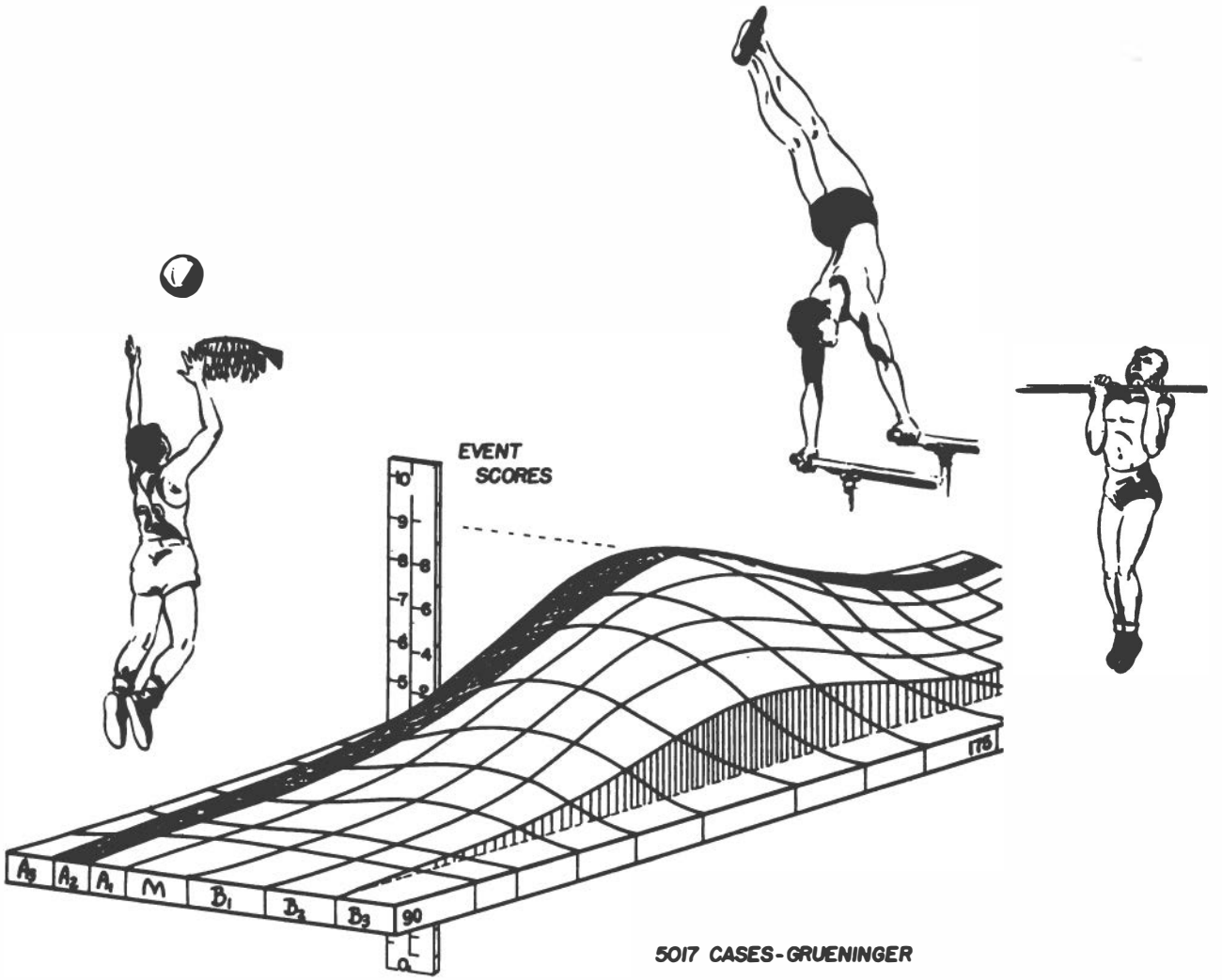

FIGURE 1
Three-dimensional "mound diagram" schematically representing how physical performance tends, in general, to vary with Wetzel grid ratings of body size and shape, as measured in levels (. . ., 90-175, . . .) and channels (. . ., A_4, A_3, . . ., B_3, . . .).[1]

In general, performance scores increase with increasing body size, expressed as grid levels; e.g., larger boys can chin more times than smaller boys, but only up to some maximal size, after which the number of chins diminishes. Physique or body shape, going from channel A_3 to M and over to B_3 (Fig. 1), has a similar limiting influence 90 deg out of phase, as the engineers describe it.

The actual shape of the "mound," of course, varies with different events, as shown by the peak values in Table 1 for world or Olympic champions and college and high-school athletes.[3] Evidently, differences in the size and shape of athletes, ranging from shot-putters down to the smallest marathon and cross-country runners, are undeniably associated with differences in physical and athletic performance. Although all or almost all other factors are favorable, top-quality performance cannot be counted on in the absence of some highly specific and preferential combinations of size and shape, as, for example, in the cases of Roger Bannister and Jim Ryun, two under-4-min milers.

To illustrate more explicitly how size and shape and their components are related to individual body composition and, hence, how all are in turn related to performance, let us examine the flowchart in Fig. 2. First, size and shape (as determined from height and weight) lead to considerations of body volume and density. The diagram next indicates, however, that we must not expect that a given height and weight will always lead, via size and shape, to a single value of volume or density. In fact, for a given height-weight-size-shape input, we shall, in general, meet three types of volume distribution, depending on the relative amounts of fat, water, and residual solids that compose the human body. Thus, in addition to an athlete's size and shape, we must also take account of his body composition if we are to help him to achieve the full performance that he is structurally capable of. (There are, of course, other factors, such as age, experience, intelligence, aptitude, incentive, and desire. But, important and decisive as each of these may be, I must pass them by here.)

With special reference to professional football, let us next consider (Fig. 3) the scattergrams of selected playing positions--DT, TE, DE, etc.--as plotted in a Wetzel grid size-shape field. Their general location is northwest of the spot (+) at which the so-called average adult male would be found, with the Astronauts midway between. Professional football players, taken as a group, tend to be 20-40 levels above the size of the general population and considerably more stocky by about three to five channels of physique than a medium-built adult. Characteristically, they occupy a band at the left of the broad physique spectrum, considerably farther removed from the center channel than the equally typical location of professional basketball players at the far right, who are just as large but much more slender. Here is sufficient evidence that physique (shape) plays a role in physical performance.

TABLE 1
Favored channels and levels in track and field events

Event	High school	College	World or Olympic champions
Shot put	A_2-191	A_2-198	A_3A_2-250
Decathlon	-	-	A_1A_2-192
Discus	A_2-190	A_2-196	A_4-215
Javelin	-	A_1-186	-
100-yard dash	A_1-170	A_1A_2-180	A_1-181
200-yard dash	A_1-170	A_1-180	A_1-181
Low and high hurdles	A_1M-169	A_1M-180	M-185
Running broad jump	A_1-175	A_1M-177	A_1-181
Pole vault	A_1-172	A_1-177	M-182
880-yard run	M-166	M-177	A_1M-156
High jump	M-170	B_1-177	A_1-196
High jump	-	-	M-198
440-yard run	M-170	M-175	M-177
Mile run	M-163	M-174	B_1-174[a]
2-mile run	B_1B_2-153	M-173	-
Marathon	-	-	A_1M-157
Cross-country run	-	-	B_3-145
Swimming:			
100-meter	-	-	A_2A_3-179
150-yard medley	-	A_2-205	-
Diving	-	A_2A_3-161	-
Women's swimming	-	A_1M-146	-
Women's gymnastics	-	A_1-126	-
Russian basketball	-	-	A_1A_2-244
American basketball	-	-	A_1M-214
American basketball	-	-	B_2-201
Miscellaneous:			
Smallest dwarf: 8 years; size of 3-month infant			B_1B_2-(-100)
Tallest man: 8 ft 10½ in. (271 cm).			A_1M-298
Heaviest man: 1047 lb (476 kg)			A_{34}-355
Heaviest woman: 772½ lb (351 kg)			$A_{33.5}$-324

[a] Time, 3 min 54.5 sec.

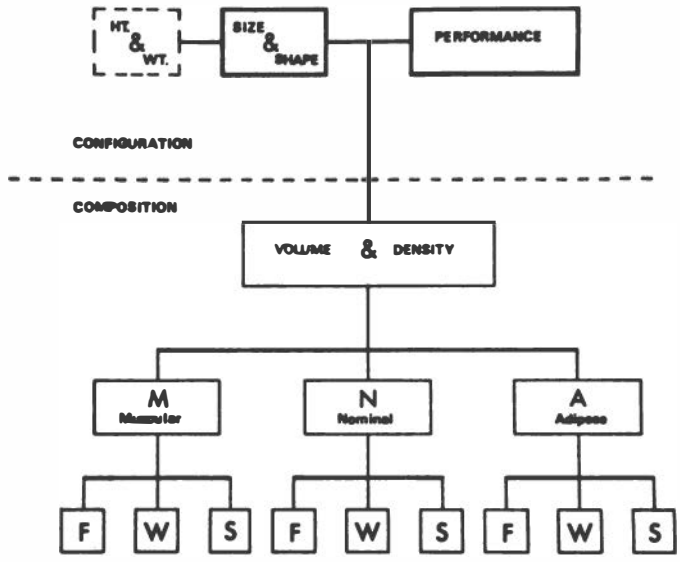

FIGURE 2
Flowchart interrelationships between size, shape, performance, and body composition for three basically distinct types: muscular, nominal, and adipose.

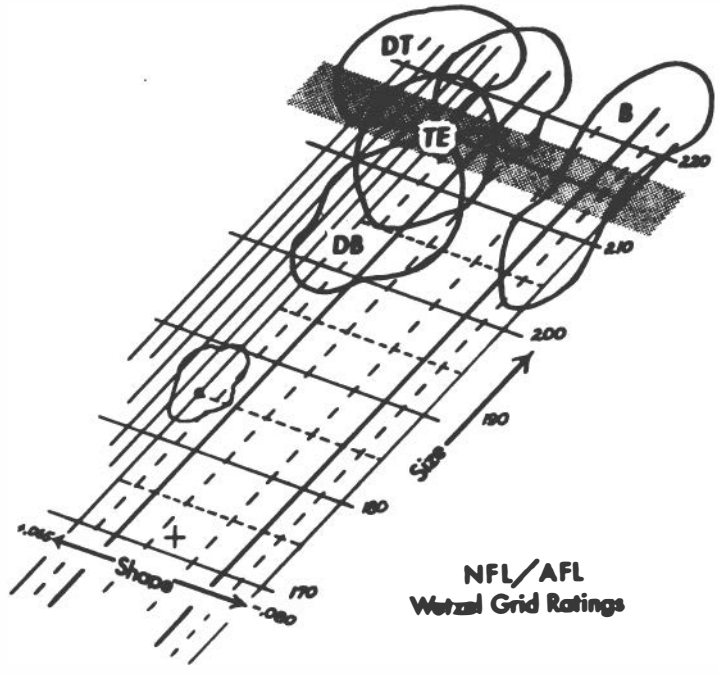

FIGURE 3
Typical grid locations of size and shape for National and American Football League players (DT, TE, DB, . . .), compared with professional basketball players (B), with the "average male adult" (+), and with Astronauts (dot about midway between).

Some overlap is seen in the football region. This suggests that a man so located might very well play two or more positions (other factors being equal). However, for a given team position, our experience indicates that chance will favor the man whose size and shape most closely approximate the position averages.

Table 2 shows how size and shape tend to vary from one specific playing position to another, and how surprisingly small are the differences between the National and American Football Leagues in average position values. Although the corresponding variabilities (standard deviations) are not included in this table, it should be stated that they, too, are essentially the same in the two leagues for each position. The overall uniformity displayed in Table 2 is all the more remarkable when one considers that performance in football is defined and categorized here indirectly (i.e., in terms of playing position), in contrast with direct scaled measurements of performance that are made on track and field athletes.

TABLE 2
Mean sizes and shapes of 640 NFL and 405 AFL Football Players[a]

Position	No. players		Size		Shape		
	NFL	AFL	NFL	AFL	NFL	AFL	Channel
LB	82	56	214.2	213.0	9.35	9.27	A_5
DB	96	61	195.8	194.9	4.95	5.14	A_3
DT	53	33	225.2	226.5	10.38	11.97	A_6
DE	49	29	222.6	223.4	9.41	10.00	A_5
E	40	28	200.2	202.1	4.92	4.82	A_2A_3
T	51	28	223.8	225.3	10.00	10.63	A_5A_6
G	49	35	220.6	222.0	10.94	11.29	A_6
C	24	13	219.5	218.7	10.60	9.34	A_5
TE	27	12	213.5	212.9	7.70	7.35	A_4
QB	37	27	200.4	199.7	5.47	5.10	A_3
RB	83	50	204.6	205.4	7.93	8.61	A_4A_5
FL	27	22	197.9	192.7	4.58	5.03	A_3
K	14	11	197.5	205.5	6.04	7.71	A_4
P	8	0	196.8	---	7.09	---	---

[a] Wetzel grid ratings.

All the results in Table 2 are "official," in the sense that they have been derived from the data on 640 NFL and 405 AFL players on the 1968 rosters that were furnished by Central Headquarters (Commissioner's Office) and that were presumed, accordingly, to represent "trained down" or "properly conditioned" values. For the computation of size, shape, and body volume and density (and some items of body composition), a Univac 1108 program that Wetzel developed for embryos, babies, children, and supersize adults (750 lb or more) was used. As the entries in Table 2 show, there is clearly no such thing as an "average football player." But, as we view the 14 playing positions distributed among more than 1000 professional players, there are as many different (and characteristic) averages as there are positions. On the basis of previous studies of various sports, there was never any doubt as to whether we would find significant differences in size and shape among professional football players in different positions, but I had not expected that the two leagues would prove to be so strikingly alike at each position.

We are compelled to conclude that for the dozen or so playing positions, there is no statistical or practical difference in body size and shape of players between the two major football leagues. Moreover, considering the wide variations among adult men at large, one must regard the virtual interleague identity so evident in all the playing positions as nothing short of phenomenal.

Whatever differences do exist must be attributed solely to differences in the performance demands of the various playing positions themselves. It would appear that a kind of natural selection--based essentially on a man's size and shape (i.e., on his physical makeup)--is at work, and not personal whim or other fleeting influences. In any event, from the statistical standpoint, we may look on these two sets of data as being samples and subsamples that have been drawn from a very well-defined, physically homogeneous population of young men who habitually manifest equally homogeneous position, team, and game performance on the field.

A graphic comparison of the size-shape relationships is shown in Fig. 4. The complete independence of size and shape, when expressed as Wetzel grid ratings, is indicated by plotting size as vertical rectangles and shape as horizontal bars. No distinction is made between the NFL and AFL values, because the actual differences are too small to be seen on the chart. Different positions can be seen to be clearly associated with, and apparently call for, distinct combinations of size and shape.

In addition to size and shape, a factor that should be discussed in connection with performance is body composition. It, too, is rather complicated, but a few examples will readily show how such information can be very useful.

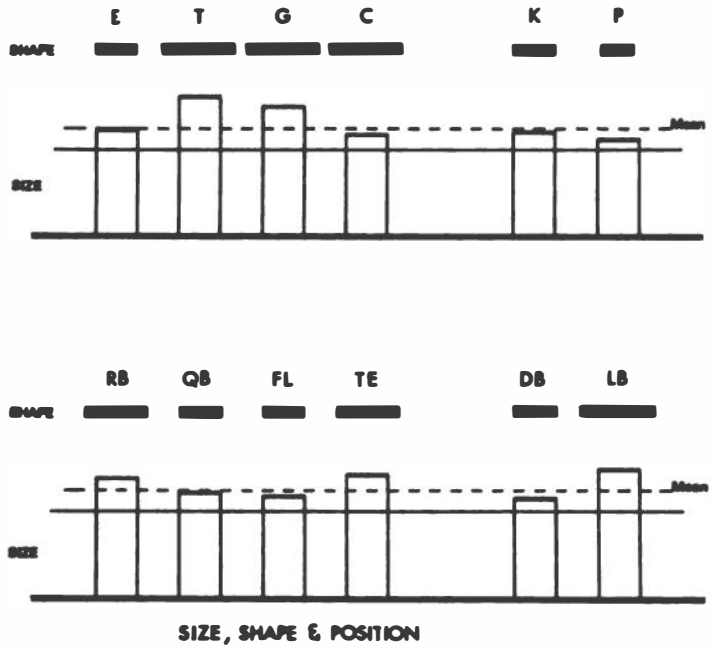

FIGURE 4
Positional differences in body size and shape of 1045 professional football players in 1968 (see Table 2).

In Fig. 2, we saw that, at any given weight (and with no serious pathology), three basically different types of composition will be encountered: predominantly muscular (M), nominal* (N), and predominantly adipose (A), depending on an individual's proportions of water, fat, and nonfat or residual solids.

In Fig. 5, M represents an actual member of the San Francisco 49er team whose body densities were measured by A. R. Behnke, U.S.N., in 1942.[1] The player's position was not stated, but his size and shape ratings were almost identical with those of the right defensive back of the Baltimore Colts of 1968. Behnke's player (M) had a measured body density of 1.0735, which is high, compared with the 1.063 that is conventionally regarded as the average for normal young adult males; it is still higher when compared with the Wetzel grid standard density of 1.0490 for a man of the size and shape of those two players, namely, A_3-205. As a result, the corresponding volume index, λ, was 0.977, which indicates definite muscular predominance.

The center block (N) in Fig. 5, for a player of identical weight-height-size-shape ratings, but of "nominal" composition, indicates that volume will be slightly greater, fat about twice that of M, and water only 5 or 6 percentage points less. The volume index, λ, is 1.00 even.

* That is, healthy, normal, etc., with only small, negligible, or trivial variations.

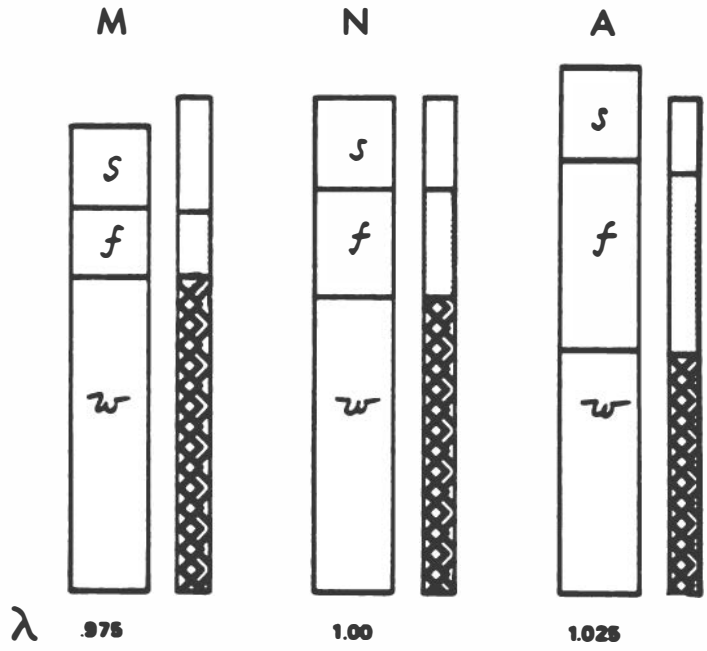

FIGURE 5
Relative amounts of water, fat, and residual solids in men of identical body configuration (i.e., same height, weight, size, and shape), but of three distinctly different volume distributions--M, N, and A, typifying muscular and adipose predominances (M and A) in contrast with no preponderance, i.e., nominal (N) or standard body compartmentalization.

The last block (A) of Fig. 5 represents a man of the same weight-height-size-shape ratings, but with fat increased to 33% and water down to 48% of weight; λ is 1.025.

According to the usual weight-height tables, each of the three men (M, N, A), at $74\frac{3}{4}$ in. (190 cm) and 210 lb (95 kg), is about 35 lb (16 kg) or some 20% "overweight," a figure that is often presumed to represent just "simple obesity" and to call for prompt weight reduction of, say, 15%-17%. What is also often assumed in such cases is that weight loss will be entirely fat loss. And it is further supposed that such loss will benefit health, as well as physical performance. That is possibly so.

But it is certainly not so in the case of football player M, who, by actual measurement, had only 11% fat and could not possibly lose 16% fat. If he were advised to reduce by a few pounds--say, 5 or 6--would it be fat alone or some valuable muscle, too? And would it be at no risk--especially if he feels well at 210 lb? Player N has 20%-21% or about 44 lb (20 kg) of fat, which would leave him with 9 lb (4 kg) of fat after all-fat reduction, or only 4% of fat in his body, which is rather low for being "normal." Player A, however, would seem to have better prospects of being "improved" by fat loss. But does he? His 33% fat gave

him 69 lb (31 kg) to start with and would leave 34 lb (15 kg), or 19.5% of his new weight, after similar reduction. This seems normal, but would actually lack about 10 lb (about 5 kg) of the expected or "nominal" value of 21%, or 44 lb (20 kg), for a man of his size and shape.

The foregoing are by no means extreme examples. They are cited here because the question of whether to advise weight reduction often arises, not only among weight-watchers, but also among trainers and coaches, many of whom seem to believe that "making weight" (to some arbitrary level) will invariably improve performance. On occasion, players have been fined for reporting to camp with what is regarded as excess fat, and others, for perhaps equally tenuous reasons, have been importuned to gain weight. The matter, however, is not as simple as that.

Nonetheless, these problems of "weight-making" and "overweight" do present some of the most frequent opportunities of putting sound principles of size, shape, and body composition to practical and correct use. To that end, one of the first notions that must be abandoned is that every young man (football player or not) between 18 and 30 years old is to be appraised in terms of a single fixed standard calling for a body density of 1.064, with 14%-15% fat and a daily caloric intake of 2800-3000. Like performance itself, fat, density, and caloric requirements depend on size and shape, with the result that 20%-25% is acceptable, for example, as long as it conforms to size, build, and M-N-A rating.

As an educator who has long been interested in physical fitness and performance, I would venture the view that men already attuned to the special demands of professional football will rarely, if ever, require a 15%-30% reduction in weight, in the hope of improving their performance, simply because they are alleged to be that much "overweight." Hence, my habitual warning to coaches and trainers: Beware of ordering weight reductions in the belief that only fat will be lost, especially if there is comparatively little fat present initially, as there is, for example, in almost every "natural athlete."

Figure 6 shows numeric ratings for the Baltimore Colts defensive team in their 4-3-4 array. That the largest players (levels 220-239) and stockiest (8.9-11.5) are in the front line is no surprise; it merely confirms what every fan knows. Notable, too, is the 10-level dropoff in the 2nd and again in the last line of defense. Comparison of these values with the league data of Table 2 yields many interesting similarities.

Such studies and comparisons of team and individual size-shape and composition ratings not only are worth studying on their own, but constitute a fundamental step toward the ultimate appraisal of par or subpar performance with which they are so directly linked.

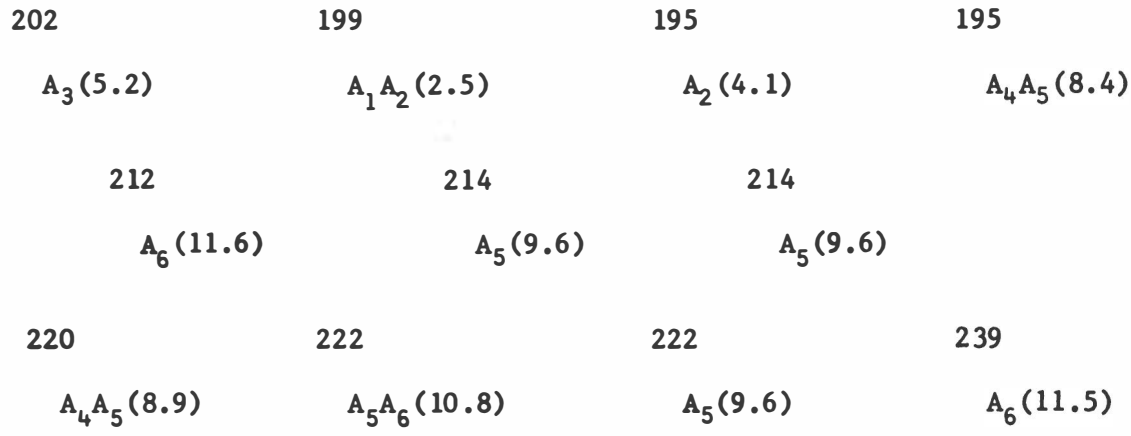

FIGURE 6
Size and shape (level and channel) ratings of the 1968 Baltimore Colts defensive team in a 4-3-4 array with the front line at the bottom. The right cornerback (A_3-202) matches the configuration of Behnke's 49er (M, in Fig. 5) very closely.

In summary, I would emphasize a few important points:

(1) Abundant evidence from a broad field of athletic activities has clearly shown that body size and shape, as expressed in terms of Wetzel grid ratings,* are directly, and often preferentially, related to the quality of physical performance that individual participants are capable of displaying. The same is true of professional football players, among whom characteristic patterns clearly indicate that overall performance depends on size and shape, among other things.

(2) A third component, body composition, is also to be included in appraisals of fitness, particularly to take account of variations in body density and thereby of fat burdens, which may differ widely even among men of the same weight.

(3) Control of size, shape, energy, and composition is definitely indicated to minimize the occurrence of the football version of getting round plugs into square holes--assigning men to playing positions that are inappropriate to their physical characteristics.

(4) Extended studies of the type described here could help to clarify the problem of injury susceptibility, which, in any event, must take due account of differences in size, shape, and performance.

(5) Arbitrary insistence that players reduce weight, on the assumptions that fat will be lost and that performance will be improved, is not sound. When advice on this matter is necessary, it should be based on a full medical review, including appraisal and evaluation of the fat and muscle content of an individual player's body.

* I.e., channels and levels.

(6) Professional football players belong to a very select group of people. The game they play calls for much discipline and dedication. They are large and stocky, they have high body density and muscular predominance (just short of typical weight lifters), their bodies have low fat content, and they have great speed, agility, and endurance. Among them, we find a dozen different specialists on a single team. To classify them incorrectly is to impair not only the individual players' efficiency and development, but those of a team as well.

ADDENDUM

A few additional data are important for comparative purposes, particularly in connection with secular trends in the size and shape of men who play football. For example, a study of my own,[4] covering 2198 college players between 1943 and 1948, produced the results shown in Table 3. It is evident that collegiate players of that era, if not as large and stocky as today, were certainly not as small as many of today's observers are inclined to believe they were. That is also indicated by the data of the University of Michigan Rose Bowl starting team of January 1948. Figure 7 shows precisely the same front-line to backfield diminishing gradient in size (10 levels) as is displayed among the professional teams today (such as the Baltimore Colts of 1968, in Fig. 6).

TABLE 3
Physique (shape) and size (level) characteristics of U. S. collegiate football teams (1943-1948)[a]

Position	Shape (channel)			
	A_5A_4	A_3A_2	A_1	M
Tackle	205	197		
Guard	196	190		
Center		190		
End			190	
Back		185	180	180

[a] Derived from Grueninger.[4]

End	Tackle	Guard	Center	Guard	Tackle	End
A_1-199	A_3-197	A_4-192	A_1M-193	A_3-190	A_3-194	A_1-192

Quarterback

A_4-188

Halfback Halfback

A_2-192 A_2-182

Fullback

A_2-189

FIGURE 7
Physique (shape) and size (level) characteristics of University of Michigan Rose Bowl champion team (1948). (Derived from Grueninger.[4])

The somewhat earlier observations of Behnke *et al.*[1] in 1942 on the San Francisco 49ers yield the team averages listed at the top of Table 4. These are followed by the grid ratings of the 49 athletes of Novak *et al.*[5] in five different sports (including 16 football players) at the University of Minnesota in 1966. The final entries in Table 4 represent the highly successful San Diego State College team and were calculated from measurements furnished me by Carter,[2] which are almost identical with those for the 49ers a quarter of a century earlier. Average size (both college and professional) has increased by only about 10 levels (allowing for the differences between college men and the slightly older professional players), whereas average shape (or physique) has remained virtually constant at A_4A_5 (7.0) over the same period.

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TABLE 4
Secular size-shape grid ratings, 1942-1968

Observer	Team	No. persons	Year	Size (level) x 10^{-2}	Shape (channel) x 10^{-2}
Behnke *et al.*[1]	San Francisco 49ers:	25		200	A_4(7.0)
	High density	19	1942	195	A_3(6.0)
	Low density	6	1942	213	A_5A_6(10.2)
Novak *et al.*[5]	University of Minnesota:				
	Football	16	1966	206	A_4A_5(8.1)
	Baseball	10	1966	192	A_2(4.4)
	Swimming	7	1966	186	A_1M(2.5)
	Track	9	1966	177	M(0.3)
	Gymnastics	7	1966	173	M(0.2)
Carter[2]	San Diego State College:				
	Offensive line	10	1967	212	A_4A_5(8.3)
	Defensive line	8	1967	207	A_5(9.2)
	Offensive backfield	12	1967	195	A_3(6.0)
	Defensive backfield	5	1967	190	A_2A_3(4.7)
	Team	35	1967	202	A_4(7.2)

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STRENGTHENING, CONDITIONING, AND THE PREDICTION OF INJURY

JOHN B. KRESS

My purpose here today is to report what we do at the U. S. Military Academy, to prevent injuries in our program of physical education, intramural athletics, and club activities. Our injury problem is perhaps greater than that of any other college or university in the United States, primarily because our mission, training objectives, philosophy, and policies are directed toward the development of young officers capable of leading troops under extreme and hazardous wartime conditions. We believe that each Military Academy graduate must be physically educated and conditioned to accomplish this leadership task, and that there is no better way to prepare him physically for his chosen profession than through varied, rigorous activities of the type that will promote the continuous development of strength, endurance, coordination, agility, flexibility, and power. We are firmly convinced, also, that the athletic field provides the most exacting laboratory in our situation in which to develop leadership qualities, and that the disciplines of sports and games contribute immeasurably to the development of a capable West Point product.

Our population approximates 3800 cadets ranging in age from 17 years 6 months to 23 years; in weight, from 125 to 230 lb (57 to 105 kg); and in height, from 5 ft 4 in. to 6 ft 8 in. (163 to 203 cm). On entrance into West Point, many of our cadets are superb athletes and others are nonperformers. Our philosophy and policy establish that every cadet will compete against his peers in either an intercollegiate, an intramural, or a club activity during every athletic season--fall, winter, and spring--of his 4 years at the Academy. Except in boxing and wrestling, little consideration is shown for the vast differences in height, weight, physical maturity, and sports experience. With over 400,000 man-hr of participation each year under these conditions, it is plainly evident that we must consider each man-hour as an hour of exposure to injury. We know our problems, and we realize that it is incumbent on us to control the rate and severity of injuries. We state our purpose very simply: to prevent injuries and to provide the best of care and treatment for injured cadets. To meet the challenge expressed in this purpose, we have for many years investigated the many

facets of our program; and I shall attempt to give a general overview of the practical application of some of the aspects of our efforts in the area of injury prevention.

PREVENTION OF INJURY AND REINJURY

We are convinced that we must know the causes of injury if we wish to prevent them. Our policy requires on-the-spot reporting of all injuries, with special attention to cause. How was the cadet injured? Why was he injured? What can we do to prevent similar injuries in the future? On the basis of our study of each injury, we make changes in rules, sizes of fields, officiating and coaching techniques, etc.

Investigations of all knee injuries over a period of several years confirmed a prior conviction that many knee injuries that occurred every year in our program were actually reinjuries of pre-Academy origin. These findings led to the initiation of a pre-Academy injury survey conducted by the office of physical education, and a concurrent study of the medical records of all incoming cadets by the orthopedic staff of our hospital to identify all new cadets with histories of pre-Academy knee injuries. With this information on hand, we initiated a program of injury screening in which we used several diagnostic techniques, including manual examinations for joint stability and muscular-strength evaluations, to determine the physical status of previously injured new cadets. Originally started to offset the reinjury cycle among new cadets, this screening program proved so effective that it was established as a follow-up procedure for all injuries to the knee. We now screen all cadets who incur knee injuries before we allow them to participate in contact sports. If a knee is not ready for contact activities, we assign the cadet to a knee-development squad and to a noncontact activity. We have found that this restrictive procedure protects cadets during a critical reinjury period and, at the same time, permits them to participate with a lessened chance of reinjury.

When we discovered that many cadets entered the academy with residual weaknesses originating from pre-Academy traumatic experiences and that these weaknesses, if left uncorrected, were major causes of reinjury, our next approach was to establish a program of specific conditioning for the knee joint in an effort to abort the reinjury cycle. Three years of continuous research followed. In a series of studies, we assessed the value of specific exercises designed to develop the strength and endurance of the musculature supporting the knee joint and studied the effects of specific conditioning in the prevention of original or recurrent knee injuries. The annual and composite results of this combined study strongly indicated that specific conditioning is most effective in reducing both original and recurrent injuries. A program of knee-strength development was recommended and accepted by the director of physical education as a permanent function

of his department. The results of this work during last fall's athletics season show the effectiveness of this program.

Forty new cadets with histories of pre-Academy knee injuries were assigned to a program of knee-strength development during the summer of 1968. Thirty-six of these cadets had histories of surgical repairs, two had previous dislocation of the patella, one had chondromalacia, and one had recurrent locking of the knee. In each of these cadets, there was a significant difference--10 lb (4.5 kg) or more--between the two legs in strength of the quadriceps or hamstring or both on the day of entrance into the Academy. These new cadets entered West Point on 1 July, and the program started on 8 July. Within the first 8 days of normal training, seven of the cadets were reinjured, and three of the reinjuries required surgery. Of the original 40 new cadets, therefore, only 33 were able to participate in the program. The program continued for 7 weeks and was conducted 5 days each week in lieu of mass athletics and corps squad screening. Cadets, on the average, were able to complete 6 weeks of concentrated work. One week was spent in the field for military indoctrination.

Among the 33 cadets who completed the program, two injuries were recorded during the fall athletics season; one was original, and the other, recurrent. The original injury required only 3 days for recovery. The recurrent injury required 20 days. Both injured cadets completed the fall athletics season without reinjury. No playing restrictions were placed on these 33 cadets. Seven of them competed in intercollegiate football; four, in intramural football; one, in intercollegiate soccer; and seven, in intramural soccer. The other 14 competed in either intramural track, triathlon, lacrosse, or tennis.

In 1965, we studied the effects of stool-stepping in the strengthening of the musculature supporting the knee joint. We found that, in the early stages of conditioning (during the first 3 weeks), 5 min of stool-stepping was as effective as isometrics or isotonics. The stool-stepping technique, however, appeared to lose its effectiveness, insofar as strength gains were concerned, once the athlete was conditioned to lift his body weight consecutively for 5 min. Longer durations of stool-stepping produced no significant gains in strength. No effort was made during this investigation to add weight to the body; but we believe that it is reasonable to assume that stool-stepping with weight added to the body would result in increased strength. As a result of this experimentation, stool-stepping is used as a conditioning agent during the first 3 weeks of our intramural season.

In another investigation, we studied the effects of the removal of heel cleats from intramural football shoes in the prevention of knee injuries. As a result of a 4-year investigation that included the use of cleats, flat heels, short front cleats, and disk heels, we eliminated the use of heel cleats completely from our intramural football program.

How effective these practical aspects of injury prevention are can be told only by examining the record. This fall, with 572 players engaged in intramural football for a total of approximately 10,000 man-hr of participation, we had a total of 33 knee injuries, only one of which required surgery. The total time lost from practice sessions was 93 hr, or an average loss of fewer than three 1-hr practice sessions per injury. The total game time lost was 61 hr, or an average of fewer than two games per injury. We are well aware that the pendulum may swing in the direction of an increase in the number of injuries, but we believe that continued efforts in the field of injury prevention will enable us to continue to reduce the number and severity of injuries.

Although I have discussed chiefly the prevention of knee injuries, our efforts are by no means restricted to knees, but include injuries of all major joints.

PREDICTION OF INJURY

In cooperation with the orthopedic staff of the U. S. Army Hospital at West Point, we are working on a project in the prediction of injuries. This project is still in an embryonic stage, but we are gathering useful data. Some of our observations follow.

(1) The presence of residual muscular weaknesses resulting from traumatic experiences leads to susceptibility to reinjury. These weaknesses can be scientifically evaluated.

(2) Various forces--stress, compression, tension, and strain--may act directly or indirectly on joint mechanisms in habitually poor postures, and faulty postures may have a direct bearing on performance and on the incidence of pain and disability.

(3) Favoring an injured limb may result in faulty posture, which, if it persists and is left uncorrected, may play a major part in the reinjury cycle.

(4) The incidence of lateral pelvic tilts among young men of college age is so great that, statistically, a lateral pelvic tilt with the right hip prominent may be considered normal for right-handed young men.

(5) Muscular imbalance, especially in the gluteus medius, is intrinsically associated with most lateral pelvic tilts. In the etiology of sports injuries, this muscular imbalance and the resulting instability of the pelvic girdle are associated with pain and disability in the hip region, the knee, and the lower back. The mechanics of the lower back cannot be divorced from the mechanics of the pelvis and legs, nor can the mechanics of the legs be separated from the mechanics of the pelvis and hip.

(6) Although flexibility is a desirable component of physical fitness, too much flexibility, as evidenced in hyperextensibility of the elbow or the knee, may be considered a precursor of laxity in all joints and of subsequent subluxations and dislocations.

(7) Restricted flexibility, as evidenced by heavy or tight musculature and restricted joint action, may serve as a valuable diagnostic observation in the prediction of muscular and tendinous injuries.

(8) Athletes who are bow-legged, either structurally or posturally, may not be prone to knee injuries, but may suffer severe pain and subsequent handicap because of either a contracture or stretch weakness of the piriformis muscle in its relationship with the sciatic nerve.

(9) Two exercises most often neglected in the rehabilitation of injured or surgically repaired knees are hip abduction and flexion of the thigh on the hip. Manual or instrument tests of the abductors and the flexors should be given to ensure the complete reconditioning of the leg musculature. Weakness in these areas may serve as a predictor of injury.

(10) Recurrent subluxations and dislocations would be less frequent if they were treated as fractures, insofar as recovery time and reconditioning time are concerned. The practice of returning the symptom-free athlete to a competitive status is, in our judgment, a serious mistake. Repeated subluxations or dislocations increase the probability of recurrences.

(11) Controlled tests, such as traversing an overhead horizontal ladder or releasing one hand from a two-handed suspended or hanging position on a horizontal bar, are not useful as predictors of subluxation or dislocation of the shoulder.

(12) A stretch weakness of the lower section of the rectus abdominis usually accompanies an anterior tilt of the pelvis, and leg-lifting exercises from a supine position may increase the weakness. Leg-lifting exercises are contraindicated for athletes with lower-back conditions. Abdominal tests are considered necessary in prediction of injury.

(13) In many instances, the so-called weak back is one in which the low back muscles are actually tight, and not weak; painful lower-back conditions often result from contractures of these tight muscles, and hyperextension exercises are contraindicated in these cases.

(14) Observation of the position of the kneecaps in standing may be important in predicting injury. If they face inward, rather than straight ahead, it may be due to inward rotation of the entire leg from hip to foot, in which case there would be an indication of weakness in the outward rotators. Or it may result from rotation or torsion of the lower leg in relation to the femur--often seen in hyperextension of the knees. It may produce a stretch weakness in the popliteus muscle.

SUMMARY

We are fortunate at the Military Academy. We have opportunities to work with a fine group of young men, most of whom believe thoroughly in developing and maintaining a high degree of personal physical fitness. We try to imbue them with a philosophy of fitness that stresses preparedness to meet the unusual demands that might arise during a lifetime career in the Army. We test them, for diagnostic and motivational purposes, at least semiannually in tests of endurance, speed, strength, coordination, and agility. We design our programs with confidence that our cadets are fit to play the game, and we do not rely on the game to develop fitness. We are indeed fortunate, because fitness for competition is, we believe, an essential requirement of any program that stresses athletics for the physical welfare of the participants.

PHYSIOLOGIC BASIS OF PERFORMANCE AND PHYSICAL-CONDITION TESTING

HOWARD G. KNUTTGEN

In discussing the physiologic basis of performance, it is necessary to consider all the factors that may interact. For the purpose of analyzing performance in the game of football, I would like to set forth a system of categorization of the many factors involved. This system involves eight categories: (1) skills, (2) knowledge of game and strategy, (3) psychologic state, (4) size and proportions, (5) strengths of movements, (6) speeds of movements, (7) energy sources available, and (8) energy-release processes (aerobic energy release and anaerobic energy release).

The first three categories are psychologic, and I will discuss them only briefly. The implications they have for injury are rather obscure, but their implications for performance are important.

Skills involve the coordination of the movements of the various body parts to bring about running, blocking, tackling, passing, etc. Although we sometimes term them "neuromuscular skills," it should be emphasized that we are actually talking about a teaching or training of the nervous system. Muscles learn nothing and cannot act on their own. The magnificent execution of the top-flight athlete is the result of a better-trained brain, spinal cord, and associated nerve cells.

Considering over-all performance in football, a knowledge of the game itself, the team's own offense or defense, the moves to expect from opponents, and the coordination of moves with teammates all determine a player's success and value to the team.

The player's ability to use his skills and his knowledge of the game (together with his more "physical" and "physiologic" qualities) depends heavily on his mental state. The importance of the game, the player's competitive spirit, and the stresses of his life off the football field can certainly prevent him from performing well or, in some cases, cause him to perform far beyond what one would normally expect from him.

Physical size and body proportions are extremely important in performance and probably play an important part in the incidence of injuries in professional football. In general, a good big man is of more value in performance than a good small man. The role of the size of particular body parts, such as the hands of a pass receiver, is fairly obvious.

In addition to a player's size, the strength and speed with which he performs particular movements are crucial. The speed with which an offensive lineman moves at the snap of the ball, the speed of a passer's release, and the speed of a linebacker reacting to a play are but a few examples. The strength with which movements are made is an added factor: the tackling move may be fast but, if it isn't performed with strength (or force), the runner breaks away. I list strengths and speeds as separate factors because research has shown that the strength of a particular movement (e.g., arm flexion) or group of movements (such as those involved in a defensive charge) bears little or no relationship to the speed of performance. I have also put everything in the plural for these two categories because a particular player's ability to perform one movement or set of movements with either strength or speed might have little relationship to his abilities to perform other movements.

ENERGY SOURCES AND ENERGY RELEASE

The immediate source of energy for muscle contraction and, therefore, physical exercise is a high-energy phosphate compound, adenosine triphosphate (ATP). As far as is known, the muscle cell (or muscle fiber) cannot contract in the absence of ATP. ATP depends greatly on another high-energy compound, creatine phosphate, whose breakdown can cause rapid resynthesis of ATP. Both compounds are formed in the cell, and neither requires the presence of oxygen to release its stored energy.

The body cells would soon become depleted of these two compounds if it were not for the foods taken in, principally carbohydrates and fats. After digestion, carbohydrates and fats are delivered to the muscle cells as glucose and fatty acids. Once in the cell, they can be broken down, eventually to the waste products of carbon dioxide and water, with the release of energy for the synthesis of high-energy phosphate compounds.

It has been shown that, in long-lasting continual activity of high intensity, the stored carbohydrate (glycogen) in the muscle cells is very important in competition. Football is not a very long-lasting activity, and it is doubtful whether anything more than a normal mixed diet (appropriate in calorie level) is necessary to enable the best performance. I do not believe that any special consideration should be given at this time to energy sources as a factor determining performance capacity in football.

The source of energy is one consideration; the process of releasing the energy (including the speed of the chemical reactions) is another. We generally divide the cellular processes into two separate but related phases: aerobic and anaerobic. I will define "aerobic energy release" as that which proceeds with the continuous supply of oxygen from the lungs. "Anaerobic energy release" is that which occurs in such quick bursts or at such high levels of physical activity that continuous supply of oxygen is neither involved nor necessary.

Research has shown that it takes at least 60 sec to become completely aerobic in energy release during physical activity and that, if violent physical exercise is confined to 10-sec bursts, the necessary energy can be supplied anaerobically (see Fig. 1). Table 1 shows that distance running depends to a great extent on the delivery of oxygen and to a lesser extent on anaerobic processes, as measured by the oxygen debt. The 1500-m race (approximately a mile) is run on energy that is about half aerobically released and half anaerobically released. Running twice the length of a football field (200 m) is predominantly anaerobic. Therefore, running distances shorter than the length of a football field would usually involve exclusively anaerobic energy release. The training of the oxygen-delivery mechanisms (chiefly the circulatory system) and the measurement of a person's aerobic capacity are probably of little concern in football.

Keeping in mind the data in Table 1 and the research evidence that delivery of oxygen to the muscle is unnecessary in bouts of exercise lasting less than 10 sec, I decided to perform a research project one Sunday afternoon to determine how long a professional football player is active at any one time (or play). With a stopwatch, I timed each play, including kickoffs, punts, etc., in the first halves of both the NFL Pro Bowl game and the similar game of the AFL. The results are shown in Tables 2 and 3. The watch was started on kickoffs when the kicker began his forward movement and stopped when the ball was whistled dead by the officials. Timing of all other plays was from the snap of the ball at center until the ball was whistled dead. Without elaborate discussion, I would point out that the average time that a player was actively performing was 5 sec (5.1 sec for the NFL and 5.0 sec for the AFL). The shortest plays were the extra-point attempts. The longest single play (in duration), a Fran Tarkenton scramble, in the NFL game was 10.4 sec long. The longest play in the AFL game, an intercepted pass, with return, was 14.0 sec long. It seems rather obvious that the game of football is a series of situations in which the energy-release processes are almost exclusively anaerobic, i.e., do not require the delivery of oxygen from the lungs.

TESTING OF PHYSICAL CONDITION

The factors involved in physical condition and, therefore, performance capacity that can be tested include movement strengths, movement speeds, aerobic capacity (maximal oxygen consumption), and anaerobic capacity.

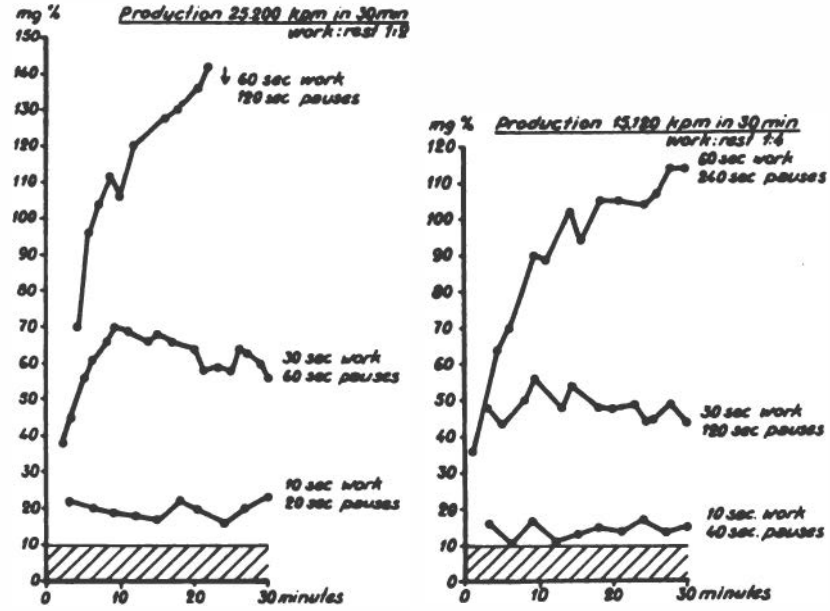

FIGURE 1
Blood lactic acid concentrations for total work production of 25,200 kpm/min (left) and 15,120 kpm/min (right) in 30-min periods through intermittent work (alternating work and rest periods). Blood lactate concentrations are plotted on the ordinates as milligrams of lactate per 100 cc of blood. (Reprinted with permission from Astrand *et al.*[1])

TABLE 1
Oxygen cost of running various distances in competition and relative contributions of aerobic and anaerobic mechanisms of energy release[a]

	Distance run, meters							
	200	400	800	1500	3000 (steep.)	5000	10,000	Marathon
Oxygen delivered from lungs to tissues, liters	1.5	3–5	8–9	16–17	38–40	60–70	135	560
Oxygen debt liters	8–9	11–12	11–13	11–12	8–10	8–10	7–10	3–5
Total oxygen used, liters	10	15	20	28	47	70	143	565
Approximate ratio of aerobic to anaerobic energy release, %	15:85	25:75	40:60	55:45	80:20	85:15	95:5	99:1

[a] Data from Saltin.[2]

TABLE 2
Average lengths of total times of plays during the first half of the 1969 NFL Pro Bowl game (including special plays--kickoffs, punts, field-goal attempts, and extra-point attempts)

			Average time, sec
1st quarter:	Run	16 plays	3.7
	Pass	15 plays	4.7
2nd quarter:	Run	17 plays	3.9
	Pass	21 plays	6.4
1st half	Special	14 plays	6.4
Total		83 plays	5.1

TABLE 3
Average lengths of total times of plays during the first half of the 1969 AFL Pro Bowl game (including special plays--kickoffs, punts, field-goal attempts, and extra-point attempts)

			Average time, sec
1st quarter:	Run	21 plays	3.7
	Pass	11 plays	5.7
2nd quarter:	Run	18 plays	4.0
	Pass	27 plays	5.2
1st half	Special	17 plays	6.8
Total		94 plays	5.0

The testing of the strengths of the various body movements has usually been confined to the few movements that can be easily tested. It is convenient that the strengths of knee flexion and knee extension can be easily tested, inasmuch as the knee is so important from the standpoints of performing football skills and the high incidence of injury. Because we cannot measure the strengths of the myriad other body movements and because those strengths show very inconsistent relationships with each other in any individual, any attempt to measure over-all body strength or representative strengths may prove meaningless.

The measurement of isolated-movement speeds, such as arm flexion, suffers from the same problem--lack of relationships among the various body movements. In addition, research has shown that there is little relationship between the speed with which a movement can be performed and the strength with which it can be performed. Therefore, the testing of speeds of isolated movements shows little promise of being valuable.

The speed of performance of a series of movements (or skills) is of great practical importance in football performance. I refer to the periods involved in such situations as the following: (1) an offensive center initiates a snap and moves up into a blocking position, (2) a quarterback receives a snap and moves back into a passing position, and (3) a punter receives the ball and kicks it. These performance speeds are determined by a combination of strengths, skills, and movement speeds. Each performance can be easily timed with a stopwatch or, in more sophisticated fashion, with electronic timers.

The ability of a person to provide oxygen to his tissues, labeled "aerobic capacity" or "maximum oxygen uptake," can be measured precisely in laboratory procedures or estimated adequately in what one might consider field tests. As we have already seen, aerobic capacity and the involvement of oxygen in energy release have little or no bearing on performance in football. Therefore, the measurement of aerobic capacity need not be considered.

Anaerobic capacity, the ability to release energy without the delivery of oxygen, can be measured by measuring the so-called oxygen debt or by measuring a player's ability to sustain a maximal effort. Measurement of oxygen debt is an elaborate laboratory procedure and applies more to extended bursts of maximal effort than to the 4- to 6-sec bursts of effort involved in a football play. A player's ability to sustain maximal effort in a field test can be estimated with a stopwatch and by requiring the player to perform such an activity as maximal speed sprinting or pushing a blocking sled with maximal effort until he stops from exhaustion. But the relationship of the results of such determinations to performance capacity in football may prove inconsequential.

Therefore, it is difficult, if not impossible, to relate physiologic capacities to football-performance capacity and success. It seems apparent that, although physiology plays a role in the entire scheme of performance, football is predominantly a game of skill, knowledge, size, motivation, and movement strengths and speeds. The first four are not related to what we have discussed as being "physiologic," and the last two, strengths and speeds, cannot be adequately assessed or evaluated.

It appears that the best way to evaluate a person's performance capacity in football is to study his actual performance. This, of course, comes as no revelation; coaches have been doing it for years with movie films and projectors.

INJURIES AND INJURY PREVENTION

Having observed professional football for the last 25 years, I think it is obvious that two factors of extreme importance with regard to injuries are the size and speed of the players. I do not have any statistics on the speed of today's players compared with those of 10 and 20 years ago, but I think it safe to assume that they are at least as fast, if not significantly faster. I do have data on the size of players (weight being the important factor) of the last 25 years. I have selected the New York Giants as a representative team and, with the aid of the office of the Commissioner of Professional Football, have been able to plot the change in the average weights of particular segments of some of the New York Giants teams, as well as the team averages. It can be seen from the data plotted in Fig. 2 that the weights of the players coming into professional football are getting higher and higher. The increase in the average weight of the New York Giants from 1942 to 1968 is 22 lb, or 11%. The increase is probably not a sign of increasing obesity in the population of the United States. Rather, these players are larger in total body size, muscle mass, and strength. Momentum equals the product of mass and velocity; there can be no doubt that these men are hitting harder and injuring each other more effectively. The question can be logically raised as to whether this change in player size might require modifications in the rules of play.

In conclusion, it appears that the professional football player who is not suffering from injury is probably "in condition" for the activity involved and that the testing of physiologic capacities offers little help for the prediction of performance capacity or injury prevention.

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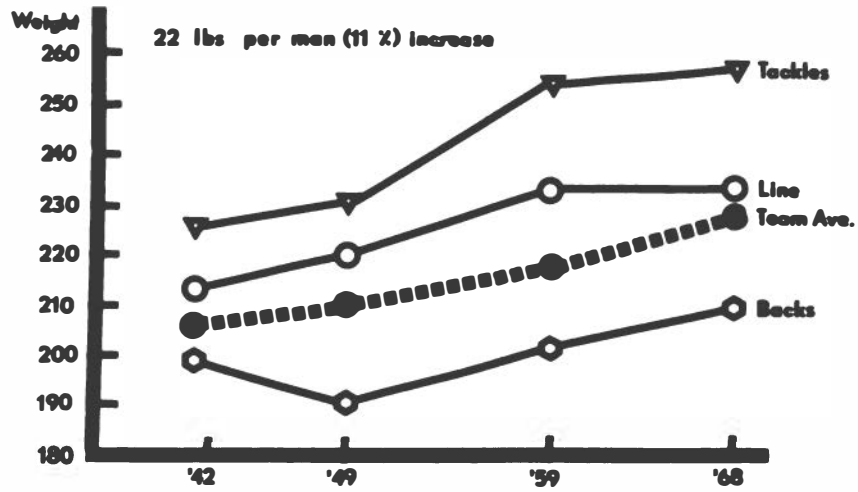

FIGURE 2
Average weights of selected New York Giants football teams from 1942 through 1968 and segments of the teams according to position (average weights of 1968 team do not include the preseason rookie roster).

TESTING AND DEVELOPING CARDIOVASCULAR FITNESS

KENNETH H. COOPER

"Physical fitness" may imply to a physician the absence of disease. To a weight lifter, it may be synonymous with large muscle mass. To a physical educator, it may be compatible with the ability to perform a required number of calisthenics or the ability to run 600 yards within a specified period. A commonly accepted definition implies adequate cardiopulmonary reserves, particularly as indicated by a high "aerobic capacity" or "endurance capacity." This latter type of physical fitness is important to the professional football player, because it may delay the onset of fatigue, perhaps reduce the likelihood of injury, and permit rapid recovery after a burst of activity--i.e., effective repayment of oxygen debt.

Because the development of a high "aerobic capacity" requires time, such capacity in football players should be developed before fall training. The players can then concentrate on developing required skills, learning plays, and even building specific muscle strength. Professional football players should keep themselves "physically fit" throughout the year.

TESTING CARDIOVASCULAR FITNESS

To assess cardiovascular fitness or aerobic capacity accurately, it is necessary to monitor a subject during continuous hard work.[7] Physiologic indices of cardiovascular status are maximal working capacity, peak heart rate, and maximal oxygen intake or consumption. Of these three indicators of cardiovascular fitness, maximal oxygen consumption is considered the most reliable.[10,11,16] However, maximal oxygen consumption involves an expensive and time-consuming laboratory procedure that cannot be adapted to large group studies. Therefore, in our laboratory we undertook the development of a field test that would correlate well with laboratory-determined maximal oxygen consumption. Following the pattern established by Balke,[2] we developed a 12-min walk-run test. We evaluated 115 men 17-52 years old on the treadmill and on a 12-min field test. The correlation between the 12-min distance (in miles) and maximal oxygen consumption (in milliliters per kilogram per minute) was 0.897. Thus, maximal oxygen consumption could be estimated accurately from the 12-min walk-run[3] (Table 1).

TABLE 1
Walk-run performance and maximal oxygen consumption
(115 men 17-52 years old)

12-min walk-run distance, miles	Maximal oxygen consumption, ml/kg-min
<1.0	<25.0
1.0-1.24	25.0-33.7
1.25-1.49	33.8-42.5
1.50-1.74	42.6-51.5
>1.75	>51.6

TABLE 2
Walk-run performance of several groups of males before training

	Average age, years			
	13.5[a,b]	19.1[c]	28.0[b]	34.0[b]
Average distance in 12 min, miles	1.52	1.41	1.35	1.27
% exceeding 1.50 miles in 12 min	61.8	37.9	17.1	10.2
No. tested	489	1442	1028	460

[a] Data from Doolittle and Bigbee.[6]
[b] Testing performed in tennis shoes.
[c] Testing performed in brogans or combat boots.

TABLE 3
Cigarette smoking and walk-run performance of 419 basic airmen on 12-min test

Smoking history, cigarettes per day	No. subjects	Average distance in 12 min, miles: At beginning of training	At end of 6 weeks of training	Increase
Never	128	1.47	1.61	0.14
Quit	64	1.44[a]	1.57[a]	0.13[a]
<10	86	1.42[b]	1.54[c]	0.12[a]
10-30	131	1.43[b]	1.52[c]	0.09[c]
>30	10	1.35[b]	1.44[c]	0.09[a]

[a] Not significant when compared with "never" group.
[b] $0.05>p>0.02$.
[c] $p<0.001$.

Most athletic teams use performance on a 1-mile run as the indicator of aerobic or endurance capacity. However, the mile run is too anaerobic to be a good indicator of aerobic capacity. The study reported by Allen and Bryan[1] showed a correlation coefficient of only 0.359 between maximal oxygen consumption and the 1-mile run. Consequently, we encourage the use of the 12-min test as an indicator of endurance capacity.

Maximal oxygen consumption of 42 ml/kg-min is generally accepted as indicative of satisfactory aerobic capacity.[2,11,16] It can be seen from Table 1 that a minimum of about 1.5 miles in 12 min is required. That minimum is applicable to all members of a football team. In addition, for players who are required to run down field repeatedly, such as offensive backs and ends, I would suggest a required minimum of 1.75 miles in 12 min.

After a distance of 1.5 miles in 12 min had been established as the indicator of a satisfactory level of fitness, we tested several large groups consisting primarily of deconditioned men. Table 2 shows a general lack of cardiovascular fitness, particularly with advancing age. The lower level of performance by 19.1-year-old basic airmen compared with that of 13.5-year-old schoolboys[6] could be explained in part by the difference in footgear--tennis shoes vs. brogans or combat boots. However, no similar explanation could apply to the lower performance level observed in 28.0-year-old officers compared with 19.1-year-old airmen.

The sensitivity of the 12-min field test to cardiovascular fitness was documented in this study, and its sensitivity to cigarette smoking, in another study.[5] Table 3 shows that the average distance covered in 12 min by the nonsmokers was significantly greater than that covered by the smokers at the beginning and at the end of 6 weeks of physical training.

DEVELOPING CARDIOVASCULAR FITNESS

Previous investigations have shown that calisthenics and strict muscle-building exercises have little if any effect on improving the cardiovascular system.[14] To the contrary, endurance-conditioning exercises--such as long-distance running, cycling, walking, and swimming--readily produce significant changes in both the cardiovascular and the pulmonary systems.

Training of the type necessary to develop competitive endurance athletes was not the objective of this physical-conditioning program. Its primary purpose was to develop the cardiovascular system so that unexpected emergencies could be tolerated[15] and, perhaps, the onset of ischemic cardiovascular disease delayed.[9] An exercise program that included some type of regular, vigorous effort was required[9] if this objective was to be achieved. Unless the training stressed the cardiopulmonary system and was of sufficient duration, only minimal benefits would be realized.

As the various prerequisites for optimal cardiovascular conditioning became apparent, it was realized that a reliable method for comparing types of exercise was needed before training programs could be developed. In searching for a method of equating different exercises, we found that the oxygen or metabolic cost was the ideal common denominator. This value could be obtained readily by collecting and analyzing the air expired during a standard performance, such as walking, running, or cycling a known distance in a specified time. Because the oxygen cost of a physical activity decreases with conditioning, it was necessary to study a cross section of highly conditioned athletes and deconditioned subjects. We also reviewed other investigators' work,[8,12,13] with remarkably comparable results (Table 4). A system estimating the metabolic cost of the exercise was developed using excess oxygen consumption--i.e., the total oxygen consumption minus the resting oxygen requirement. The energy cost was converted into milliliters per kilogram per minute, and a simple point system established. The points, in multiples of seven, represent the approximate excess cost or consumption in milliliters per kilogram per minute. An example of the oxygen requirement for a moderately built 78.5-kg man to walk and run 1 mile is shown in Table 5. It should be noted that points are awarded in a stepwise, rather than linear, manner, with the slowest performance in each interval. Consequently, in some instances, fewer points are awarded than the energy expenditure of exercise actually indicates. Ultimately, more effort will be required to reach any particular point goal.

Various types of physical activities were quantitated, and the next objective was to establish a fitness goal--the number of points per day or per week required to enable most persons to achieve the minimal oxygen consumption of 42 ml/kg-min (the equivalent of running 1.5 miles in 12 min). A number of studies demonstrated that exercise averaging 30 points per week would produce the minimal level of cardiovascular fitness in most persons, provided that this fitness was achieved progressively over 10-16 weeks. To achieve higher levels of fitness, more points per week were necessary: 50 points per week were compatible with a performance of 1.60-1.65 miles, whereas 75 points per week were needed to achieve the excellent level of fitness, 1.75 miles in 12 min. Roughly 80%-85% of the subjects who participated in this program and worked up to the previously mentioned point levels were able to achieve their goals. Those who were unable to achieve the desired level of fitness after completing the training programs usually fell into two categories: (1) those who were 10%-15% overweight and did not lose weight in response to their exercise program, and (2) those who smoked more than 20 cigarettes per day.

TABLE 4
Metabolic cost of walking and running on treadmill, Kcal/min

Mile time, min:sec	Cooper,[4] net cost (78.51 kg)	Cooper,[4] total cost (78.51 kg)	Pollack *et al.*,[12,13] total cost (66.0-86.0 kg)	Margaria,[8] total cost (72.5 kg)
5:10	--	--	19.0[a]	--
6:15	16.6	18.0	--	--
6:45	--	--	15.0[a]	--
7:00	15.0	16.0	--	--
8:00	14.2	15.6	--	--
8:34	--	--	13.5[a]	--
9:00	12.8	14.0	--	--
10:00	12.2	13.5	--	--
10:20	--	--	11.0[a]	--
11:00	12.1	13.5	--	--
11:36	--	--	11.25	--
12:00	10.1	11.5	--	--
13:00	7.5	9.0	8.00[a]	--
13:24	--	--	8.34	--
14:00	6.6	8.0	--	--
14:30	5.7	7.2	--	--
15:00	5.0	6.5	--	5.8
16:00	4.6	6.0	--	--
17:06	--	--	5.29	5.0
18:42	--	--	4.38	--
20:00	3.4	4.9	--	4.4

[a] 66.0-kg male.

TABLE 5
Total excess oxygen[a] requirements for walking and running by 78.5-kg man

Mile time, min:sec	Average oxygen cost, ml/kg-min	Assigned point value
≥20:00	<8.7	0
14:30-19:59	8.7-14.4	1
12:00-14:29	14.4-25.7	2
10:00-11:59	25.7-31.2	3
8:00- 9:59	31.2-35.8	4
6:30- 7:59	35.8-42.1	5
<6:30	>42.1	6

[a] Total excess oxygen = (total oxygen consumption) - (resting oxygen consumption per minute) (duration of run, in minutes).

SUMMARY AND CONCLUSIONS

For the purpose of this study, the term "physical fitness" implied adequate cardiovascular-pulmonary reserves, as indicated by maximal oxygen consumption. A field test of fitness was developed because of the impracticality of mass laboratory testing. The field test consisted of a maximal performance in 12 min; the correlation with the laboratory-determined maximal oxygen consumption was 0.897. After the development of a field test for evaluating fitness, an exercise program was designed that would develop adequate cardiovascular-pulmonary reserves. To accomplish this objective, a point system was developed on the basis of the excess oxygen requirements for given physical activities, and a specific weekly point goal was established. This goal could be reached through various exercise programs varying in length from 10 to 16 weeks, or perhaps even longer. Roughly 80%-85% of the men tested in this conditioning program were able to achieve the minimal level of fitness.

The application of this study to the training regimens of professional football players is obvious. If a high aerobic capacity is present at the beginning of fall training, the athletes can expect a delayed onset of fatigue, quickened reflexes, and perhaps a reduction in the number of athletic injuries.

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THE POTENTIAL FOR CARDIOVASCULAR ACCIDENTS IN ATHLETES WITH CARDIAC PROBLEMS

KENNETH D. ROSE

Whether diseases of the cardiovascular system are sufficiently common in athletes to warrant attention in sports medicine has been the subject of considerable discussion in the past. As in many areas of human discourse, the clear understanding of the problem that could lead to its resolution is clouded by divergent opinions, often coming from recognized authorities each of whom is undoubtedly biased—in this case by his own attitude toward the relative value of athletic competition in modern American culture. One finds in the literature, for instance, such anomalous statements as that attributed to Dr. Marshall Franklin:[5] "If . . . this [murmur] is a functional murmur or a mild cardiac defect, I will probably let him go ahead, telling him what symptoms he might expect--shortness of breath, palpitation, etc. If he has these symptoms, he must stop whatever he is doing--even if he is about to make the winning touchdown"; or that of Dr. Albert Hyman:[9] "Patients with grossly abnormal findings from most standard diagnostic tests including electrocardiograms, phonocardiograms or angiograms may be able to participate in continuous high levels of activity--*even contact sports*." I quarrel, not with the above statements coming from cardiologists long known for their interest and competence in sports cardiology, but with the implication of generality, which leaves the casual reader with a skewed view of the importance of abnormal cardiovascular function in competitive sports.

Expert opinion such as that expressed by Dr. Hyman is based on a lifetime of experience in sports cardiology encompassing numerous athletes with cardiac anomalies who apparently were untroubled by their disease. I have encountered some myself. One, who at the age of 8 was thought to have rheumatic heart disease, was later found to have a small, physiologically unimportant interventricular septal defect. A distance runner in college, he still runs 15 miles daily at the age of 29. In spite of this knowledge of his history, I still could not conscientiously recommend that a person with a known interventricular septal defect become involved in cross-country running. The reason is that an isolated example, such as his, is never a sound basis for generalization.

My point is that the controversy surrounding cardiovascular disease in sports stems from inadequately controlled data and biased opinion. What is needed is an objective approach. Instead of the premise that "athletic competition is good for cardiac patients and here are some cases to prove it," or the premise that "athletic competition is bad for cardiac patients and here are some cases to prove it," the approach should be: "Given a specific cardiovascular disease, what is the evidence that athletic competition affects the patient one way or the other?" In 1967, at the Ninth Annual Conference on the Medical Aspects of Sports, I delineated a few areas in which some practical information was available.[15] I would like to expand those observations here.

Table 1 is a representative list of cardiac deaths that occurred during or immediately after athletic competition. (It should be mentioned that heat exhaustion and heat stroke have been carefully excluded as a cause in these cases.)

Some general observations may be made. Of the 44 persons who died, 38 were between the ages of 14 and 19; most were 15-17 years old. That suggests that most sports-related cardiovascular deaths occur at the high-school level. Most of the deaths were related to football; thus, most occurred during football season, although one (case 31) occurred in spring football practice. Inasmuch as 36 of the 41 football-season deaths occurred between 15 August and 30 September, some attention should be given to activity at time of death. From this standpoint, 14 occurred during practice sessions, three during wind sprints, five during laps, four during calisthenics, two during unspecified warmup exercises, and only eight during a football game. Those whose hearts were physically unable to withstand the rigors of football succumbed during the preseason conditioning program. With those thoughts in mind, attention should be directed toward the known antecedents of these 44 cardiac deaths.

CONGENITAL HEART DISEASE

Six of the 44 victims had congenital heart disease. Only one of the reports specifically identified the lesion as an atrial septal defect, although it may be presumed that the other five had chamber or vascular anomalies identifiable by auscultation, chest x-ray, or both. It can be seen that patients with congenital heart disease *do* die during athletic activity; blanket approval of such activity without complete investigation should be condemned. Not all congenital anomalies are identifiable by routine methods. Jokl, McClellan, and Rose[10,12] have reported on seven persons 11-22 years old who collapsed and died during strenuous activity (five in organized sports) and who were found at autopsy to have anomalous coronary arteries.

Three other examples will illustrate decisions relative to congenital lesions in sports. One patient presented to his examining physician, at the age of 18, with grade IV systolic and diastolic murmurs over

TABLE 1
Cardiac Deaths in Athletes[a]

Case	Age, years	Activity	Data	Remarks
1	14	Football warmup	5 Sept. 1967	"Violent exertion"
2	16	Football calisthenics	8 Sept. 1967	Collapsed and died
3	17	Football calisthenics	Sept. 1967	Ruptured aneurysm
4	17	Football wind sprint	2 Sept. 1967	Collapsed and died
5	15	Football practice	19 Aug. 1967	Fatal heart seizure
6	14	Football warmup run	Sept. 1967	Collapsed and died
7	9	Baseball	16 Aug. 1967	Struck in chest by pitched ball
8	15	Baseball	13 July 1967	Struck in chest by pitched ball
9	12	Football and run	4 Oct. 1967	Cardiac arrhythmia and arrest
10	14	Football game	5 Nov. 1966	Heart disease (autopsy)
11	14	Football game	27 Sept. 1966	Organic heart disease (autopsy)
12	15	Football practice	10 Oct. 1967	Organic heart disease
13	18	Football game	23 Sept. 1967	Ventricular arrhythmia, rheumatic heart disease (autopsy)
14	17	Football practice	12 Sept. 1967	Heart attack (autopsy)
15	19	Football practice	31 Aug. 1965	Right-sided heart failure (autopsy)
16	18	Football practice	2 Mar. 1966	Cardiac arrest, electric irregularity
17	16	Football calisthenics	23 Sept. 1966	Focal and diffuse myocardial fibrosis, anomalous right coronary artery (autopsy)
18	16	Football game	9 Dec. 1968	"Collapsed and died"
19	11	Recreational sports	Dec. 1968	Cardiac arrest
20	17	Rope climbing	29 Nov. 1968	Congenital heart disease
21	16	Football practice	Fall 1965	Electric irregularity (autopsy)
22	14	Wind sprints, football	27 Aug. 1965	Electric irregularity (autopsy)
23	17	Wind sprints, football	18 Aug. 1965	Electric irregularity
24	16	After doing laps, football	23 Aug. 1965	Congenital heart disease with heart failure (autopsy)
25	19	Football practice	19 Aug. 1965	Subendocardial myocardial infarction (autopsy)
26	19?	Football game	11 Sept. 1964	Heart failure
27	19	Football practice	1 Sept. 1964	Congenital heart disease
28	15	Football game	Fall 1964	Acute myocarditis (autopsy)
29	15	Football calisthenics	5 Oct. 1964	Collapsed and died
30	15	Laps	10 Nov. 1964	Chest pain, collapsed and died, no overt heart disease in autopsy
31	17	Football practice	20 May 1963	Congenital heart disease
32	17	Football game	14 Aug. 1963	Heart attack (no strenuous exercise)
33	18	Football practice	5 Sept. 1968	Chest pain, excessive exercise
34	16	Football practice	18 Aug. 1961	Ventricular fibrillation, congenital heart disease (autopsy)
35	18	Football warmup	27 Oct. 1961	Possible heart attack
36	15	Football, laps	15 Sept. 1961	Heart attack (autopsy)
37	16	Football practice	28 Aug. 1961	Congenital atrial septal defect (autopsy)
38	16	Football practice	21 Aug. 1961	Acute ventricular fibrillation
39	19	Football practice	18 Oct. 1961	Heart failure, history of heart disease
40	22	Football game	8 Nov. 1961	Heart attack (coronary)
41	36	Horserace	12 July 1967	Struck in chest by horse's head
42	14	Basketball	6 Dec. 1967	Collapsed and died (natural causes, coroner's report)
43	17	Basketball	Dec. 1967	Collapsed and died
44	17	Basketball	4 Dec. 1967	Collapsed and died

[a] Data from files of C. S. Blythe and D. C. Arnold,[2] A. J. Ryan, and K. D. Rose.

most of the precordium and transmitted to neck, back, and axilla. Under ordinary circumstances, this patient would have been totally restricted in his athletic exercise, because his original diagnosis was rheumatic heart disease. However, he was referred for cardiac-catheterization studies, including valvulography, which revealed a congenital bicuspid aortic valve with minimal functional impairment. This 18-year-old boy had played 4 years of metropolitan-league high-school football and other sports without symptoms. The murmur was discovered at examination for employment after graduation from high school. In college, he was not restricted in his activities and played in all intramural sports without difficulty, graduating at the age of 23. At his last examination, as a senior, there was no x-ray or ECG evidence of left ventricular hypertrophy.

The second case involved a freshman student whose screening chest x-ray on entrance revealed enlargement of the right side of the heart. He was referred to his physician, and complete cardiovascular analysis, including angiocardiography, revealed congenital downward displacement of the tricuspid valves into the right ventricle (Ebstein's disease). Although some persons with this anomaly live to middle age and even advanced age without symptoms, the average age at death is 22, and sudden death from arrhythmias is common. The young man in question had played high-school football, although the anomaly should have been easily detected on physical examination. He was strongly advised, by his physician, against participation in sports demanding severe physical stress and was given a restricted health classification at the university. In spite of that, he continued to play intramural football and basketball. One can conclude only that he has been fortunate, although foolhardy. This emphasizes the difficulty in controlling known cardiac-risk patients when they are athletically inclined. The answer, of course, is to attempt to steer them into less-demanding sports, thus protecting them from the rigors of physically and psychologically strenuous contact sports while affording them an outlet for their drives.

The need to understand the latent danger in cardiac anomalies is emphasized by the third case that came to my attention indirectly but nevertheless authoritatively. A high-school basketball player had known aortic stenosis, but his angiocardiographic analysis, ECG, chest x-rays, and stress studies all revealed normal cardiopulmonary function. Clearance was given for him to compete. He died in acute cardiac arrhythmia, presumably ventricular fibrillation, while playing in a high-school basketball game. Ordinarily, cases of this nature never come to the attention of those who need to know, and I mention this one only to re-emphasize that known cardiac disease cannot be treated lightly when the issue concerns whether its bearer should subject himself to the stresses of 20th-century competitive sports. My recommendation would have to be that all persons with known cardiovascular anomalies, regardless of their current functional capacity, be encouraged to dispel their physical and psychologic drives in pursuits less physically demanding, but as satisfying, as competitive athletics.

MYOCARDITIS

Two of the 44 victims died of myocarditis that was proved at autopsy, although some of those listed as dying from collapse, violent exertion, arrhythmias, and organic heart disease may have had myocarditis. Burch *et al.*[3] have concluded that the common occurrence of clinically occult coxsackie B virus infection may be related to the etiology of sudden, unexpected cardiac deaths. The incidence of coxsackie B, as well as other viruses, in the college-age group is well known. In the spring of 1967, we observed two cases of transient and one of sustained arrhythmia in athletes in whom acute virus disease was diagnosed.[15] In the fall of 1968, a fourth case was identified. The ECG findings are shown in Fig. 1. Note that the arrhythmia reverted to normal with time. Because the physiologic stress of athletic competition superimposed on an inflamed myocardium is potentially dangerous, it is our recommendation that in all cases of acute virus illness in athletes, other than common colds, myocarditis be ruled out before return to active competition. Such a study can be done with small expense in time and money and is worth the effort. If there is evidence of myocarditis, release for full participation should be withheld until a return to normal.

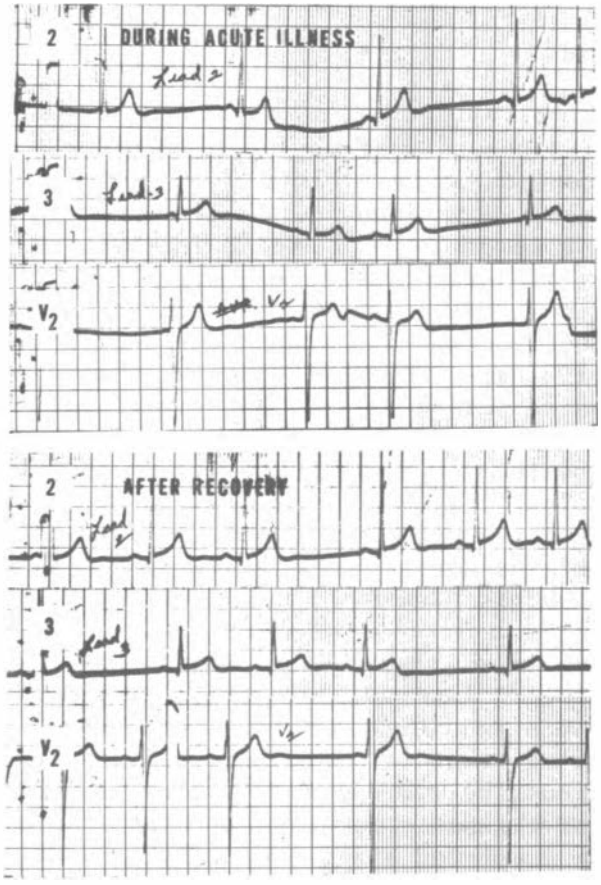

FIGURE 1
Arrhythmia during viremia.

RHEUMATIC FEVER

Only one case (case 13) was officially identified as rheumatic heart disease, although others listed as organic heart disease or death from arrhythmia may also be included as possible cases. Although the consensus prohibits participation during the acute phase of rheumatic fever, disposition of the quiescent rheumatic patient is far from settled. Even the late Joseph Wolffe, eminent sports cardiologist, appeared to contradict himself in this regard. In the early 1960's, he stated:[21,24]

> After apparent recovery from the acute phase of fatigue, auricular or ventricular ectopic beats may appear in showers, and may give rise to auricular or ventricular fibrillation. This serious disturbance of the conductive system may account for the sudden death during or immediately following a strenuous game, without any evidence of congestive cardiac failure. This may be classified as electrical death.

A few years later, he said:[22] "Coincidental abnormal ECG findings of any type should, in the absence of organic heart disease, be considered functional in nature."

The pertinent question, of course, is whether organic heart disease is present. In some instances it is obvious, but evidence of cardiac enlargement or hypertension in an athlete is not necessarily a sign of organic heart disease. Nor is an increased ASOT,* CRP,** or sedimentation rate, or even some of the ECG signs, such as the juvenile pattern T inversions in V_1-V_2. However, absence of all the traditional findings of organic heart disease does not preclude its presence. Thomas *et al.*,[20] for instance, reported the sudden death of an 18-year-old football player and a 15-year-old wrestler, both of cardiac arrhythmias. The standard autopsies were negative, but serial sections across the conduction mechanism, including the bundle of His, revealed multiple areas of focal necrosis. Although there was no definitive etiologic diagnosis, it was clear that both boys died in acute cardiac arrest as a result of a pre-existing but unrecognized disorder affecting the conduction mechanism of the heart. Thus, even in the absence of clear evidence of organic heart disease, such disease may be present.

One of the ECG signs of rheumatic heart disease is first-degree block, a prolongation of the PR interval. In 1967, we reported the few cases observed in our 1200 screening ECG's.[15] In one case, the patient had no evidence of heart disease or history of illness, and his high-school sports record was good; he was allowed to play, but he was found later to have a history of rheumatic fever as a child. He had concealed this information, but he completed his 4-year college scholarship without incident.

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* Antistreptolysin "O" titer.

** C-reactive protein.

First-degree heart block may be associated with later problems. In 1968, a varsity basketball player, whose screening ECG had revealed a PR interval of 0.21 sec, was seen to clutch his chest and stop during a practice game. Questioned as to his trouble, he denied having a problem. A few minutes later, he again clutched his chest, gasped for breath, and fell to his knees, this time admitting severe chest pain. Shortly thereafter, an ECG (Fig. 2) recorded at the health center showed obvious gross irregularity--a typical atrial fibrillation. Later tracings revealed a return to complete normalcy, and he continues to participate without difficulty. There is some support for a liberal approach to this type of problem.[1,4,7,8,18] But again, more longitudinal studies are needed to furnish a basis for definitive recommendations. Until they become available, decisions on such cases must be made on the basis of continued observation.

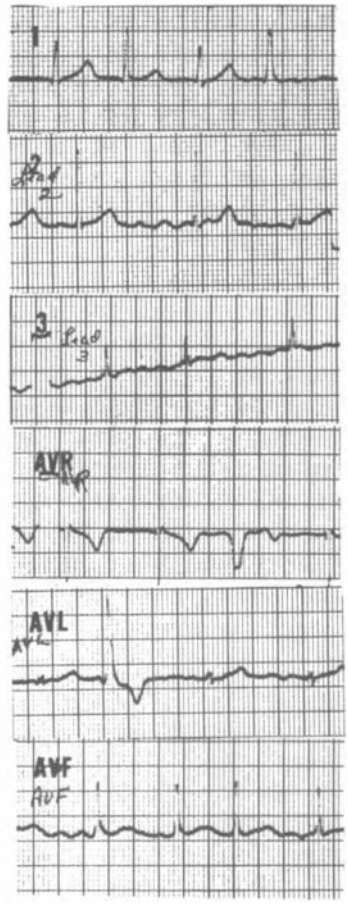

FIGURE 2
Atrial fibrillation in athlete with previous first-degree heart block.

The valvular diseases of rheumatic fever once were considered positive contraindications to active sports participation. But newer methods for studying cardiovascular dynamics are leading to a more liberal approach, particularly in the mild, treated case kept under close observation.[11] However, the desirable follow-up is difficult, if not impossible, except under ideal conditions of patient-physician relationship. For practical purposes, therefore, it is advisable to continue recommending restriction from strenuous sports.

CORONARY HEART DISEASE

Although a medical problem primarily of the over-40 age group, coronary artery disease is not unknown in younger patients. In the series reported here, two athletes died of this ailment; one was 19 and the other, 22. In 1967, a promising 25-year-old professional basketball player reportedly suffered a coronary occlusion. Fortunately for the professional athlete, there is evidence of an inverse relationship between the physical activity of work and coronary heart disease. In a study of early precursors of fatal coronary heart disease in former college students, Paffenbarger and co-workers[13] found college athletic participation to lower the incidence of fatal coronary heart disease significantly. It is reasonable to assume that continued participation would continue that favorable relationship.

Smith and co-workers[19] reported on several patients who developed acquired bundle-branch block. Their ages ranged from 29 to 39 years. One had angiographic evidence of coronary artery disease. One with right bundle-branch block (RBBB) had typical angina pectoris without x-ray evidence of coronary artery disease. Another died of acute myocardial infarction 8 months after routine ECG revealed an acquired RBBB. Autopsy revealed extensive coronary artery disease. The possibility of such an unfortunate accident's happening in varsity or professional sports could be decreased by routine annual ECG, with extensive cardiovascular study being reserved for persons whose tracings show significant changes, such as acquired bundle-branch block.

ARRHYTHMIAS

Disorders of the conduction mechanism of the heart present a special problem to the physician interested in sports medicine. Of the 44 persons listed in this series, 11 were recorded as having died from an arrhythmia, but ventricular fibrillation was most likely the terminal event for many of those whose history indicates only that they "collapsed and died." In any respect, at least 35% of the cardiac deaths in this series resulted from disorders of the conduction mechanism. What should the physician do when confronted by a varsity player in midseason whose chief complaint is inordinate fatigue and whose only positive finding is a second-degree heart block with shifting nodal rhythm and incomplete right bundle-branch block? In the case illustrated

in Fig. 3, the athlete's preseason screening ECG was normal; thus, these findings represented a change in cardiac status. There was no evidence of obvious heart disease.

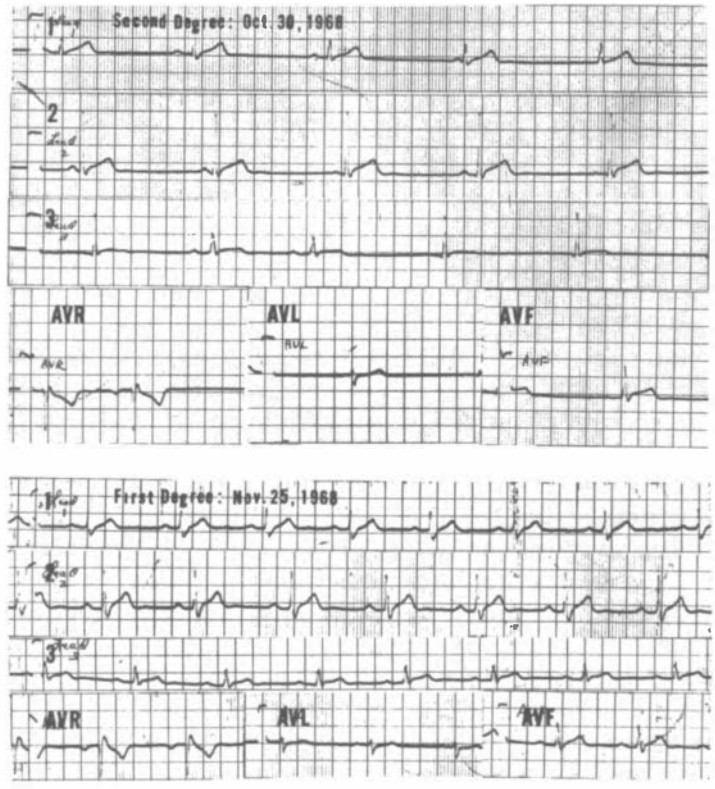

FIGURE 3
Atrioventricular block in athlete; etiology unknown. Screening ECG in August 1968 was normal.

Wolffe, reporting on a survey of 3212 ECG's of athletes actively engaged in a variety of sports, stated that he observed only 28 instances of heart block, including all types.[22] In his opinion, heart block of any kind ought not prevent participation in athletics. He was supported in his belief by Hovak, who further reported on 25 cases in a total of 2447 trained athletes from Czechoslovakia and who felt that disorders of this nature were the result of vagotonia.* There is, however, considerable evidence, not only clinical but also experimental, to indicate that in the susceptible heart sudden vagotonic influences seriously modify cardiac conduction and physiology, even to the extent of producing cardiac arrest and death.[23] In addition, Schneider[17] has found that coronary patients with the greatest tendency to bradycardia in response to a sudden frightening stimulus have the poorest prognosis, in terms of death from subsequent coronary occlusion. Thus, the

* The incidence of arrhythmias in these reported series was only 0.9%.

appearance of good health in a person with supposedly "vagotonically induced" heart block is not *a priori* evidence of a completely normal conduction mechanism. Rather, susceptibility to vagotonically induced heart block is *a priori* evidence of an abnormal conduction mechanism.

TRAUMATIC HEART DISEASE

Of the 44 deaths listed in Table 1, three resulted from direct blows to the chest: cases 7, 8, and 41. Cases 7 and 8 are unusual, in that death followed immediately after the blow, in both instances with a thrown baseball. Case 7 was a 9-year-old boy who was struck by a pitched ball in a Little League practice game. Case 8 was a 15-year-old boy who was struck by a wild toss, catcher to pitcher covering home plate, as he tried to score a critical run from third base. Both boys died in acute ventricular fibrillation or arrest precipitated by a direct blow over the heart. Autopsy findings are not available on case 7 to give us the needed definitive diagnosis. The diagnosis of acute arrhythmia is more easily substantiated in the second case; the medical examiner's report reveals no evidence of injury.

Such injuries are less likely in varsity or professional players because of the heavier musculature in the adult contestant. However, cardiac trauma does occur, with an occasional fatality. We reported one non-fatal instance in a blocking back who was "speared" by an opposing lineman.[16] Selective coronary angiography and ventriculography revealed no residual evidence of injury,[15] and the player has just finished his elegibility, serving as Nebraska's first-team defensive corner linebacker. His ECG (Fig. 4) is entirely normal, and he has had no evidence of cardiac disorder in spite of a strenuous season. I am strongly opposed to the vicious practice of "spearing," as well as to the use of an elbow or knee when the sole purpose is to disable an opponent. Those who watch football critically from a medical standpoint are appalled at the obvious efforts to disable. I would add my voice to the others in urging that deliberate "spearing" or other attempts to injure be cause for ejection from a game.

One cardiac death occurred in a freak accident in horseracing (case 41). The deceased, a 36-year-old jockey, was struck in the chest by his horse's head. He complained of chest pains after the race and was examined at a medical center, where x-rays revealed no injury. He died suddenly 3 hr later, while riding home in a car. This is a typical case history for cardiac contusion. Ferré and Steward[6] report the case of a golfer who was struck in the chest by a golfball. He was in shock for 15 min and recovered, only to suffer a fatal myocardial infarction 2 days later. It is common for significant cardiac trauma to become apparent only after several hours or even 2 or 3 days after injury. One should be suspicious of cardiac injury in all acute anterior chest injuries and should observe the patient closely. Our patient developed symptoms only 12 hr after injury.[16] Fortunately, he had been hospitalized immediately after the game by an orthopedic team

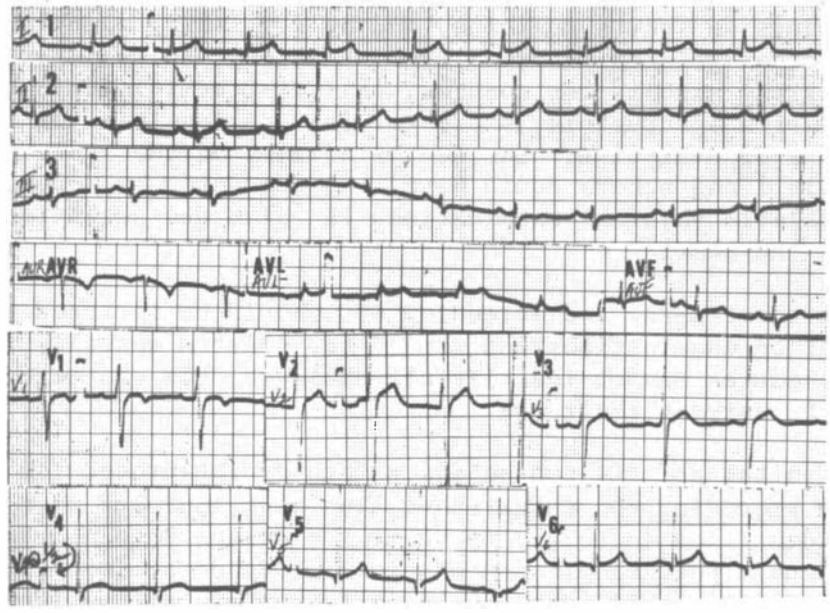

FIGURE 4
Cardiac contusion 2 years after injury.

physician specifically for observation for possible cardiac contusion. In this disease, a previously recorded ECG is of inestimable value and would aid in making a decision concerning future participation. Pollard[14] reported a case of cardiac contusion in 1963, wherein the diagnosis was based on some x-ray evidence of cardiac enlargement and incomplete RBBB and peaked T waves in the ECG. The results of all other laboratory tests were normal. This man was a senior in college and a vigorous competitor, but he was removed from further football participation. Because RBBB, and particularly incomplete RBBB, is such a common ECG finding in healthy young adults and is usually the so-called congenital form,[19] only acquired RBBB following an incident of this nature could be considered significant. In this respect, a previously recorded ECG would have given the answer.

SUMMARY

I have tried to make some practical suggestions relating to the abnormal heart in the athlete or would-be athlete, illustrating wherever possible with specific examples from my experience and from the experience of others. These suggestions can be summarized as follows:

(1) There is no substitute for prevention, and the best preventive measure is a good history and physical examination by a knowledgable physician. The American Medical Association's Committee on the Medical Aspects of Sports has devoted itself to furthering this goal by recommending procedures and suggesting criteria, by sponsoring local and national meetings on sports medicine, and by furnishing pamphlets, folders, and other printed material free or at cost to colleges, high schools, and grade schools for coaches, trainers, administrators, physical-education instructors, and physicians responsible for the medical aspects of athletics. The deaths of many of the athletes listed in Table 1 and others could have been prevented by annual comprehensive athletic physical examinations in which due attention had been given to the heart.

(2) A routine chest x-ray and screening ECG should be included in all pre-participation examinations and should be repeated annually. Screening procedures cannot identify all diseases, but the return is great enough to warrant their use.

(3) Most congenital defects can be identified by the procedures mentioned in the preceding two paragraphs. The best approach when such defects are identified is to try to steer the person into sports that lack the strenuous psychologic and physical stresses of competitive team sports. Failing this, one should insist on a positive identification of the lesion and should be guided by the known prognosis in making recommendations. Frequent functional follow-up is mandatory, and the coach and administrators should be apprised of the potential risk.

(4) At any age, one should suspect myocarditis in any athlete who does not recover promptly from a virus illness. Routine testing for this ailment as a prerequisite for return to full competition is the ideal.

(5) Rheumatic fever, past or present, must always be suspect. Temper the irrational drives of the sports-oriented postrheumatic person with calm judgment and guide him into noncontact individual sports that do not demand physiologic output to the point of exhaustion.

(6) Coronary artery disease can occur at any age, but it is progressively more common with increasing age. One episode of coronary occlusion should permanently restrict the player (if he is fortunate enough to survive it), because it is rare for only one artery to be involved. For prevention, lifetime physical fitness is mandatory. Routine annual ECG's, particularly for the professional, can identify subclinical changes, such as acquired bundle-branch block, and should identify those requiring a more thorough cardiovascular survey.

(7) Cardiac contusion probably occurs more often than we recognize. Awareness of its likelihood in the presence of anterior chest injury is the best prevention against serious sequelae. When it is suspected, the subject should be hospitalized for observation. Spearing should be outlawed, and deliberate attempts at injury should be cause for ejection from the game.

A concerted attempt is being made at the primary, secondary, and college levels to accumulate valid death statistics backed by autopsy reports, and professional sports should enter into this endeavor if the occasion arises.[2] The purpose is to establish a valid scientific basis for identification of problems from which future recommendations concerning participation can be made.

Forty-four deaths are probably not many when compared with the number of man-hours of contact sports played per year, and few indeed when compared with Vietnam and automobile accidents. But most of them are preventable by examination and common sense, and they ought not to be permitted to occur.

I wish to thank Drs. Jerome E. Bleicher, Richard Hammer, and William J. Kuzman for specific details of some cases presented herein.

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LACTIC ACID, OXYGEN DEBT, AND TRAINING

CHARLES M. TIPTON and KENNETH M. BALDWIN

It is widely held that the presence of lactic acid in the blood is directly responsible for the existence of an oxygen debt. We plan to examine some aspects of this relationship and to discuss the influence of training on them.

The basic relationship between glycogen, glucose, pyruvic acid, and lactic acid is shown in Fig. 1. Details concerning the enzymes responsible for these reactions, equilibrium constants, and cofactors can be found in most biochemistry texts. The over-all reactions of the glycolytic process lead to the production of pyruvic acid and a small amount of adenosinetriphosphate (ATP). For lactic acid to be produced from pyruvic acid, the oxidation-reduction state of the cell has to favor the following reaction, which is catalyzed by the enzyme lactic dehydrogenase (LDH):

$$\text{pyruvic acid} + \text{NADH} + \text{H}^+ \underset{}{\overset{\text{LDH}}{\rightleftharpoons}} \text{lactic acid} + \text{NAD}^+$$

The production of lactic acid is related to the total metabolic requirements of the cells; therefore, it is important to consider the cellular factors that determine oxygen consumption.

Data from isolated mitochondrial preparations and from tissue slices indicate that the cellular levels of adenosinemonophosphate (AMP), adenosinediphosphate (ADP), inorganic phosphate, and reduced nucleotides determine the amount of oxygen consumed by the cells.[12,13] These considerations compel one to review the metabolic reactions within the cell and the involvements between lactic acid and these reactions before ascribing a cause-and-effect relationship between measurements of lactic acid and oxygen consumption.

It has been known since 1806 that lactic acid can be produced in muscles.[8] The reasons for its production are complex but can be said to be related to aerobic or anaerobic conditions. Under aerobic conditions, the oxidation-reduction state of the cell favors the reactions of pyruvic acid and NADH* within the Krebs cycle of the mitochondria.

* NADH and NAD^+ are the abbreviations of the reduced and oxidized forms of the coenzyme nicotinamide adenine dinucleotide.

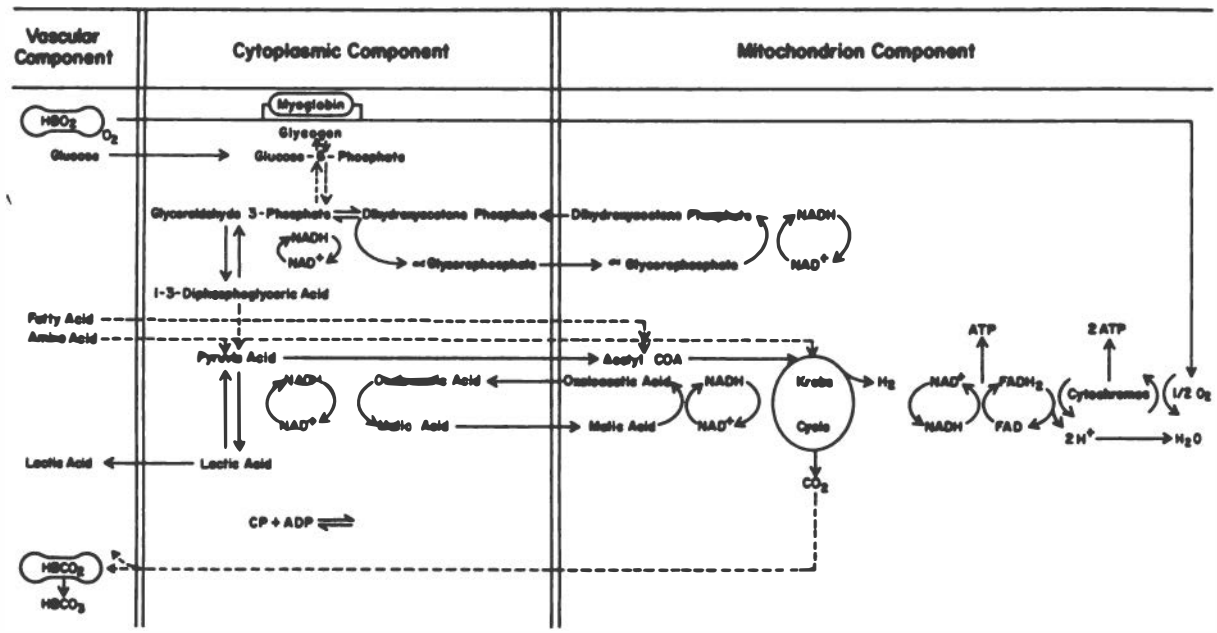

FIGURE 1
Elementary metabolic relationships within muscle cells.

However, at rest and even under oxygenated conditions,[33,53] a small amount of lactic acid will be produced. The nature of the blood flow, the redistribution of flow, transport characteristics, and mass-action considerations appear to be primarily responsible.[3,9,10] When anaerobic conditions prevail, the energy needed for oxidation of the nucleotides is inadequate; thus, the NAD^+:NADH ratio changes, and pyruvic acid is reduced to lactic acid in the presence of LDH. Because lactic acid can easily cross cellular membranes, its concentration within the blood-stream will increase soon after the intensity of exercise is increased. As mentioned by Diamant *et al.*,[14] there is at rest, as well as during exercise and recovery, a marked gradient between the lactic acid levels in muscle and blood. That finding emphasizes the difficulty in using blood levels to predict concentrations within tissues. Another point worth emphasizing is that lactic acid can be produced and used in adjacent regions of working tissue.[25] Moreover, lactic acid can be actively transported against a concentration gradient.[19,51]

In the blood, lactic acid can be involved in chemical reactions that have different ATP requirements (Table 1). In the liver and kidney, lactic acid can be converted into glucose. These reactions require

ATP, thereby increasing ADP levels, which in turn elevates the oxygen requirements of the cells. In cardiac tissue[11,34] and muscle,[15,26] lactic acid can be reconverted into pyruvic acid and then oxidized to carbon dioxide and water. When that occurs, ATP is produced, intracellular levels of ADP are reduced, and oxygen requirements are lowered. Other factors that bear on the oxygen consumption of the cell are temperature, active transport and absorption requirements,[19,51] blood flow and redistribution of flow,[3,9,10] hormonal influences (e.g., of catecholamines, thyroxine, glucagon, and steroids),[45,49] the resaturation of myoglobin,[4,38,52] and the resynthesis of phosphocreatine.[7,32,41]

TABLE 1
Simplified view of lactic acid metabolism

Tissue	Vascular role	Chemical reactions	Energy consideration
Skeletal	lactic acid can diffuse from muscle to blood and from blood to muscle	lactic acid ⇵ pyruvic acid → CO_2+H_2O	ATP is produced
Cardiac	lactic acid can be extracted from blood by heart	lactic acid ⇵ pyruvic acid → CO_2+H_2O	ATP is produced
Hepatic and renal	lactic acid can be extracted from blood	lactic acid ↓ pyruvic acid ↓ phosphoenolpyruvic acid ↓ glucose-6-phosphate ↓ glucose ↓ glycogen (liver)	ATP is required

For all these reasons, we believe that it is extremely difficult to relate the level of lactic acid in the blood directly to a measure of oxygen consumption.

Let us examine the origin and meaning of the term "oxygen debt." About 50 years ago, Krogh and Lindhard[37] observed that the pattern of oxygen recovery after exercise was associated with a rapid component and a slow component. Several years later, Hill, Long, and Lupton[28-30]

introduced the term "oxygen debt" to describe the oxygen requirements that existed after the cessation of exercise. They also observed a recovery pattern that contained fast and slow components. The results of the classic study of Margaria, Edwards, and Dill[38] in 1933 led to the labeling of the fast component as the "alactacid debt" and the slow component as the "lactacid debt." Obviously, lactic acid was in some way associated with oxygen requirements after exercise.

To understand the basis for this nomenclature, it is essential to summarize briefly the state of knowledge in 1933. The isolated-muscle experiments conducted by Meyerhof[39,40] in 1920-1921 had clearly shown (1) that lactic acid accumulated with muscle stimulation and (2) that oxygen was required for its reconversion into glycogen. Meyerhof reported that 80% of the lactic acid that was produced was converted into glycogen, and the remaining 20% was oxidized to carbon dioxide and water. Because Meyerhof had stoichiometric data to support his statements, most investigators were impressed and convinced by his findings. Furthermore, it was then generally accepted that carbohydrates were the only energy source for muscular contraction. Thus, the concept of an oxygen debt evolved during an era when lactic acid was held responsible either directly or indirectly for the amount of oxygen used during recovery. It was inevitable that the terms "lactic acid" and "oxygen debt" would become inseparable.

It might appear that we are trying to dissociate lactic acid levels from oxygen-debt measures completely, and some investigators have adopted that attitude.[1,2] We believe that there is a relationship between lactic acid and oxygen debt, but that it cannot be rigidly classified into two conventional compartments.

It is well known that muscle tissue does not contain the necessary enzyme kinetic conditions to reverse glycolysis; lactic acid cannot be converted directly into glucose or glycogen and must either diffuse from the tissue or be converted back into pyruvic acid. The kidney and the liver are also unable to convert lactic acid directly into glucose. However, they can convert it indirectly because of the existence of the dicarboxylic acid shuttle system within these tissues.

Lardy and his associates at Wisconsin[17,18] have shown that tryptophan and quinolinic acid will selectively inhibit one of the enzymes involved in the process of converting lactic acid into glucose--e.g., phosphoenolpyruvic carboxykinase. Barnard[5] had the original idea of studying the influence of these inhibitors on oxygen consumption, oxygen debt, and lactic acid in exercising dogs. Using dogs that exercised under control and test (tryptophan and quinolinic acid) conditions, he found that the mean values for resting oxygen consumption, exercise oxygen consumption, and oxygen debt were reduced in the presence of the inhibitors. From a percentage standpoint, these changes represented reductions of 8%, 11%, and 44%, respectively. Blood measurements of lactic acid and glucose were not as extensive as the oxygen

measurements because of problems with the permanently implanted catheters; but, in general, the recovery lactic acid levels after tryptophan and quinolinic acid were lower. In contrast, the blood glucose levels after tryptophan and quinolinic acid rose after the first 30 min of the debt period. Unfortunately, fatty acid levels were not measured, and the contribution of this substrate to the oxygen requirement during exercise or recovery could not be ascertained.

The study of Barnard showed that blocking of the Cori cycle (conversion of lactic acid into glucose or glycogen) makes it possible to change the magnitude of the oxygen debt. The study did not tell us of the changes in intracellular ratios of NAD^+ to NADH, the ratio of lactic acid to pyruvic acid, the levels of AMP and ADP, hormonal concentrations and influences, blood-flow pattern, substrate preference, etc., but it did indicate that lactic acid can contribute to the factors responsible for an oxygen debt. Barnard and Foss (unpublished data) studied the influence of propranolol on the oxygen debt of exercising dogs. They observed that the debt decreased with blockage of the beta receptors. These studies make it clear that one should not generalize that a measure of oxygen debt is also an absolute measure of solely anaerobic metabolism.

The metabolic adaptations associated with training are superimposed on the numerous considerations previously mentioned (Fig. 2). Beginning with the classic studies of Robinson and Harmon,[46,47] it became known that the oxygen requirement, the oxygen debt,[27,47] and the blood lactic acid level are lowered for a given work task after completion of an effective training program. Besides lowering oxygen debt and blood lactic acid levels (Fig. 3) at given workloads, training will increase the capacity for oxygen consumption.[35,46] According to Robinson and Harmon,[47] the mechanical efficiency of their subjects markedly improved with training. It is reasonable to assume that the acquisition of skill in running would reduce the energy demands or requirements of working muscles.

In recent years, investigators have initiated numerous studies on the intracellular changes accompanying exercise and training. Glick and Bronk's[20,21] results from liver studies clearly show that exercise increases the activity of the enzymes associated with the Krebs cycle. The findings by Holloszy[31] indicate that trained rats have a greater capacity than nontrained rats to oxidize pyruvic acid; this enhancement was associated with greater activity of the enzymes of the electron transport system. One possible advantage of such a change would be an increase in the capacity to utilize oxygen and to produce ATP. Gollnick and King[22] found that the skeletal muscle of trained animals has more and larger mitochondria than that of nontrained animals. Studies on the enzyme (LDH) that catalyzes the reaction between lactic acid and pyruvic acid suggest that training will enhance its activity.[23,24] Training has been reported to cause an increase in the amount of myoglobin within muscle.[42] Although myoglobin has traditionally been

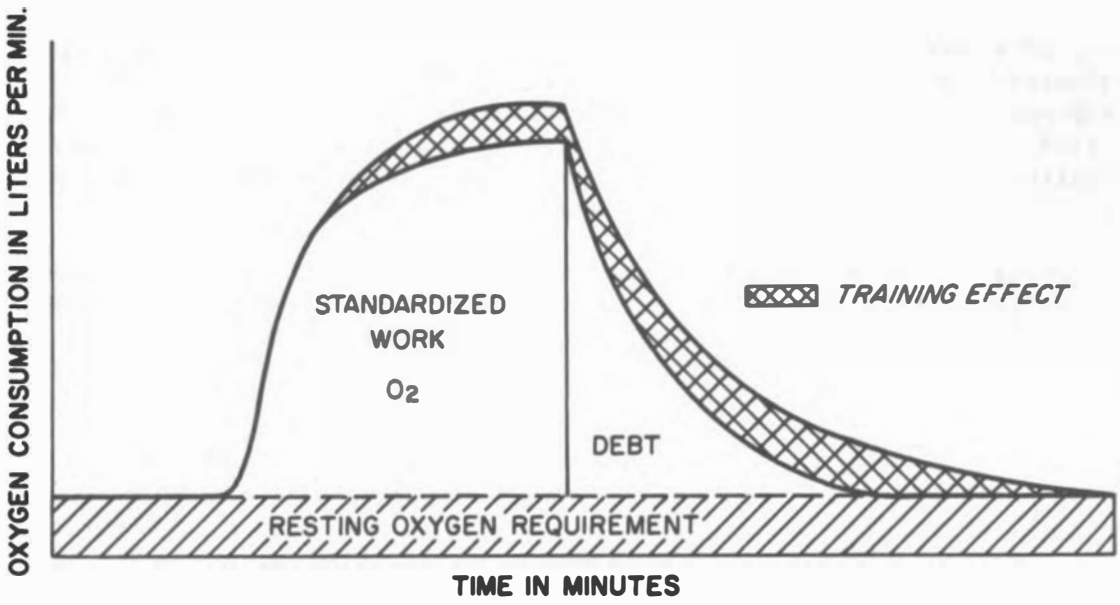

FIGURE 2
Influence of training on oxygen consumption.

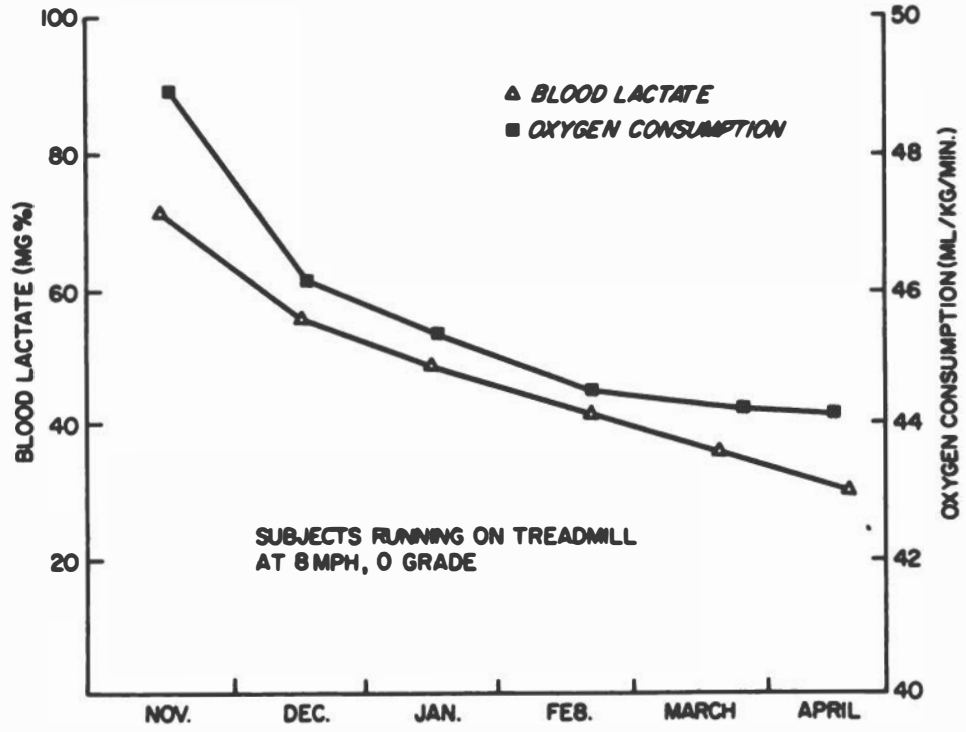

FIGURE 3
Training influences on blood lactic acid and oxygen consumption. (Based on findings of Robinson and Harmon.[46,47])

considered to be an "oxygen store," the newer concept holds that it is an oxygen facilitator.[54] By any interpretation, the ability to deliver oxygen to the cell is increased. Because total body hemoglobin appears to be higher in trained persons,[50] any change in the oxygen-delivery capacity of the blood must also be considered. Although it is accepted that training increases the number of capillaries within the heart[43] and skeletal tissue,[36,43] it is not known whether training improves the blood flow to exercising muscle. In view of recent findings on the anatomic and physiologic differences between white and red skeletal fibers,[6,16] it is possible that training alters not only the blood supply, but also the metabolic character of these fibers. A summary of these considerations can be seen in Table 2.

TABLE 2
Comparison of red and white skeletal muscle fibers[a]

Characteristics	Red fibers (example: soleus)	White fibers (example: gastrocnemius)
Work	Slow and sustained contractions	Rapid and brief contractions
Metabolic	Subcellular composition and enzyme concentration favor aerobic processes	Subcellular structure and enzyme concentration favor anerobic processes
Blood flow at rest and during electric stimulation	Higher	Lower
Capacity for vasodilatation	Higher	Lower
Oxygen consumption at rest and during electric stimulation	Higher	Lower

[a] Based on data of Beatty *et al.*[6] and Folkow and Halicka.[16]

It has been clearly shown that exercise decreases muscle ATP and phosphocreatine levels (CP in Fig. 1).[7,32] Piiper and his associates[44] tried to quantify the relationship between phosphate compounds and oxygen debt and showed in their muscle preparation that there was a relationship. (However, training was not a factor in their study.)

Using the data provided by Pardee and Ingraham,[41] Hultman *et al.*[32] estimated that 1 kg of muscle tissue would require 70 cc of oxygen to regenerate the phosphate bonds altered by exercise. Muscle is considered to be approximately 40% of the body weight and 48% of the lean body mass in man. At present, there are no available data on how training influences the amount, efficiency, and replenishment of high-energy phosphate bonds. Obviously, such research is needed.

We believe that training improves the capacity for and the efficiency of oxidative metabolism, with the production of more ATP and less lactic acid. Of the lactic acid that is produced, we think that the heart[11,34] is capable of extracting a greater percentage of accumulated blood lactate than the kidney or liver. The removal of lactic acid by the kidney and liver has been shown to be proportional to its arterial concentration.[48,49] The result of training is to decrease the amount of oxygen required for gluconeogenesis. With more ATP being produced in the cells and with an enhanced capacity for oxygen delivery to the cells, less oxygen should be required for restorative purposes. Thus, under standardized work conditions, less oxygen will be needed for exercise and recovery periods. Whether this does explain the lower oxygen debt associated with training should be the subject of further research.

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THE FORMATION, MATURATION, AND DESTRUCTION OF COLLAGEN

LEROY KLEIN

Many football injuries involve sprains, tears, and ruptures of tendons, ligaments, and menisci of joints and bones.[30] These injuries involve connective tissues that are predominantly mechanical and whose major constituent is collagen. Collagen constitutes 75%-95% of all the organic matter in tendons,[5] ligaments, and bones.[28] The tensile strength of collagenous tissues depends on the collagen content. The breaking strength of pure collagen[8] is quite high, between 10 and 50 kg/mm^2.

The repair of injuries involves the production of new collagen by incoming cells (fibroblasts) as a means of re-establishing the continuity of the collagenous framework. The new collagen is quickly attached to or mixed with the pre-existing (old) collagen and then matures over a period of months into a well-ordered structure. If the new collagen becomes excessive and remains random and poorly organized, it is considered to be scar tissue, which frequently interferes with the mobility of joints.

STRUCTURE AND METABOLISM OF COLLAGEN

The quantitative importance of the proteins of the musculoskeletal system (collagen and muscle protein) is emphasized by the observation that together they represent 80%-90% of the body's protein.[6] Collagen alone accounts for one-fourth of the body's protein; some 55% of the collagen is found in the skeleton and about 35% of it in the skin.[15] In different sites of the body, collagen differs in amount, in fibril size, and in fibril or fiber arrangement.

Collagen differs in several respects from other proteins in the body. It contains two amino acids--hydroxyproline and hydroxylysine--that are not found in other proteins. Some of the essential amino acids (methionine, phenylalanine, and tryptophan) are found in very small amounts or not at all in collagen. Hydroxyproline and hydroxylysine are made after the collagen molecule has been synthesized by the

addition of hydroxyl groups to some of the polypeptide-bound proline and lysine.[32] Thus, neither hydroxyproline nor hydroxylysine can be used in the synthesis of collagen.

The ultimate source of new collagen is the specialized cells,[4] fibroblasts and osteoblasts. A soluble molecular precursor of collagen is synthesized intracellularly on the microsomes[32] and is then extruded[25] into the extracellular medium as an elongated rod, approximately 2800 Å long and 14 Å wide with a molecular weight of 300,000. Collagen is one of the largest macromolecules synthesized biologically. The completed collagen molecule, consisting of three polypeptide chains, will mature and cross-link extracellularly to form insoluble collagen fibrils and eventually fibers. The degree of internal and external cross-linking affects the physical properties of collagen; e.g., the tensile strength of collagen appears to increase with the number of intermolecular cross-links. The increase in number of cross-links appears to be much slower[14,17] than was heretofore recognized. The architectural pattern of the tissue, as created by the organization of collagen fibrils into fibers and fibers into bundles, also affects its physical and mechanical properties.

Of particular significance to injured tissue is that collagen is relatively inert metabolically,[23] in the sense that it is nonrenewable, little collagen being destroyed and replaced by new collagen. Thus, any injury requires a substantial increase in synthesis of new collagen if structural continuity is to be re-established. However, it has been demonstrated that pre-existing (old) collagen of rats could be reused in the formation of "new" collagen and in the remodeling of new and old collagen.[14,19] It is not known whether that can occur in man. Because collagen is metabolically inert, collagen that is made during growth in childhood and adolescence is still present in adulthood and old age. Collagen stays around long enough to age, and various physical properties (such as fibril size, crystallinity, and solubility) change with age.[7] This aging aspect of collagen is believed to be partly responsible for the physical slowing down of athletes with age.

DEGRADATION OF COLLAGEN

Measurements of urinary hydroxyproline have provided a means for studying collagen degradation in animals and humans. The finding that in adolescence[10,35] and infancy[18] the excretion of urinary hydroxyproline is much higher than in adulthood has suggested that urinary hydroxyproline is derived in part from newly synthesized collagen. This has been more clearly shown by isotopic studies.[21] From additional isotopic data it has been concluded that in young adult rats[27] and adult monkeys[1] approximately 57%-70% and 80%-86%, respectively, of the urinary hydroxyproline comes from insoluble collagen, whereas only 30%-43% and 14%-20% comes from the breakdown of newly synthesized collagen. Recent studies[13] on 20-month-old rats that were labeled with ^{3}H-proline at 1-2½ months of age have demonstrated that only 10%-25% of urinary hydroxyproline is derived from old, insoluble collagen, and 75%-90% from the synthesis of new collagen.

The quantitative relationship of urinary hydroxyproline to total collagen degradation, irrespective of its biologic source, has been determined[33] to be approximately 20%-25%. Thus, the major amount of urinary hydroxyproline appears to be derived from *de novo* collagen synthesis. However, the breakdown of new, salt-soluble collagen could occur intracellularly or extracellularly. The administration of penicillamine to prelabeled rats caused a sixfold to eightfold increase in the neutral salt-soluble collagen pool but was without effect on the total amount or specific radioactivity of urinary hydroxyproline. These data indicated that urinary hydroxyproline was not derived from the extracellular breakdown of salt-soluble collagen and suggested that newly synthesized collagen was degraded intracellularly, after the hydroxylation step of protocollagen and before its extrusion from the fibroblast.

EFFECT OF TRAUMA AND NONTRAUMATIC CONDITIONS ON COLLAGEN DEGRADATION

The degradation of collagen, as seen by the excretion of urinary hydroxyproline, is markedly increased in traumatic states, such as burns,[16] acute paralysis,[12] and fracture healing.[12] In widespread bone disorders--such as hyperparathyroidism, hyperthyroidism, and Paget's disease--much higher levels of urinary hydroxyproline have been found, compared with those observed in acute paralysis or fracture healing.[20] Urinary hydroxyproline data from astronauts in the weightlessness of space[34] suggested that a significant increase did not occur; the absence of gravity apparently did not have an effect, although disuse denervation caused a marked breakdown of bone collagen in man. An effect of starvation on men aged 18-27 was a small increase in urinary hydroxyproline.[15]

When rats were placed under the influence of low-frequency mechanical vibration for 3 hr daily over a period of 3 weeks, there was no effect on urinary hydroxyproline.[31] However, when rats underwent marked physical exercise (swimming for 90 min), there was a definite increase in urinary hydroxyproline.[2] Those results suggest that fatiguing exercises are a stimulus to collagen metabolism. The urinary hydroxyproline level appears to be a sensitive measure of small changes in collagen metabolism in the normal, abnormal, and traumatized man.

RELATIONSHIP OF COLLAGEN TO FOOTBALL INJURIES

An important, but unknown and highly variable factor in football injuries, particularly those involving ligaments and tendons, is the rate of healing of the injured site, with return of tensile strength to the original level and the development of scar tissue. Excessive scar formation in tendon healing will limit the motion of the restored tendon; thus, the chief problem is usually not the repair process itself, but limiting the amount of scar tissue. Any interference with the blood supply to the tendon will also limit the repair process.

In either tendon or ligament healing, the initial injury is the stimulus to the onset of collagen production as part of the repair process. The production of collagen appears to be a self-limiting process. Excessive fibrous tissue can be further stimulated by subsequent trauma (either surgically or athletically induced), excessive motion or tension,[26] or infection. Partial removal of the scar tissue is believed due to the remodeling of the repaired area. What starts or controls the remodeling process is not known, but the reuse of citrate-soluble collagen[14] may be involved.

The amount of difficulty in repairing tendons and preventing adhesions varies[3] with the type of tendon--extensor or flexor. The extensor tendons are more superficial and have less tendency to retract. They have a more diffuse blood supply[29] via the paratenon than the flexor tendons, which depend on a few small blood vessels that course through the tendon sheath. Flexor tendons have little regenerative ability and are subject to much scar formation. But the prognosis is good in injuries to extensor tendons, which have good regenerative ability and undergo only minimal scar formation.

Scar formation could be prevented (although healing would also be hindered) by inhibiting collagen production, once repair of the injury gap were almost complete, in one of three ways.[24] The intracellular synthesis of collagen could be blocked with protein inhibitors like actinomycin D and cortisone; however, these compounds have severe systemic effects. Another approach is to block collagen production by inhibiting the hydroxylation of proline and lysine via specific iron chelators or scurvy, which is caused by vitamin C deficiency; neither of these methods could be used safely. A third approach is to interfere with the maturation of soluble collagen to the insoluble state. The maturing of collagen is the result of the formation of new intramolecular and intermolecular cross-links, which can be blocked by administering beta-aminopropionitrile[11,22] or penicillamine.[9] Such treatment would prevent the new collagen of scar tissue from becoming insoluble; thus the scar would be weak and the increased solubility would permit removal of all or part of the collagen. The use of beta-aminopropionitrile in man[11,22] has resulted in some toxic symptoms, and some side reactions also occur with the administration of penicillamine.[9] It is not known whether, on cessation of treatment, the soluble collagen will readily mature to insoluble collagen and thus negate the early beneficial effects. The use of penicillamine or beta-aminopropionitrile locally at the site of injury, instead of systemically, may circumvent some of the side reactions. Of the various ways of controlling the physical properties of scar tissue, the latter approach appears to be the most specific and the safest.

Outside of the use of bacterial or mammalian collagenases to destroy insoluble collagen, there is no pharmacologic agent available to solubilize mature collagen. Biologically, it appears that fibrous collagen,[14] which is not strongly cross-linked intermolecularly, can be solubilized; but it is not known whether heavily cross-linked, insoluble collagen can be solubilized as a means of removing scar tissue.

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STRUCTURAL AND FUNCTIONAL CHANGES IN MUSCLE AFTER EXERCISE AND TRAINING

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Skeletal muscles are the immediate effectors in the performance of work, so it is only logical that they should have been the subject of numerous investigations to determine what adaptations, if any, occur in them during training to enhance their functional capacity. Perhaps the most overt response of skeletal muscle to training is an increase in size. To be sure, hypertrophy does not occur every time a muscle performs work, but only when the intensity of the work exceeds that to which the muscle is normally exposed. From a morphologic standpoint, it appears that three changes (or combinations of the three) could take place to produce the increase in muscular size in response to training: (1) an increase in the sarcoplasmic fraction, (2) alterations in the amount of structural material, and (3) changes in the number and/or size of the contractile proteins.

The search to determine which changes may occur in response to exercise is old. One of the earliest investigations into the phenomenon was that reported by Morpurgo[38] in 1897. He removed the sartorius muscle from one leg of each of two dogs, and then removed the contralateral sartorius muscle after a 3-month training program of arduous running. Comparison of the muscles taken before and after exercise revealed that the average fiber diameter had increased by 55%, whereas the total number of fibers was unchanged. These results were later confirmed by Thörner[47] and Hoffmann.[31] More recently, Holmes and Rasch[34] were unable to find any change in the number of myofibrils per fiber in the sartorius muscle of exercised rats, nor any evidence of hypertrophy in this muscle after a rather mild 7-week training program. On the basis of the work of Morpurgo, it has generally been assumed that the increase in cross-sectional area of muscle after training was due to an increase in the sarcoplasmic fraction of the muscle, not the addition of new fibers to the muscle.

Helander[26] provided the first indication that changes in components other than the sarcoplasm might account for the increased muscular size after training. He found that after 4 months of running 1 km/day the protein of the myofilament fraction of the gastrocnemius muscle of adult guinea pigs was 15% greater than that of the sedentary controls.

However, total sarcoplasmic protein of these muscles was not significantly affected by the training. Denny-Brown[10] reported that a twofold increase in the myofibrillar count accompanied a 25% hypertrophy in the soleus muscle of the cat. The hypertrophy was induced by removal of the gastrocnemius from one leg of the cat while leaving the soleus intact. The soleus muscles from the experimental and control sides of the cats were examined 3 months after this surgical treatment. Similarly, Goldspink[15] found that a threefold to fourfold increase in myofibrillar count accompanied the work-induced hypertrophy in the biceps brachii of mice; in these experiments, a 30% increase in fiber area was produced by forcing the mice to lift weights to obtain their food.

Goldberg[14] has attempted to elucidate the mechanisms by which hypertrophy occurs by applying the technique of sectioning the tendon of the gastrocnemius muscle in the rat. In this preparation, the weight of the soleus and plantaris muscles of the experimental leg is significantly greater than that on the contralateral side 24 hr after surgery, with the maximal difference between control and experimental legs being reached in 5 days. Thereafter, growth of the hypertrophied muscles parallels that of the controls. Average fiber diameter in these hypertrophied muscles was 24% greater than that in controls. This compensatory hypertrophy also occurs in hypophysectomized animals and thus is independent of growth hormone. In the hypophysectomized rat no growth occurs in normal muscle after hypophysectomy or in the muscle of the surgically treated leg, once the maximum hypertrophy has been attained after the fifth day. Total increase in fiber diameter was 29% in these animals, or approximately the same as that occurring in the normal rat. Goldberg[13] has also shown that an increased synthesis of contractile protein accompanies the compensatory hypertrophy in the soleus and plantaris muscles of hypophysectomized rats after sectioning of the gastrocnemius tendon. In these studies, injected leucine ^{14}C was found to be incorporated into the myofibrillar protein in direct proportion to the increase in muscle weight. He also found, however, that it was being incorporated into the other components of muscle--such as the mitochondria, calcium-binding grana, microsomes, and the soluble protein--in equal proportion to the change in myofibrillar protein. Thus, there was no disproportionate production of one muscular component at the expense of the other; the result was the development of additional muscular tissue with normal functional capacity.

The mechanisms producing cardiac hypertrophy have been found to be similar to those producing hypertrophy of skeletal muscle. It appears that the basic processes producing the increases in size are similar, if not identical, for both tissues. The incorporation of ^{14}C-labeled amino acids into myocardial protein during hypertrophy occurs at rates similar to those during skeletal-muscle growth.[22,45] Richter and Kellner[44] have also shown that a general increase in the fiber diameter of heart muscle accompanies its hypertrophy. This hypertrophy is the result of a large increase in the number of myofibrils per fiber. Examination of these myofibrils by electron microscopy revealed that the basic geometric arrangement of the filaments was unchanged by

hypertrophy. These results suggest that hypertrophy occurs from an addition of myofilaments to the muscle through the formation of new myofibrils or possibly by addition to pre-existing myofibrils. In support of this concept, Carney and Brown[6] found that the diameters of the filaments in normal and hypertrophied rat myocardium were similar. Thus, the increase in myofibrillar protein does not occur as the result of an increase in size of the existing filaments, but by the addition of new filaments similar in size to those already present.

It has also been reported that in animals the vascularization of both heart and skeletal muscle increases during training.[43] Presumably, this occurs by an opening of existing capillaries, and not by the formation of new ones. Whether this effect occurs in human muscle is unknown. However, Grimby *et al.*[21] did not find any differences in blood flow through the muscles of trained and untrained subjects at rest or during work.

Increases in strength and work capacity usually accompany training. The increase in strength can be accounted for by the change in muscle size and perhaps also by an improved capacity to use all the muscle fibers. The question remains of whether any adaptations occur in the metabolic apparatus to increase functional capacity. Because of the need to obtain tissue for analysis, these investigations have been conducted almost exclusively on animals.

For the most part, training has been found to have little or no effect on enzymes of the glycolytic pathway, such as aldolase,[24] lactic dehydrogenase,[16,19,20,49] phosphorylase,[20] hexokinase,[42] and alpha-glycerophosphatase.[9] This lack of a positive adaptive response may be due to such factors as the normal capacity of the system, the choice of enzymes to be studied, and the nature of the work used in the training programs. The capacity of this system seems to be quite high; very high levels of blood lactate occur during heavy work, even in untrained subjects. Thus, further augmentation of this system may not be needed. As for the choice of enzymes for study, those which have been studied are not rate-limiting to the total system, whereas phosphofructokinase, which is rate-limiting, has not been studied. And finally, most of the investigations have used endurance swimming or running as the exercise for training. It is commonly known that such exercise relies primarily on the oxidative system; therefore, it cannot be expected to stress the anaerobic system.

The effect of training on the aerobic capacity of skeletal muscle is somewhat more controversial. Perhaps the first report on this subject was that of Chepinoga,[8] who found a significant increase in the succinic dehydrogenase activity of rabbit skeletal muscle after a training program of treadmill running. However, Hearn and Wainio[25] and Gould and Rawlinson[20] found no change in this system in rat skeletal muscle after training programs of swimming 30 min/day for various periods. More recently, studies by Holloszy[33] have demonstrated that the oxygen

uptake, enzyme activity, and total protein of the mitochondrial fraction of rat skeletal muscle were significantly increased by a strenuous program of treadmill running. Like Hearn and Wainio[25] and Gould and Rawlinson,[20] he found no such adaptive response in rat muscle after a 4-week program of swimming 30 min/day. Pattengale and Holloszy[41] also found a significant increase in the myoglobin of rat muscle after training. This increase occurred specifically in muscles that were actually used during exercise and did not occur in all muscle as a general body response to training. A functional role for this increase in myoglobin may be to aid in the transport of oxygen from the blood to the aerobic system.

In support of the findings of Holloszy, we[18] have found the concentration, and perhaps size, of mitochondria in rat skeletal muscle to be increased after a strenuous training program of treadmill running. The approximately twofold increase in mitochondria per unit area of muscle tissue closely parallels the enzymatic and protein changes reported by Holloszy. These findings indicate that the aerobic capacity of skeletal muscle can be enhanced by training when the exercise program is sufficiently severe and long.

Considerably less information is available on the immediate effects of exercise on muscle structure and function. However, Altland and Highman[1,30] have shown in a series of papers that prolonged exercise can produce structural damage to skeletal muscle. Skeletal muscle of rats was examined histologically either after a single 16-hr exercise bout or after three to five daily 6-hr exercise sessions. Tissue damage from this exercise was characterized by the existence of scattered necrotic or partially necrotic muscle fibers, with some of these fibers having been replaced by mononuclear cells. Other changes after exercise included "fragmentation and atrophy of scattered muscle fibers, indistinct striation, cytoplasmic basophilia, marked proliferation of muscle nuclei, formation of multinucleated giant cells, and focal interstitial fibroblastic proliferation and infiltration by various types of inflammatory cells."[30] Increases in serum enzymes, some of which originated in skeletal muscle,[12] also occurred after these exercise bouts. This leakage of enzymes from the muscles into the blood is probably a direct manifestation of the tissue damage. Similar changes in serum enzymes have been observed in humans after strenuous exercise[11,27] and may also be an indication of damaged skeletal muscle. Training before the exercise test eliminated or greatly reduced the damage to the tissue and the rise in serum enzymes in the rat. This tissue damage may be responsible for the soreness that occurs in skeletal muscle, particularly in unfit persons, after exercise.

We have found that exhausting exercise produces a general tissue edema and massive swelling and cristae degeneration of the mitochondria of rat skeletal muscle.[18] These changes were temporary; they were not found in muscle from rats killed 24 hr after exhaustion. They also

depended on the severity of the exercise used to produce exhaustion, occurring after running but not after swimming. The mitochondrial changes after exhausting running resemble those occurring in heart and liver after ischemia, acute and chronic hypoxia, and permanent anoxia.[4,5,7,28,37,40,46] These similarities suggest that hypoxia may occur in skeletal muscle during exhausting exercise.

We have also examined human skeletal muscle after exhausting exercise.[17] We took samples from the vastus lateralis before and after working to exhaustion at 75%-80% of maximal aerobic capacity on the bicycle ergometer. No ultrastructural damage was observed in these muscle samples.

When a muscle or group of muscles works at a given rate, a point is ultimately reached when this work cannot be continued. The time required to produce this fatigue depends on the intensity of the exercise. The inability of the muscles to continue indefinitely at given work outputs may result from the failure of the metabolic processes to provide a continual supply of adenosine triphosphate (ATP) to the contractile apparatus. The decrease in muscular ATP and the depletion of phosphocreatine during heavy exercise supports this concept.[32,35] However, there is no evidence that the capacity of the metabolic systems to produce ATP decreases during exercise. On the contrary, J. Karlsson *et al.* (personal communication) found that lactic dehydrogenase activity of skeletal muscle increased after exhausting exercise. What effect the swelling and cristae degeneration observed in rat muscle mitochondria after exhausting running may have on metabolic capacity is unknown. However, it is well known that, in isolated mitochondria, disruption of the basic structural configuration such as occurs with swelling can loosen or completely uncouple oxidative phosphorylation.[48] Work capacity of muscle would probably be reduced if these changes occurred during exercise.

One factor that may contribute to the onset of fatigue is the unavailability of appropriate energy compounds to the metabolic systems. This may be especially important during heavy exercise, when muscle glycogen is rapidly consumed and almost completely depleted at exhaustion.[17,29] Fats can serve as energy sources during exercise,[23] and they are mobilized even during heavy exercise.[17] However, it appears that muscle glycogen is essential for high-intensity work,[29] with work time depending on the initial glycogen concentration of the muscle.[2]

A concept that is often used to explain the failure of muscles to respond after prolonged activity is that of motor end-plate fatigue, in which fatigue is seen as resulting from failure of the neural impulse to cross the myoneural junction and depolarize the muscle fibers. Experimental results of Merton,[36] Brown and Burns,[3] and Naess and Strom-Mathisen,[39] however, have shown that normal action potentials from motor nerves occur in muscle at exhaustion but fail to produce contraction. Thus, it appears that fatigue is due to some failure in the excitation-contraction coupling, rather than to lack of stimulation of the fibers.

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THE EFFECTS OF ENVIRONMENT ON PERFORMANCE

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The major natural environmental factors that influence physical, psychomotor, and mental performance are heat, cold, and altitude. Heat and cold are important because maximal performance requires that body temperature be maintained within a rather narrow range, only a few degrees. Altitude is important because maximal performance is limited by the amount of oxygen supplied to the tissues, and the available oxygen in the air decreases with increased altitude.

There are many nonenvironmental factors that modify the amount of deterioration in performance caused by environmental factors, including age, sex, body build, physical fitness, nutritional status, health status, clothing and protective equipment, fatigue, previous exposure (to heat, cold, etc.), and knowledge of ways to minimize the effects of exposure.

The multiplicity of interactions among these many factors precludes a comprehensive examination of the effects on performance of all the possible combinations of factors. This discussion will therefore be limited to a general review of the effects of cold, heat, and altitude on performance.

PERFORMANCE IN HEAT

Excessive environmental or metabolic heat can be the causative factor in several types of heat disorders. All heat disorders are sufficiently incapacitating to prevent any effective competitive performance; some may persist for hours or days, and one usually leads to death. The literature on heat illnesses has recently been reviewed[25] and will not be discussed in detail here. It should suffice to point out that patients with heat illness should receive medical treatment, that no heat illness should be dismissed lightly, and that heat illness may occur in susceptible persons under environmental conditions that are considered extreme. Fatal heat stroke[39] has occurred in young men who had been working hard for 1-2 hr at temperatures of 86-92 F (30-33.3 C). Those who saw the All-Star baseball game in St. Louis during the heat

wave in July 1966 observed first-hand some of the performance problems associated with high temperatures. During the 6 days of the heat wave, more than 500 excess deaths occurred in St. Louis. Many of the victims were young adults and middle-aged persons who were normally healthy.[22] How many heat illnesses occurred that did not end in death will never be known, but thousands of persons, no doubt, were incapacitated by the heat.

Literally hundreds of studies have been conducted in which the physiologic and psychologic effects of various combinations of environmental heat and work were observed. The effects of heat on performance are influenced by the capabilities and limitations of the individual (human factors), the environment to which he is exposed (environmental factors), and the specific demands of the activity (task factors).

I will discuss the effects of heat on performance in two broad categories: (1) physical performance, or performance that involves mainly muscular work, and (2) psychologic performance, or performance that involves receiving and processing information, making decisions based on this information, and making other appropriate responses.

Physical Performance

The major factors associated with heat that influence the ability to perform physical work are the level of heat stress, the physical condition of the worker, and the state of acclimatization to heat. Physiologic strain accompanying light work, as expressed by the increase in pulse rate, will increase by 30%-50%, (1) if 15 min of hard work is done, or (2) if the air temperature is increased from 70 to 115 F (21.1 to 46.1 C), or (3) if the relative humidity (RH) in a hot climate is increased from 20% to 65%.[9]

Productivity during a working day is strongly influenced by the air temperature and RH. Output of ore and rock drilling may be only 50% of normal when the temperature in the mines reaches 92-95 F (33.3-35 C) with an RH of nearly 100%.[12,48] In these hot and humid conditions, the predicted incidence of fatal heat stroke is about 1 per 1000 workers per year.[47] On the basis of laboratory studies, it has been proposed that for an 8-hr daily work routine the environmental conditions should not exceed a dry-bulb temperature (DB) of about 100 F (37.8 C) and 50% RH for light physical work, 95 F (35 C) DB and 55% RH for medium physical work, or 90 F (32.2 C) DB and 60% RH for heavy physical work.[27] Beyond these limits, many workers will suffer excessive strain, with high pulse rates and high body temperatures. Even below these limits, some of the less heat-tolerant workers may become heat casualties.

Tolerance times for moderate work (282 kcal/hr) have been worked out for more extreme heat stress.[43] The average tolerance time was 87 min

at 99.5 F (37.5 C) DB and 75% RH, 44 min at 104 F (40 C) DB and 80% RH, and 26 min at 113 F (45 C) DB and 80% RH. The physical-fitness index on a pack test decreased by about 15% when the air temperature was increased from 65 to 90 F (18.3 to 32.2 C) with 75% RH.[32]

Most types of athletic performance that require a high rate of energy expenditure have an intermittent character. The short bursts of heavy work separated by short rest periods could be accomplished with less physiologic strain in severe heat than could continuous work.[26] However, a frequently overlooked source of heat stress is solar radiation. Being in full sunshine will produce about the same amount of heat stress as will an increase in air temperature of 10-15 F (5.6-8.3 C).[24]

Acclimatization to heat greatly enhances the ability to tolerate work in heat.[1,2,23,25] If a team is to play in heat, it must practice in the heat for several days to build up a tolerance that will enhance performance.

Psychologic Performance

Three recent reviews of the effect of heat on psychologic performance have all demonstrated that there is a positive but complex and poorly defined relationship between heat and performance decrements.[3,34,35] The extent of deterioration of performance is determined by many factors, including the severity of the heat stress, the length of exposure, the demands of the task, and the skill, motivation, and capabilities of the person.

Performance on tasks requiring vigilance and visual skills decreases over time. A hot environment aggravates the rate of deterioration and the total decrease.[3,4,6,10,28] A funneling of the area of awareness toward the center of the visual field in heat stress is an especially interesting observation because of its implication for many types of job, including the sports of football and basketball, in which wide visual fields are an asset.[10] It has also been observed that decrease in performance occurs at a lower level of heat stress if the task is very complex than if it is simpler. What part of the deterioration results from changes in visual function and what part from the irritability, discomfort, and distraction of the hot environment cannot yet be identified.

Many tasks that require several components of manipulative skills have been studied under a wide range of heat stress. These components include aspects of simple and complex reaction time, speed of arm movement, hand and arm steadiness, tracking skill with and without a handle load, rotary pursuit, and hand and finger dexterity.[3,8,13,20,33-36,41,42,45]

Performance is generally best at neutral or comfort conditions, and a decrease in performance becomes apparent as the heat load is increased.[41]

However, a moderately severe heat load is necessary to induce change in performance. A 30% deterioration in performance on a complex tracking task occurred when the tests were performed in a hot and humid environment.[36] Performance was more sensitive to an increase in humidity than to an increase in air temperature. In general, the greater the physical activity required or the more complicated the task, the less will be the heat stress at which a decrement in performance first appears.

Studies on the effects of heat stress on mental performance have shown deterioration ranging from almost none to significant amounts.[14,18,29,35,44,46] As with other types of performance, the decrease becomes progressively greater as the total heat stress and the period of exposure are increased and as the task is made more complex. The upper thermal tolerance limit for unimpaired performance seems to be lower for mental than for physical tasks.[46]

Apparently, heat stress has little effect on learning ability, although the learner may think his achievement is poorer.[30] However, under heat stress, as under most other stresses, variability in performance is increased.[5,19] This, of course, could have a significant impact on the final outcome in many individual or team sports.

PERFORMANCE IN THE COLD

Exposure in cold weather can have serious health consequences by causing excessive loss of body heat (hypothermia) or freezing of the tissues. Except under unusual circumstances, cold-weather clothing prevents a serious reduction of internal body temperature. However, even a small drop in body temperature may stimulate shivering, which can interfere with precisely coordinated muscular activities. Consequently, performance of most types deteriorates during shivering. And, of course, heavy body clothing has a hobbling effect.

Of more practical importance, however, is the problem of keeping the hands warm without interfering with their use. A commonly experienced, but not frequently recognized, phenomenon is the cooling of the hands sufficiently to reduce finger dexterity while the rest of the body remains comfortably warm.[15,16,40]

Several controlled laboratory studies have been conducted to establish the effects of cooling on two important hand functions--tactile discrimination and finger dexterity. The ability to distinguish the distance between two points with the finger tips decreased by a factor of 4 when the finger skin temperature was reduced from 87 to 40 F (30.6 to 4.4 C), even while the body was kept comfortably warm.[31] Loss of tactile discrimination was roughly proportional to the decrease in finger skin temperature. When the hands were rewarmed, the return of tactile sensitivity lagged behind the return of skin temperature, so that the sensation of warmth did not truly indicate that function had returned.

Studies of manual dexterity and hand cooling show much the same relationship between skin temperature and hand function.[15,16,40] When the hand skin temperature was lowered from 70 to 45 F (21.1 to 7.2 C) in 15 min, dexterity (knot-tying) was reduced by about 25%; but when the cooling time was 35 min, dexterity was reduced by 50%.[15] The return of function when the hands were rewarmed also was slower when the hands had been cooled more slowly, and some loss of function was present even after the skin temperature had been returned to normal. An important observation was that a heated muff was more effective in rewarming than warm air.[16]

There seem to be some ethnic differences in cold tolerance. Field experiences in Korea suggested that cold injuries were proportionally more frequent and more severe in the Negro than in the white troops.[17] In laboratory studies of cold exposure, the finger skin temperature was several degrees lower in the Negro than in the white subjects. The spontaneous rewarming of the finger, which was frequently observed in the white subjects, was not present in the Negro.[38]

PERFORMANCE AT HIGH ALTITUDE

The scheduling of the Olympic games in Mexico City in the summer of 1968 raised the questions in many countries of whether athletes who had trained at lower altitudes would be at a disadvantage at the altitudes of Mexico City and whether training and competition at the higher altitudes would have any deleterious effects on subsequent performance at low altitudes. Several studies during the last few years have considerably clarified the relationships between increase in altitude and decrease in performance.[11,21,27] (Denver is the only city with a professional football team at an altitude that might affect performance.)

In considering the problem of altitude and performance, it is necessary to separate maximal aerobic work capacity (i.e., hard work that can be sustained for only a few minutes expressed as maximal oxygen utilization capacity) from light or moderate work, which can be maintained for up to an hour or several hours (and may be at a rate of only 25%-50% of the maximal work capacity). Maximal aerobic capacity decreases with increased altitude from sea level.[11,17,37] The rate of decline in capacity becomes sharply greater at altitudes above about 5000-6000 ft. At 5000 ft, maximal aerobic capacity is about 95% of sea-level performance; at 13,000 ft, it is about 70%; and at 24,000 ft, it is only about 40%. A general rule of thumb is that maximal aerobic capacity decreases by about 3% for each 1000-ft increase in altitude above 5000 ft. Below 5000 ft, the decrease is not large and would probably be critical only under some special circumstances. The deterioration of maximal aerobic capacity is reversed during a stay and training at higher altitudes, but the improvement is slow. For example, the period for which the subjects in one study could pedal the

bicycle ergometer at very hard work levels was about 88% of that at sea level for the first week or two at 13,000 ft, but had returned to 100% by the end of the third week.[11]

Hyperventilation is another characteristic of working at higher altitudes. Pulmonary ventilation may increase by 35%-50%, so that the work of breathing may use up a large part of one's available oxygen.

Heart rate at maximal work is not influenced by altitude. That is not surprising, inasmuch as maximal heart rate should be reached if maximal work levels are achieved, regardless of other factors (except possibly the degree of physical fitness).

Running times for 1 mile and 2 miles at sea level and at several altitudes were compared.[11,17] At 13,000 ft, speed was 75%-80% of that at sea level, and at 7500 ft it was 92%-95%. The difference in running was about the same for 1 and 2 miles and was of the same magnitude as the percent decrease in maximal aerobic capacity for similar altitudes. From these data, it could be concluded that a football team that trains and competes at altitudes near sea level will not perform as well at 5000 ft as at sea level.

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THE EFFECTS OF DEHYDRATION ON PERFORMANCE

SID ROBINSON

Dehydration, excessive reduction of the body's water content, is known to affect the work capacities of men in two major ways: (1) it interferes with temperature regulation and may result in heat pyrexia and even heat stroke in men attempting to work for long periods in hot environments; and (2) even in moderate degrees (4%-5% of body weight), it significantly reduces the work capacity in short periods (3-6 min) of exhausting work, in which temperature regulation is not a limiting factor. Both effects may interfere with athletic performance; in the first case, lives are occasionally endangered.

Normal men may be dehydrated rapidly during work in hot environments if water lost through the sweat glands is not replaced by drinking. The rate of sweating may vary up to 5 lb/hr (2.3 kg/hr), depending on the intensity of the work, the effective temperature of the environment, and the clothing worn. Large football players may sweat more than 5 lb/hr (2.3 kg/hr) during practice sessions on a hot summer day, particularly if they wear the full football uniform. A 200-lb (90.9-kg) man sweating at 3 lb/hr (1.3 kg/hr) without drinking water would be dehydrating at a rate of 1.5% of his body weight per hour. A 150-lb (68.2-kg) man who is well acclimatized to work (3.5 mph; 2.5% grade) in severe heat (dry-bulb temperature, DB, 50 C; wet-bulb temperature, WB, 28 C) may sweat at an average rate of 3 lb/hr (1.3 kg/hr) for 6 hr, provided he keeps up his water and salt balance by drinking 0.2% NaCl solution in water frequently during the work period.[13] Without replacing water losses, even acclimatized men become seriously dehydrated, overheated, and exhausted long before completing such an exposure.

WORK IN HOT ENVIRONMENTS

Dehydration significantly reduces the tolerance of men for prolonged work in hot environments, whether the loss of body water is due directly to failure to replace water lost through the sweat glands and kidneys or is secondary to sodium chloride deficiency due to failure to replace salt lost in sweat and urine.[1,12] This effect on the tolerance of men for

work in the heat undoubtedly depends largely on reductions in sweating[11,12] and circulating blood volume.[1,10] Both interfere with temperature regulation and may result in heat pyrexia and even in heat stroke in men working in the heat. Under those conditions, high rates of sweating are required for evaporative cooling, and increased, rather than decreased, plasma volumes are required to meet the great demands for circulation to the working muscles and for heat transport to the skin. Dehydration may well contribute to heat stroke and other heat illnesses in prolonged athletic performances in hot weather. Notable examples are collapses of distance runners in championship races in hot summer weather and fatal and near fatal attacks of heat stroke among college and high-school football players during long practice sessions in August and September.

The effect of dehydration in reducing sweating during work in the heat is a powerful and consistent influence, which persists in spite of increased thermal stimuli associated with the elevated skin and rectal temperatures that result from reduced evaporative cooling.[1,11,12] If men walk on a treadmill at 3.5 mph up a 2.5% grade in dry heat (DB, 44 C; WB, 26 C) without drinking water, their rates of sweating in the first hour will be an average of 10% lower than in similar exposures in which they replace lost water by drinking.[11] If they continue the exposures for 4 hr without drinking, they will be dehydrated by 3%–4% of body weight, and sweating rates will be an average of about 20% lower than in exposures in which they deliberately drink water to replace sweat and urine losses. In addition to this moderate dehydration and reduced sweating, evaporative cooling of the skin is reduced, skin and rectal temperatures are raised, and further strain on the circulation is indicated by high heart rates and in many cases symptoms of syncope. More severe dehydration increases the symptoms of strain and results in even higher body temperature.[12]

During work in hot environments, an expansion of the blood volume reduces strain on the circulation and consequently helps to maintain circulatory stability and temperature regulation. Numerous workers have reported that the circulating blood volumes of men increase above basal values during actual exposure to heat.[4,5,7,9,14,19] We[14] and others[3,18] have found that men who are acclimatized to the heat have normal basal blood volumes during rest in a cool environment. We found increases in blood volume averaging 13% during 2- to 6-hr exposures to work in severe heat (50 C) in which water balance was maintained by drinking 0.2% NaCl solution. Bass *et al.*[4] found that blood volumes of men increased by 15% by the 5th day of continuous acclimatization to severe heat, and by the 14th day they had returned to control levels. The increase was due largely to expansion of the plasma volume. Expansion of both plasma and total extracellular fluid volumes was found to depend on retention of sodium chloride by kidneys and sweat glands.

In contrast with the favorable effects of increased blood volume of normal men during work in hot environments, dehydration reduces their

blood volumes when they can least tolerate it. Adolph[1] found in acutely dehydrated men in the desert that reductions in circulating plasma volumes were 2.5 times as great as would be expected if water loss from the plasma were in the same proportion as the loss from the whole body. More recently, Kozlowski and Saltin[10] have confirmed that finding, and we found that the plasma volumes of men dehydrated by an average of 3% of body weight were reduced by an average of 6.5%.

The effects of dehydration on temperature regulation and circulatory stability of men during work in the heat were studied in great detail by Adolph[1] and by Pitts *et al.*[12] They found that water deficits as low as 1%-2% of body weight caused measurable evidence of increased circulatory strain, as indicated by increases in the heart rates and rectal temperatures of men resting or working in hot environments. The strain, under otherwise constant conditions of metabolic rate and heat stress, increased linearly with water deficit. Accompanying the increased heart rates of the men associated with dehydration were parallel increases in rectal temperature, indicating a failure of the circulation in its function of heat transfer from tissues to skin. Such failure, accompanied by characteristic symptoms of decreased work output, drowsiness, faintness, dyspnea, dry mouth, and restlessness, Adolph called "dehydration exhaustion."

A resting man who is acclimatized to a hot climate will maintain water balance accurately by drinking enough water to keep his thirst satisfied, and the water content of his body, measured each morning before the day's work begins, will be remarkably constant. In contrast, Dill[8] and others[2,12] have found that thirst does not always cause a working man to keep his water intake up to its output. That is the case particularly in unacclimatized men, who may, in working in a hot environment, secrete large volumes of sweat containing nearly as high a concentration of salt as the body fluid from which it is produced. Sweat formation then involves little modification of the osmotic pressure of the extracellular phase and, therefore, does not greatly reduce the water content of the cells and does not cause a degree of thirst proportional to the water deficit. An acclimatized man whose sweat is very dilute in salt (10-20 mEq/liter) is much thirstier for a given water deficit and will come nearer than the unacclimatized man to maintaining his water balance by voluntary drinking. In the acclimatized man, thirst is more intense because the loss of water raises the osmotic pressure of the extracellular fluid and causes a withdrawal of fluid from the cells.

Dill,[8] Pitts *et al.*,[12] and Adolph[1] found that even acclimatized men working in the heat never voluntarily drink as much water as they sweat, even though it would be advantageous for temperature regulation, but usually drink at a rate of about one-half to two-thirds the water lost in sweat. Serious dehydration, with consequent circulatory strain and hyperpyrexia, may result from such voluntary abstinence from water by working men, even when plenty of water is available to them. Adolph[1] has termed it "voluntary dehydration" and found that it varies

directly with the rate of sweating and the rate of work. It seems probable that voluntary dehydration may be even more pronounced and debilitating in football players during a hard practice session of 2 hr or more on a hot day than it would be in men marching at a moderate rate in the desert or the tropics. Prevention of voluntary dehydration during work in the heat requires forced drinking of water, preferably containing salt in the same concentration as the sweat. The sodium concentration in human sweat varies widely, ranging from 5 to 75 mEq/liter and averaging about 34 mEq/liter, which is equivalent to about 0.2% NaCl solution in water. This concentration in the drinking water during 2-3 hr of hard work in the heat would generally protect men against serious dehydration and salt depletion, provided they drank enough to replace about 80% of the water being lost through the sweat glands.

MAXIMAL WORK CAPACITY

A number of investigators have found that dehydration reduces the capacity of men to perform hard work, even in cool environments.[6,15-17] The effect is most pronounced if dehydrated men attempt maximal competitive work in hot weather. Buskirk *et al.*[6] found a consistent reduction, averaging 0.22 liter/min, in the maximal oxygen consumptions ($\dot{V}O_2$ max) of men dehydrated by an average of 5.7% of body weight. However, Saltin[16,17] found that dehydration up to 5.2% caused no reduction in $\dot{V}O_2$ max or in maximal cardiac output but that substantial reductions occurred in work tolerance and in the ability to elevate blood lactate in exhausting work on a bicycle ergometer.

We[15] studied the effects of dehydration (4.2%-4.7% of body weight) on both the aerobic and anaerobic capacities and endurance of men in exhausting treadmill runs (3-6 min) in a cool environment. The men were dehydrated by moderate treadmill work (MR 190 Cal/m^2-hr) in the heat (DB, 50 C; WB, 27 C), and then rested in a cool room (24 C) for 3 hr without ingesting any fluid before performing the exhausting runs. Control experiments were carried out for comparison. The most significant effect of dehydration on performance in the exhausting runs was a reduction from 4.6 to 4.0 min in the average time the men could continue the runs before they reached exhaustion. This decrease in endurance depended, at least in part, on decreased efficiency, as indicated by a small (5%) increase in oxygen requirement per kilogram per minute of running time. Inasmuch as the average $\dot{V}O_2$ max and the amount of oxygen debt accumulated in the runs were the same, the increased oxygen requirements of the men in dehydration experiments depended on increments in the rates of accumulating the oxygen debt. On the average, the men accumulated about the same oxygen debts in both runs; but, because the average time of running was lower in the dehydration experiments, the buildup of the oxygen debt was faster in those experiments than in the controls.

Dehydration did not decrease the acceleration of oxygen consumption during the first 2 or 3 min of the runs. The retention of the maximal capacity for increasing oxygen consumption in exhausting work indicates that in dehydration the men were able to make adequate compensatory vasoconstrictor responses in the skin and viscera and thus shunt most of the blood flow to the working muscles. Another factor that probably helped in this response was the hemoconcentration that occurred in dehydration; the men's hematocrits at the start of the runs averaged 46.6% in the controls and 49.0% in the dehydration experiments. This increase in oxygen capacity of the blood could compensate for a moderate reduction in maximal cardiac output; such a reduction might be expected to occur with the reduction of blood volume associated with dehydration.

PRACTICAL SUGGESTIONS

Dehydration by more than 2% of body weight should be avoided by athletes during prolonged strenuous contests or practice sessions in hot weather. That is made clear by the physiologic evidence presented here and by the frequent occurrence of heat illnesses, including occasional heat stroke and fatalities, in athletes performing in summer heat. The reduction in work capacity of dehydrated men, even in cool environments, indicates that severe dehydration to reduce weight for athletic contests should be avoided. It is also important not to overhydrate and not to take more salt than that required to replace current losses through the sweat glands during contests.

Avoidance of dehydration is not the only appropriate precaution for preventing heat illness in men under these conditions. Even the most superbly trained athletes can overtax their capacities for heat dissipation if they work hard and long enough in a situation in which heat dissipation is resisted by a hot environment and clothing (such as a football uniform). In fact, athletes may be in more danger than less fit men who are less competitive and more likely to develop symptoms of syncope and to stop work before serious heat pyrexia develops. It is particularly important to remember that football clothing and protective gear add greatly to the stress of work in a hot environment; if they must be worn in hot weather, the sessions must be greatly shortened.

Prevention of dehydration of players during long football practice sessions and games requires planning and cooperation by the team physician, the coaches, the trainers, and the athletes. In the severe stress of football contests, "voluntary dehydration" may be more of a problem with players than it is with men performing more moderate, routine work. Therefore, a deliberate planned program of drinking water is required.

Water should be replaced with 0.2% NaCl solution, because that is the concentration of salt in the average man's sweat. Addition of some

sugar and lemon juice or other flavoring to improve taste may be desirable. At least partial replacement of salt is important to protect against heat cramps. It is better to take the salt in solution than in solid form, to ensure that adequate water is taken with the salt. About three brief relief periods from work each hour should be provided for drinking. The men should be convinced that water and salt replacement is important and that their thirst during contests will not be an adequate guide to their water need. Each man should learn how much water he can drink in the relief periods without gastric or other disturbing symptoms. The water consumed by each should be measured and recorded. A person consumes very little water by briefly spraying his mouth during relief periods.

Nude body weights should be accurately recorded before and after work. Each man should learn in repeated practice sessions to drink enough to prevent losing more than about 1%-2% of his body weight. He should never drink enough during a session to gain weight.

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CLINICAL ASPECTS OF DEHYDRATION

ROBERT J. MURPHY

In a story in the 7 October 1968 issue of *Newsweek*, the following passages appeared:

> Big Ray Schoenke, guard for the Washington Redskins, appeared at training camp this summer carting ten cases of giant-size cans filled with a murky fluid called Gatorade. Schoenke, who drinks at least 3 quarts a practice session, knew that his team was still using water buckets, soaked towels and soft drinks to slake its heavy thirst. "But I converted them fast," he says. "Now they're all on Gatorade."
>
> So are all but two of the other professional football teams, 300 college teams and 1,000 high-school squads. When Notre Dame lost to Gatorade-drinking Purdue last fall, the Fighting Irish put in an order for the stuff the following Monday. It is used by professional basketball teams, major-league baseball teams, tennis players, big-game hunters, rodeo cowboys and even trapshooters. Actors in the energetic Broadway production "Hair" say it beats LSD. It is, in fact, one of the hottest new products to hit the U. S. in years, with a potential market that may go well beyond sports.
>
> . . . The new drink is absorbed into the system in three minutes, twelve times faster than plain water, quickly restoring an overheated player's physical and mental agility.

Also appearing in November 1968 in a full-page ad for Gatorade was a statement that it is 12 times better than water in quenching thirst.

In 1968, I think Purdue University was still using Gatorade. Ohio State beat them and became national champions. Ohio State uses plain water.

As the previous two papers have stated, sweat is a hypotonic solution. Actually, more water is lost than electrolytes, and theoretically it is logical to replace sweat with an equivalent fluid. As doctors, we are geared to the replacement of fluids over a 24-hr period. When a child is sick and losing fluids, we are concerned with adequately replacing the electrolytes, as well as the water. But in football, the longest period involved in practice is about 2 hr, usually 1½ hr. Our bodies have an amazing adaptive mechanism, and it is generally unnecessary to go into all the problems that we would face in handling an athlete who had been exercising for 6-8 hr.

Dr. Cade's original research, which produced the statement that Gatorade is absorbed from the gastrointestinal tract 12 times faster, was not done on a dehydrated subject. In my opinion, it is not valid, because the experimental work must be done on a dehydrated subject to make a proper evaluation. In the dehydrated animal--in this case, man--the primary intravascular deficiency is of water. In a 1½- to 2-hr period, the stores of sodium, potassium, and the other electrolytes are adequate if there is enough fluid to mobilize them.

Dr. Don Matthews (a physiologist), Duane Eddy, Dr. Ed Hyatt of the Department of Physiology at Ohio State University, and two graduate students undertook this study. The purpose was to determine whether water, Gatorade, Coca-Cola, or Kramer's Take-Five was best as a replacement fluid. Kramer's Take-Five was quickly abandoned, because it was hypertonic when mixed to the recommended proportions. That left water, Gatorade, and Coca-Cola.

The effects of rehydration on performance were measured. Six college athletes were dehydrated by 3% of their body weight in a sweatbox. Before and immediately after dehydration, urine and blood samples were taken. Within 30 min after dehydration, the subjects consumed a volume of replacement liquid equal to the amount lost during dehydration. Then blood and urine samples were taken every ½ hr for 1½ hr. The subjects then performed a treadmill test, and the data were handled by one-way, two-way, and interaction analysis of the variants.

Water and Gatorade were associated with significantly lower plasma sodium concentrations than Coca-Cola. Potassium concentration diminished throughout the experiment. Gatorade was absorbed at least as rapidly as water, and possibly a little faster, but not significantly. Performance times to exhaustion associated with the three drinks were not significantly different.

This study was done at the request of Stokeley-Van Camp, which promotes Gatorade, but I think an attempt was made by all to be as objective as possible. The following conclusions can be drawn about

Gatorade. This study revealed no contraindication to its use. The glucose it contained provided ready energy, as indicated by an increase in respiratory change ratio. It was better than water from the standpoint of replenishing water lost in sweat. It is physiologically sound. The subjects liked it better than water. It was easy to drink. But there was no difference between water and Gatorade in performance, and there was no significant difference between water and Gatorade in absorption.

The three clinical syndromes that we face on the football field are: heat cramps, the painful contractions of skeletal muscles when a person sweats a lot; heat exhaustion, the state of physiologic breakdown of the body without increased temperature; and heat stroke, in which an absence of sweating (for reasons not yet understood) is accompanied by dry, hot skin and a rapid increase in temperature. When the temperature rises above 105 F (40.6 C), serious changes usually occur. The first organ affected is the brain; unconsciousness occurs. The second is the liver; if the victim recovers, he will usually have jaundice.

My first experience with heat stroke involved a young man who was unconscious when admitted to the hospital. His temperature went to about 105 F (40.6 C). With icing and prompt administration of fluids, he awoke in about 3 hr, and he was apparently well the next day. Three days later, he came down with a typical case of jaundice. That was about 14 years ago. At the time, we thought he might be coming down with infectious hepatitis. However, about 2 years later, a monograph described some 14 cases of jaundice following heat stroke.

It is often asked whether a boy who suffers heat stroke is more prone to heat stroke later and whether he should participate in athletics again. The boy I described came back the next year and was an All-America guard at Ohio State. Once it became routine for him to replenish his fluids throughout practice and game situations, he never had any further problems.

For a long time, it was fashionable not to drink water on an athletic field. It was primarily a discipline problem; coaches apparently felt that a boy was a weakling if he had to drink water on the field. I think that idea has been overcome in American football. It still is prevalent in many places, but I know that Allen Ryan has been working in this area for years, and I think the word has gotten around that salt and water replacement are important.

There are some misunderstandings, however. A high-school boy in Ohio died 4 years ago. His coach had him wear a rubber sweatsuit in August to lose weight. The boy came in unconscious and later died. When we asked the coach whether he had given the boy water, he said he had not, but he had given the boy four salt tablets before he went out. Obviously, they had contributed to the death.

Bill Ashe, who was the head of the Department of Preventive Medicine at Ohio State, and with whom I worked originally in this field, was in the Army during World War II and told of an experiment. A general that he worked with was of the old school that believed that a quart of water a day was adequate for horse and man. Dr. Ashe said to the general: "You give me a platoon that you consider your worst in terms of marksmanship, and you take the best platoon; let us have them work in the morning before they go out for target practice. Then we will go on a forced march in the desert. You give each of your men a quart of water, and let me give each of mine replacement as I desire. Then let us test them when they come back." The best platoon dropped from about 92% in the morning to about 64% in the afternoon. Dr. Ashe's group dropped from about 62% to about 58%--not a very substantial change. This shows that dehydration has a definite effect on performance. Euphoria seems to accompany dehydration, however. The dehydrated men in Dr. Ashe's experiment were a little giddy and jolly, and they really felt that they were much better able to perform than they proved to be in the actual testing situation.

Another experiment was done, with wrestlers. The treadmill test was used. A group of wrestlers and a control group could walk for 21 min on a gradually increasing level. When they were dehydrated by 3% of body weight, the time dropped from 21 min to 14 min. The same group was rehydrated, and tested again; they could walk for 18 min. Rehydration did not let them return to a normal time, but it was much improved over the dehydrated state. We are all concerned about wrestlers, particularly in high school, and even junior high school, who dehydrate themselves to make a specified weight and then go out to perform.

Some work done by the Department of Physiology at Ohio State showed the difference in water loss when a football uniform and a scrub suit were worn, with a markedly increased loss when a football uniform was worn. As opposed to a loose-fitting scrub suit, a constrictive football uniform does not allow evaporation from the skin. We use the long-sleeved jerseys and stockings only about once a year, usually in late November when it is very cold. I do not know how much control team physicians have over uniforms; but I am often concerned, as I watch the professional games in August, to see the players come out in full stockings and long-sleeved jerseys, both of which decrease the amount of skin available for evaporation.

We think it is important that players be given water. We keep six cans of water on the field, and the boys are given the privilege of drinking *ad lib*. We check their weights very carefully; if a boy is losing a great deal, then we see to it that he drinks more.

Salt tablets have the disadvantage of not being absorbed in some persons. We have tried to use various solutions on the practice field, but some of the boys will not drink them. We find that, if we use ice water *ad lib* throughout the period, they can maintain their balance between practice sessions with salt in their food and salt tablets.

We depend heavily on wet-bulb temperature as a guide. If the wet-bulb temperature is under 60 F (15.6 C), the boys can practice for 8 hr and never get into trouble with heat. If the wet-bulb temperature is 60-65 F (15.6-18.3 C), we watch the players very closely, but do not push them very hard on the field. If it is 66-70 F (18.9-21.1 C), we insist that salt and water be given on the field (Gatorade, as far as I am concerned, would be fine in this case). If it is 71-75 F (21.7-23.9 C), we attempt to provide a rest period every 30 min. And if it is 76 F (24.4 C) or higher, we suggest that practice be postponed or conducted in shorts. These criteria must, of course, be varied on the basis of climate. For example, in Texas raising each one by 4 F (2.2 C) would probably be adequate. But the real purpose of the numbers is simply to alert the coaching staff and the training staff when conditions favor heat problems. If they see a boy who is weak and does not feel good, they should not simply tell him to lie down, but should take hold of the situation and be sure he gets the proper medical help.

I would like to emphasize that heat stroke is a true medical emergency. Probably no emergency in medicine requires as prompt action as heat stroke, and the basic treatment of heat stroke is to lower the body temperature. If you can keep the temperature below 105 F (40.6 C) until you can get the patient to a hospital, you will rarely lose a boy. It is vital to have ice available on the field. An iced blanket or sheet can be put over a victim, or he can be immersed in a bath of ice. Or four boys with ice in their hands can rub the victim's arms and legs; that will do a great deal toward lowering the body temperature.

EFFECT OF FATIGUE AND MENTAL STRESS ON FOOTBALL PERFORMANCE

CHESTER M. PIERCE

The cost of injuries in football is enormous. Some would put the cost in dollars; for example, the *New York Times* stated that the National Football League estimated that it would lose nearly $2 million last season, merely paying men whose injuries kept them from performing.[2] Others recall that the cost could be calculated in terms of positions--thus, the fantastic loss for football lovers when 16 quarterbacks are seriously injured in one season. In doctors' dining rooms across the nation this fall, the cost was reckoned in terms of anatomy--how many players would require knee surgery as a result of injuries incurred on the football field.

However, to the psychiatrist the astonishing statistic is not in terms of injury. What is more staggering is that more men are not injured. The name of the game, in psychologic terms, is killing your opponent without insulting your own pride. When expert football players, with maximal experience as performers and magnificent all-around physical equipment, use their keen minds to dedicate themselves intensively to contest, the wonder is that there is not serious injury on each play. As though these attributes themselves were not sufficient to wreck many bodies, one must contemplate that all the attributes are aggravated by a relentless motivation to succeed in the game--to render your opponent useless--admixing such basic desires as demonstrating one's masculinity in public and earning an excellent livelihood.

Keeping these psychodynamic ideas in mind, I will comment on some psychiatric aspects of injuries. The comments will be based on laboratory experience, clinical observation, and theoretical speculation. Finally, I will suggest a few research pathways that might diminish football injuries and, incidentally, might have spinoff value for other areas of research, such as preventing automobile accidents and understanding the process of aging.

Fatique is defined as a "state of increased discomfort and decreased efficiency resulting from prolonged or excessive exertion."[1] All physicians are aware that fatigue may be defined functionally in terms of a person's psychologic interest. Thus, the bored teen-ager complains

of being too tired and too fatigued to do her homework but is indefatigable the same evening when dancing the Watusi. Emotional stress, even of a chronic or subacute variety, can result in the increased discomfort and decreased efficiency that characterize fatigue.

THE PROBLEM OF MAKING THE TEAM: A PHYSIOLOGIC BASIS FOR FATIGUE RESULTING IN INJURY

Laboratory studies are now defining what constitutes good and restful sleep. The good sleeper, who has a smaller risk of being fatigued during consciousness, spends more of his sleep hours in the deep stage of sleep. His EEG indicates that he uses up less of this deep sleep, which is represented by delta or slow-wave activity, during the first half of the night. His slow-wave activity is spread more smoothly throughout the night than that of the poor sleeper. As one grows older, he has less delta sleep. Thus, the 5-year-old spends 50% of his sleep time in delta, or deep, sleep. The 85-year-old has no delta sleep. If this slow-wave sleep activity is aborted, in laboratory tests, the subject becomes noticeably depressed and apathetic when awake. Subjectively, one is fatigued.

The results of fatigue in psychophysiologic terms include decreased motor speed, lessened critical judgment, and lowered susceptibility to pain.

Let us hypothesize that a professional football player, whether veteran or rookie, begins to worry about making the cut. His anguish interferes with his sleep. More specifically, he alters his amounts of deep sleep and dreaming sleep. Soon, both the quantity and the quality of his sleep are very different, and in fact his biologic rhythm is put out of kilter. These physiologic alterations manifest themselves in ways that jeopardize football success. The rookie quarterback cannot read blitz patterns, not because of lack of comprehension, but because his thinking speed is reduced as a result of loss of slow-wave sleep. The veteran guard misses a blocking assignment because of an error of omission in his thinking process. The cornerback's misjudgments, only a critical second less than usual, might be due to a lack of sustained attention, which is secondary to a chronic, subclinical sleep disturbance. After a "physiologically wretched" night in bed, occasioned by worries over mistakes in practice, a giant defensive tackle becomes much more susceptible to pain. Despite even valid protest that subjectively he had had a good night's sleep, the altered sleep cycle could be the reason for this man's crumbling under a relatively innocuous blow.

In a game in which speed and reflex action are so critical, the fatigued player will suffer erratic performance. Loss of slow-wave sleep, associated with and perhaps the cause of fatigue, results in slower reactions. This slowing, no matter how slight, makes the once-invincible athlete vulnerable, particularly when he is playing against adversaries all of whom are, by definition, expert football craftsmen. It is not inconceivable that someday coaches will take brain-wave scans to help calculate potential injury.

A player may respond to his fatigue by two other reactions that can claim our attention. On the one hand, he may become more irritable and labile in his interpersonal relationships, such as that with his wife. On the other hand, he might become anxious over his escalating emotionality, pain, and sleep loss, and prescribe medicines for himself. Let us consider first the player's relationship to his wife as a possible cause of football injury.

THE PROBLEM OF PLAYING ONE'S BEST: A PSYCHOLOGIC BASIS FOR FATIGUE RESULTING IN INJURY

This is a genuine case history: John, a draft-choice running back, desperately loved Mary. Just after his senior year's football season, his jealousy, deliberately provoked by Mary, resulted in Mary's being beaten by John at a public festivity. On the basis of these skimpy details, most psychiatrists would wonder immediately about Mary's attitude toward her father and John's attitude toward his mother. Because the couple had distorted attitudes, rooted in their own childhood perceptions of marriage, a therapist would be concerned about a situation that could result in extreme aggression--i.e., murder and/or suicide.

Despite medical advice to the contrary, Mary decided to marry John. John went on to win a starting assignment on his NFL team. Predictably, Mary, although living a chaste and faithful life, made John so concerned about their relationship that friends speculated that "that girl has his head all messed up; he won't be able to play any football." This diagnosis was sustained by the same doctor who advised Mary not to marry John. In fact, the doctor, who saw Mary only on her infrequent visits back home, went on to predict to his colleagues that John's castrative anxiety would probably result in serious injury to himself. The doctors speculated, also, that Mary might become a victim of John's extreme aggression.

Typically, John would boast of his new car. Mary would take this as her cue to confess about the "cute" NFL owner who tried to "hit on" her and the wife of another player as they sat innocently flirtatious in a night club. John would counter, in a *non sequitur*, that he didn't even have to train to play professional football. Such masculine bravado and unnecessary pride were interpreted by the psychiatrist as further evidence of John's pursuit of self-destruction. The doctor continued to worry about Mary's safety.

In interpersonal reactions, John, amazingly enough, invited behavior from his wife that any observer could see obliged him to be demeaned, belittled, compared unfavorably with other men, and, in short, infantilized. That is, John demanded that his wife keep him a little boy. His wife accommodated him. Every psychiatrist will know that in all likelihood this was the same pattern of stimulus and response that John

had known with his own mother. No doctor will be surprised to hear that at intervals John was physically abrasive to his petite wife, who behaved in such ways as to make him feel inadequate.

Mary hinted that she might be with a handsome, rich, smart professional man while John was playing in a large metropolitan area. In truth, she was going to be with an aunt. John was much aggrieved and shot back at her that, just a couple of weeks previously, he had asked her, "How can I have 300 pounds of Roger Brown all over my back and you too!" Indeed, on that last Sunday of his football career, it was not only the enormous load of defensive linemen on his back that led to ruination, but also the heavier and unremitting load of jealousy, anger, despair, and doubt. In psychologic terms, his injury represented a self-punishment and an attempt to manipulate his wife and garner her sympathy. The injury spelled the end to both John's football career and his marriage. Long after career and marriage had terminated, Mary lived in fear that John might kill her.

Without the stormy marriage, John might have proved to be a better investment for his team. The incessant marriage problems constituted a mental stress that fatigued John and made him more prone to injury. At an unconscious level, the marriage problems may in fact have precipitated the injury and even been critical in selecting the part of the body that was injured. The scouts who recruited John for the draft, had they known of the public fracas and his jealousy, might have taken steps to ensure a happier marriage for John and a better investment for their club. In advance, medical consultants could have foretold the mutually self-destructive nature of the marriage. If one wishes to be self-destroyed, is there a better place to be than on a professional football team?

THE PROBLEM OF KEEPING ONE'S POSITION: A PSYCHOSOMATIC BASIS FOR FATIGUE RESULTING IN INJURY

A psychosomatic illness is one in which organic changes are caused at least partially by chronic emotional conflicts, usually mediated through the involuntary nervous system. Theoretically, the remorseless pressures to keep one's position on a professional football team could precipitate all manner of psychosomatic illnesses.

In terms of injury, the chronic stress of playing could act separately, but it would more likely be a part of a constellation in which fatigue was brought about by sleep disturbance, interpersonal stress, and other intrapsychic or personal stresses. The result would be the same impairment of speed and judgment and the same heightened sensitivity to pain as described in regard to sleep disturbances or mental conflict.

A chorus of denial will greet this hypothetical speculation, but I must repeat an axiom taught to me by a Nobel Prize winner: Until it is proved otherwise, consider every patient as being an alcoholic, a carrier of venereal disease, and a drug addict.

Some players are battered so badly that the football season can be conceptualized as one large bruise. Such men continue to play, stoically disregarding their wounds. Their motivation includes a comfortable salary, but also fierce pride and a corporate image. Such men would be driven to play so that a hungry competitor could not get the opportunity to display his own talent.

Under such circumstances, there must be among our Sunday heroes some who treat themselves with an assortment of drugs, including pain killers, hypnotics, and stimulants. Naturally, the medical and staff administrators would not be informed of this sort of drug abuse. The formula would have countless variations. For example, a young, strong, fast veteran competitor appears at summer practice. In the ensuing struggle, the player becomes more and more tense about the certainty of his position. He projects blame onto inferior equipment, poor ground conditions, etc. He begins to be obsessive about contractual arrangements. Yet he is pressed continually by the newcomer. He takes some over-the-counter nonbarbiturate drugs to sleep. Then he finds that he must get some stimulants to give him a hoped-for sharpness and acuity. If pain appears, he might seek out medicines to apply over the damaged area. When such a cycle is established, the chronic emotional conflicts, even without mediation through the involuntary nervous system, place the player in increased danger. He might be injured directly, owing to impaired judgment and slowed reflexes. Or he might injure himself by playing with part of his body so damaged that he should have been medically disqualified to prevent aggravation of the damage. Doctors and trainers need more help in identifying and controlling cases of this type.

SOME POSSIBLE PATHWAYS FOR RESEARCH

Generally, there are three areas of psychiatric research that might pay dividends to professional football: (1) sleep and dream studies, (2) drug studies, and (3) studies of individual and group psychodynamics. Many specifics could be mentioned. Naturally, many details and much thought would have to go into planning any research program. For instance, whenever a person were injured, an interview could be conducted to elicit the immediately preceding events in the person's psychosociocultural life. These data could be computed with other information gleaned from that player and others. Soon, thematic patterns might emerge that could have much preventive value. Studies of biologic rhythm, including sleep-activity cycles and temperature peaks, should be studied and correlated with injuries, including their time of onset, where they occurred on the field, and dozens of other features. Another fruitful area would be the player-to-player group relationships and the player-coach relationships.

Emphasis should always be on singling out the conditions associated with either never being seriously harmed or being injured frequently or severely. Telemetered information about brain-wave cycles over 24-hr

periods might be especially valuable. Once men were used to having behavioral scientists talk to them, specific studies could be designed to learn more of the motivation to play football and such aspects as the relationship of body-image pride and injury. Football would be one of the major beneficiaries if ideal drugs could be found that killed pain, promoted sleep without altering brain-wave patterns, or stimulated psychomotor activity without causing spurious judgment. Brief psychotherapy for players' wives would probably aid considerably in reducing injuries. Developing appropriate therapy for selected players and family members would constitute another avenue of inquiry.

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PSYCHOLOGIC SCREENING

THOMAS H. HOLMES III

I would like to discuss the development of a measuring device that seems to have an amazing ability to predict major health changes--including accident, injury, infectious disease, psychosomatic disease, and fractures--and to show how it works when applied to predicting injuries in a football team.

Essentially, my thesis is that injury and disease are byproducts of man's goals and the techniques that he uses to achieve them. There seems to be a striking relationship between life style, life events, and disability.

Research into such a thesis begins at the bedside, where the data from the medical history are organized as a medical biography, beginning with birth and including childhood, growth, development, aging, maturation, etc. It is simple to tabulate the age of occurrence of illness. The rest of the medical history--having to do with economic status, education, marriage, occupational history, and so on--should also be included.

When one does this, one is impressed with the striking relationship between the appearance of illness or accident and various events--such things as marriage to an unsympathetic woman, a job change, a jail term, and financial problems.

We have found such relationships in the case of tuberculosis, cardiac disease, skin disease, hernia (not why it happens, but why it happens when it does), and many diseases--even pregnancy.

Rather than do this in a purely qualitative way (just counting up the number of life events), we thought we needed a quantitative way of relating life events to the onset of disease.

Over the years, on the basis of some 10,000 patients, we have evolved a series of 43 life events that empirically show a striking relationship between their occurrence in clusters and the onset of some kind of major health change.

What we were interested in was being able to say that, for example, the death of a spouse probably is more salient to a person than getting a traffic ticket or experiencing the Christmas holidays. We were able to demonstrate that such comparisons have validity.

Our method was derived from psychophysics, the branch of psychology that addresses itself to man's amazing subjective ability to quantify objective physical phenomena, such as the intensity of light and the loudness of sounds. Mathematically, this may be expressed as an equation in which the response to the intensity of sound is a power function of the stimulus.

One of the methods used in quantifying sound is the method that we used to quantify our 43 life events that have been empirically demonstrated to cluster at the time of illness onset. We took one of these events. Arbitrarily, we chose marriage. Marriage is a social event requiring change and ongoing adjustment. It was arbitrarily given a value of 500: the amount of social change required by marriage is 500 units. Then we asked our subjects (some 400): "Does trouble with the boss require more change and adjustment than marriage, or less change and adjustment than marriage? If more, put down the proportionately larger figure. If less, put down the proportionately smaller figure." And so on with each of the other 41 items, each being compared with marriage.

We recorded the mean value of each of the 42 items, as judged by some 400 persons in Seattle. The item requiring most change was the death of a spouse (1000); those requiring the least were a vacation and a traffic ticket (about 110). Actually, we divided the figures by 10, to use a scale of 100, rather than a scale of 1000. We then knew that, for example, the death of a spouse requires twice as much change or adaptation as marriage, and about 10 times as much as a traffic ticket. It takes about as much change to divorce a spouse as it does to get married in the first place.

We broke down our sample for purposes of validation, comparing the ranking of all these items by males and females and by single and married persons. The coefficients of correlation are all above 0.90, no matter along what lines or with what variables these samples are compared with each other. We have done the same kind of work to validate the method in Japan on Japanese, in Watts on Negroes and Mexican Americans, and last summer in France on Frenchmen, and in Belgium on Belgians, and in Switzerland on French Swiss. All agree with each other and with Americans, at about the same level, about 0.90. The world over, the death of a spouse requires more change and adjustment than any other of these items.

We feel now that we have a scale that is quantitative, and that we can say how many units of life change are involved in any particular life style, instead of having to say "death of spouse," "Christmas," "fired," "trouble with the boss," or "in-law trouble."

We then applied this to a retrospective study. We had a group of about 80 resident physicians fill out the questionnaire, listing how many of these 43 events occurred in each of the last 10 years and, on a separate sheet, what health changes they had had in the last year. When we plotted the life-change units by year over a 10-year period, the illness episodes could be seen to correspond strikingly with peaks or major crises in life-change units--a depressive episode at 300 life-change units, prostatitis with gross hematuria at about 275 life-change units, and so on.

Not all the crises, not all the accumulations of life events, are associated with illnesses. We divided our life-change units and subjects into low-risk, medium-risk, and high-risk groups--that is, many, not so many, and relatively few life changes. At a low level of life change, 37% of the crises or peaks in life-events accumulations are associated with major health changes. In the medium range, from 150 to 300 life-change units, about 50% of the crises are associated with illness. Where the score adds up to over 300 life-change units, about 79% of the crises or peaks are associated with major health change.

When one accumulates the data from the questionnaire and assembles the life changes in each person's past, an assessment is made; 8 months later, it is repeated. The greater the life change, the higher the probability of illness or accident in the next 8 months.

If we divide people into high-risk, medium-risk, and low-risk groups on the basis of life change, there is good correlation with illness or accidents. An 80% correlation would be required for prediction.

A 2-year follow-up revealed essentially the same figures as the original assessment, in that 30% of the people in the low-risk group (that is, 0-149 life-change units) became ill, 50% of the people in the medium-risk group (150-299) had illnesses, and about 80% of the people with scores over 300 became ill.

One of the young psychiatrists who worked with me in the development of this material did an epidemiologic study in the Navy. Personnel of three cruisers completed the questionnaires and were divided into three groups: high-, medium-, and low-risk. The high-risk group always had a significantly higher illness rate than the low-risk group.

We then attempted to test the hypotheses by studying a relatively homogeneous population in which age and sex were uniform and risk of injury and motivation were relatively constant. A local northwest football team seemed to satisfy these criteria. A significant injury was deemed one that prevented a player from suiting up for 3 or more days of practice, whether that included a regular game or not. We found that, of a squad of 100, 26 players sustained 36 injuries and three additional players had three illnesses that caused them to miss

3 or more practice days--two cases of infectious mononucleosis and one of influenza. In our other populations samples, there was a much higher risk of illness than of injury. On the football squad, 50% of the high-risk players had injuries or were ill during the season, 25% of the medium-risk players, and only 9% of the low-risk players.

We were interested in seeing whether there was any relationship between the seriousness of prior illness or injury and that of later illness or injury. There does seem to be a correlation.

I would like to summarize by saying that, by using an empirically derived set of test items, an assessment of life stresses can be made. This is true, whether one is studying general populations or collegiate athletes. Of those who are classed as high-risk, the probability is that eight of 10 will have some significant health change within the ensuing 2 years.

COACHING, GAME SKILLS, AND INJURY

DONALD SHULA and EDDIE BLOCK

Every football coach is acutely aware of the problems of teaching game skills that will protect individual players and prevent injuries. An injury to a football player is a nightmare to a coach as much as it is to the player. Every coach wants his men to go through the football season uninjured. That is the basic formula of success for a winning football team. Everything else being equal, any team that has fewer injuries is usually going to finish higher in the standings.

Football is a contact sport, and the problems associated with collisions in football are compounded by the fact that the opponent is also aware that football is a contact sport. The result is that each side must resort to strategy. Strategy makes it more difficult to teach injury-preventing football skills, because for each skill there is a counter-skill or offsetting skill. Generally, it takes years of experience to learn enough of the game skills and the counterskills to become a polished and successful player.

More complexity results from the fact that there are 44 basic components to consider in teaching game skills--i.e., you have 11 offensive players and 11 defensive players, and your opponent has 11 offensive and 11 defensive players. And there are still other factors involved.

Game skills vary not only with the 44 component positions, but also with each individual player. Each player has his own capacities in regard to strength, speed, agility, weight, size, etc.; and the skills of football must be adapted to the different capacities of each player.

Another formidable factor is that game skills vary with each play and with each of the almost unending series of plays. We have basic plays and we have variations of them; all involve the skills that must be taught to the players.

Football is a team sport. That means that the skills of each player must be structured to dovetail with the skills of his fellow players if the proper timing, which is so important in football, is to be maintained. Faulty timing of a play can result in physical injury, not to mention psychologic trauma. The psychologic status of a player

under game conditions is almost impossible to assess. But psychologic factors are certainly an important part of injury prevention in relation to football skills.

We also have to consider fatigue, cardiovascular status, pain tolerance, biomechanics, previous injuries, and biochemical factors in their ever-changing nature.

If ever these few foregoing factors were reduced to a mathematical model, such a model would indeed be imposing. Yet with all these complexities, much has been left unsaid of such factors as rules of the game and interpretation of the rules in competition.

In the face of such complexity, what can a football coach do, by way of teaching skills, to prevent injury without compromising the chance to win games?

The first step that we take in a practical approach is to realize that no one person can do the entire job; so we hire assistants who are specialists. The assistants include football assistants who are specialists in teaching skills and medical assistants. We also need help from scouts and from the business personnel.

The next step is to organize an over-all plan, with each of the specialists contributing to the plan. The plan includes an order of skills to be taught. Organizing and reorganizing the over-all plan take place either during the season or, preferably, during the offseason. Professional football involves a constantly changing strategy. We work 11 months a year; we take June off. Most of our organizational work is done in the offseason. We re-evaluate our performance on the field and try to find ways to improve, as far as the medical handling of the players is concerned. All that is done in the offseason; once the season starts, we are too concerned with the aspect of winning to have much time for anything else.

Where new theories of teaching skills are concerned, we have to be wary. New theories of teaching skills are generally developed in a laboratory and often break down because they are based on only parts of game skills, whereas, as we have said, football is very complex. It is almost impossible to develop a workable theory apart from actual game conditions. There are just too many unknown factors in actual competitive football. Most coaches consider that new theories are for the future. Coaches must think absolutely in terms of the present, the practical, the workable. The problem is to develop winning football in the present. That is often overlooked by research workers, with resulting distrust between the practical and the research man. The solution is to stick to the traditional methods. Of course, we are always alert for ways to improve methods that are successful. "Traditional methods" are those which a coach and his assistants have found to be workable in their own experience. A coach is not likely to change his methods of teaching skills unless his methods have proved unsuccessful, especially if a new method is based on a theory derived from a single case.

Our traditional methods, as well as those of most teams, begin ideally in the offseason. We base our skills, first of all, on preventing deconditioning of our players in the offseason, and then developing physical strength with particular emphasis on the legs and upper body. For developing strength in the offseason, we put a lot of emphasis on heavy-weight programs. We also encourage games in the offseason. The game that we prefer is handball. We think it is an excellent offseason conditioner for almost any position in football. Basketball is also good, because it requires great endurance and use of the hands. But handball involves concentration, the use of both hands and feet, watching the ball come off the wall, and getting into new positions quickly.

Our next consideration is the development of cardiovascular efficiency through running. Last year, we started the 12-min walk-run, as developed by Col. Cooper. We started applying his method in our offseason conditioning program. For the sake of experiment, we had some of our rookies see how far they could go, either walking or running, in 12 min, to get some sort of standard that we could measure other players against next year. The results were amazing. Many supposedly conditioned athletes cannot go very far in 12 min. Some big linemen, for example, cannot run for any substantial distance. For them to cover a mile in 12 min would be quite an accomplishment. Some of the defensive backs and receivers might go 1.5 or 1.75 miles. Anything over 1.5 miles is said to reflect pretty good conditioning; 1.75 miles represents very good shape. One advantage to this kind of offseason conditioning is that it is accessible; all it takes is a measured distance and a watch. One can set up his own program, and take 12 min two or three or four times a week, depending on how much time he has.

Probably the best part of our offseason physical conditioning program consists of individualized programs. These programs are set up through discussions between the coaches and the medical staff. We discuss physical weaknesses and review game movies and medical records of injuries. We keep film clips of all injuries. Then we send out individual conditioning programs to each player, feeling that one player might need more work on the upper body or might have a weakness in the legs, etc.

There are some weaknesses in this offseason program, however. The first big weakness is that there are not any provisions for physical contact in this program; the games talked about do not involve physical contact. The player is not conditioned in the same kind of body loading as he encounters in his game skills when the season comes. Probably most important, it is difficult to have proper supervision, because we cannot supervise the players who are out of town and the ones in town all have different offseason occupations and schedules. So they are left to get it accomplished individually.

Our offseason program begins about 4-6 weeks after the end of the season. It is designed for three sessions a week, with an additional day suggested for the 12-min walk-run. Six weeks before the start of

the season, a letter is sent to all the players, advocating an increase in the strenuousness of their programs. We also send out a program designed to increase endurance, increase body flexibility, and polish general conditioning to a readiness peak for the opening of training camp. During the offseason, contact is maintained with all the players, either by letter or in person.

We expect all our players to report to training camp in full readiness to go all out from the first day. We give each one a weight at which he has to report on the first day of training camp. It is not a weight that he can get down to shape from; it is the weight at which he will play the entire season. If he is over that weight it costs him $10/lb per day.

We also stress speed and agility. Our first practice is in shorts. We test them in the 40-yard dash. We do not run them against each other, because we found that players running against each other in a 40-yard dash get so keyed up that they have a tendency to pull a little bit more that first day. So we have them compete against the clock. We put the players through an agility test. Five hurdles are spaced over a total of about 25 yards. The players run around the hurdles, not over them. This test gives us an indication of agility and quickness to go along with over-all speed. Straightaway speed is fine. It is nice to know that a player can run 40 yards in 4.5 sec. But he might not have the stop-and-go speed that is necessary in a good football player. The ideal is to have good over-all speed plus stop-and-go speed, or agility speed.

Our second practice session is in full pads, and in this session we begin establishing contact, or hitting. This is done by what is probably the simplest drill in football. We call it the Oklahoma drill. It has the basic ingredients of what football is all about: a ball carrier, a blocker, and a defensive man. I give the offensive blocker the signal, and on the cadence he comes off the ball and fires straight into the defensive man with the idea of driving him back. The defensive man meets his blow and tries to ward him off, stand him up, and then be able to tackle the ball carrier, who is coming in between two dummies. This is a controlled drill, but it is a full-speed contact drill. Starting off this way gets the players who have been in the offseason programs ready to become involved in the spirit of football--the contact part.

When training camp starts, we do not allow any time specifically for physical conditioning. We have our regular practices, and my whole thought in our practice sessions is not to spend a lot of time on the practice field. When we are working two days in training camp, our morning practice will be no longer than 1 hr 10 min, and our afternoon practice from 1 hr 10 min to 1 hr 20 min. We expect the players to be in good physical condition (the running aspect of conditioning) when they report to camp; when camp starts, we begin teaching skills.

Each assistant-coach specialist begins his group with the basic techniques, for example, stance, and then starts on the contact that we want in our blocking, and then the followthrough after contact is made. Each skill is usually studied on an individual basis, depending on the skill being studied. It is first described to the player, it is usually in his notebook, and we try to have a movie of the particular skill that we want to teach on the field that day. We show this movie to the players and then we follow it up with actual practice on the field.

The next step is to study skills on a man-to-man basis. At this point, the first conditioning takes place. That is the Oklahoma drill. After the initial practice session, when we have established the man-to-man basis, we get into team skills and try to put things together. For example, we will take our offensive line--center, two guards, and two tackles--and put it against our defensive line--two tackles, two ends, and a middle linebacker--with a quarterback and two backs involved. We have what we call controlled key drills; the defensive players are taught to read and react; they are now faced, not with one man in front of them, but with one man in front and men on either side. They have to recognize blocking patterns and be able to meet the various offensive maneuvers that are thrown at them. Offensively, it produces a feel for teamwork. These are full-speed drills, but they are controlled, in that there is not a lot of pursuit and gang tackling, which leads to many injuries.

We also work our offensive backs and receivers in what we call skeleton pass offense and defense against our defensive backs and linebackers in controlled full-speed drills; the receivers or backs run out for passes, and the defensive backs cover. When the ball is in the air, it is up for grabs--it is anyone's ball at that time. Contact is made. Play is at full speed. The defensive man tries to intercept. But, again, there are not a lot of people involved in the impact or contact areas, so it helps cut down on injuries, as opposed to over-all scrimmages. The old idea was to throw the ball out there and start scrimmaging, and then come up with the toughest people you could have to build your team--and also with numerous knee injuries and concussions.

When these skills are put together in total teamwork and tried out in scrimmage, movies of the scrimmages are studied to disclose weaknesses and evaluate players. We try to find out why we are successful or unsuccessful in teaching particular skills. We do very little scrimmaging, because, of course, we are now involved in five or six preseason ball games, in addition to 14 league games. We played some 23 ball games last year, from the time that we went to camp, which was in the middle of July, until we finished up in the Super Bowl in Miami on 12 January. That is a long period, with a lot of physical contact. We try to get all of our teaching done in controlled groups, so that the players are not exposed to serious injury.

When the preseason games begin, we continue to polish the game skills that we have taken on in training camp. When the season starts, the

practice of the skills continues, and they are continually practiced throughout the season. Some of the physical problems that occur year after year with no solution are pulled muscles (generally in the legs, and affecting the muscles on both the front and the back of the thigh) and ankle and knee injuries, both with and without outside forces. We get many impact or crush injuries, particularly on the front of the thigh. Sometimes our players like to go light in practice and will go out without thigh boards; any kind of blow can then produce crush or impact injuries of the thigh.

It seems that, once they are in good physical condition, the only time the players get pulled muscles is when they are playing on a combination baseball-football field. A player might be running from the sod and hit the infield part. Say he is a pass receiver going out and looking up; he comes off the sod, and suddenly his cleats hit the sand or infield part of the field. This is likely to cause a pulled muscle. After practicing all year in Baltimore, we went to Santa Barbara to prepare for our game against the Rams. The turf was totally different from the turf we worked on in Baltimore, and one of our players got a pulled muscle because of the difference, probably, in the type of turf. If we can ever get to a uniform type of surface, it would definitely cut down on these types of muscle pulls, and also on the knee injuries. Those are what really plague us: when a player gets a bad knee, he is probably going to be lost to us for the entire year.

Although it is very hot, especially in our training camp around Baltimore, and humid, we have had only a few mild cases of heat exhaustion. In the last 15 years, no one has been hospitalized on account of the effects of heat. This is partly due to the very short time that we spend on the practice field, rather than being exposed to the hot sun for long periods. We stress conditioning of the body to heat, both in the offseason and during training camp.

THE ROLE OF PROTECTIVE EQUIPMENT IN INJURY CONTROL

ALLAN J. RYAN

Although sport is as old as recorded history, and probably antedates it, with a few exceptions the development of protective equipment for sports is a modern phenomenon. Archery offers one exception. The archer in sport as well as in his military role has worn a band of leather covering the wrist and forearm of his bow hand from time immemorial. It is an obvious, simple, and practical expedient to absorb the snap of the bowstring. There has been essentially no change in the design of this equipment since it was first introduced. We have a greater variety of materials to make it from now, but it is safe to predict that, as long as man shoots a long bow, he will wear this particular protective device.

Another outstanding exception was the knight in armor. Armor was developed for protection in warfare, starting apparently with the shield, then adding the helmet and gradually other pieces. Out of the training that a knight received for warfare there developed a sport, the sport of jousting. This consisted of riding on horseback against a similarly mounted opponent and attempting to unseat him by a thrust with a wooden lance. The concept was simple; it offered a practical means of training for armed combat, which was carried on chiefly by knights on horseback; the encounter was brief (although it was repeated until at least one was unseated); and a clearcut decision was usually obtained. Special heavy armor was gradually developed for this sport. It finally became so heavy that men had to be lifted up onto their horses by a crane. If they were dismounted, they were helpless until someone came to their assistance. In spite of this, serious and sometimes fatal injuries were sustained. What developments would have eventually resulted we will never know, because a change in the style of warfare made the knight in armor outmoded, and jousting in that particular form disappeared.

If parallels to the history of the dinosaurs and the progress of American football are detected here, I must admit that they were consciously drawn. The dinosaurs, with their seemingly invulnerable armor, great size, and strength, failed to survive, whereas their near contemporaries, the grizzly bears, with only a shaggy coat of hair to protect them, have survived to this day. When one looks at today's

typical professional football player, it is hard to find an area of his body (at least when viewed from the front) that is not protected by some type of equipment.

Protective equipment in football has been introduced gradually over the years, whereas Rugby, from which football sprang, has contented itself with a rather inefficient ear protector for some players and occasionally a very light pad sewn into the shoulder of the jersey. Having examined the evidence as to the reason for this different development quite carefully, I would have to say that the reason was the introduction of techniques of play that laid open the possibility of serious injury to life and limb in American football.

The first of these was the abandonment of the scrum in favor of a scrimmage line of nine men. In 1883, interference ahead of the ball carrier was allowed. Tackling below the waist was permitted in 1888, but not below the knees. The flying wedge was introduced by Harvard in 1892, and flying interference by Pennsylvania in 1894. Players could be pushed or carried forward by teammates if they held the ball. Hurdling over the defensive line was permitted. Although mass plays and hurdling were prohibited in 1906, the game changed from a fluid to a more static action, so that it was possible to contact an opponent more solidly and accurately, thus increasing the danger of serious injury. The development of headgear, shoulder pads, and other protective devices followed naturally. Because of the development of this equipment, a player could hit harder with a greater assurance of his own safety. This required the development of more rugged equipment, setting up the cycle that has resulted in the best modern equipment based on high-impact plastic.

We have arrived at a point where, by the use of the face bar or mask and a molded protector for the teeth, we have virtually eliminated the serious facial and dental injuries of the past. But in the face mask we have a lever that the rules committee considers potentially so dangerous to the integrity of the neck that a 15-yard penalty is imposed for grabbing it. We have a helmet that provides extraordinarily good protection of the head, compared with models of 20 years ago, but 90% of the fatalities in football today are due to head and neck injuries, and there are many boys who survive but suffer irreparable brain damage or are paralyzed from injuries to the brain and spinal cord. This very good piece of equipment is being used as an offensive and defensive weapon--purposes for which it was never intended.

What injuries are we trying to protect against in football? If we classify injuries according to whether they are self-induced, result from contact with others, or result from contact with objects in the environment, we can see that there is little possibility of influencing the first group and that the main emphasis must be on the second and third groups. However, many football injuries are in the first group,

including especially those of the knee, which are a matter of utmost concern to all connected with football today.

We can protect the ankle joint to a considerable extent by taping it. And we tape other joints when they have been injured to support them, both during the recovery period and in a preventive way often for some time afterwards. We can put a harness on a shoulder and restrain its movement in such a way that recurrent dislocation may be prevented. No one as yet has developed a brace or support for the knee that can simultaneously allow the necessary range of motion and prevent injury. For protection against self-induced injuries, we rely principally on conditioning for development of strength in the muscles which help to support the joints.

With regard to injuries caused by violent contact with others, we are faced with a dilemma as far as the knee is concerned. But we are better off with the shoulder pad, which offers under some circumstances good protection for the acromioclavicular joint, although it plays only a minor role in preventing shoulder dislocation. We are trying primarily to prevent abrasions, contusions, and lacerations, and to some extent we are successful. We can damp the forces of deceleration in blows to the head, which have the potential of producing concussion or more severe brain injury, but we cannot eliminate them to an entirely satisfactory degree.

The environment of the football player is filled with hazards other than the hardness of the playing surface and the goalposts. The latter may be generously padded, and the effect of the former modified to some extent by the use of artificial turf. Control of coaches, players, and others on the sidelines, placement of benches and other impediments, and padding of walls and fences near the playing area are matters of environmental control that are often neglected but should not require the player to wear protective devices.

At a national conference on protective equipment in sports, held at the University of Wisconsin on 14-16 June 1968, there was general agreement that a variety of protective equipment was available, that there were great differences in quality, and that manufacturers had generally been responsive to public demand and had made sincere efforts, based on the information available to them, to provide satisfactory products. But research in the development of protective equipment has been more notable for its absence than for its achievements. Accordingly, the following recommendations were adopted:

Protective Equipment in General

(1) The further application of the use of protective equipment to injury prevention in sports depends on the availability of more accurate data on the incidence of all types of injuries in the sports

in question. The compilation and analysis of such data on a comprehensive basis depend on the establishment of a center for the continuous collection and reporting of pertinent information. This center must be adequately funded and staffed with personnel competent and experienced in the interpretation of such data. The success of its efforts will depend on the cooperation of all agencies sponsoring competitive and recreational sports and all persons supervising athletes, especially physicians and trainers. The establishment of uniform reporting techniques using standard nomenclature for sports injuries will be necessary. The cooperation of all committees, agencies, and organizations connected with the supervision and conduct of sports at all levels of amateur and professional competition, both individual and team, should be secured in obtaining raw data on injuries and fatalities for processing in the central registry.

(2) A committee on protective equipment in sports should be established. This committee's chief objectives would be: the maintenance of direct contact between individuals and organizations that represent various disciplines, professions, and business concerned with the safety of athletics and the development and use of protective equipment for sports; and the standardization of accident research methodology as it relates to this special field. To ensure the stability and perpetuation of its impact, the committee should be established under the auspices of an existing organization with related purposes, such as the American College of Sports Medicine. The membership of the committee should consist of representatives from the fields of physical education, sports supervision, recreation, medicine, safety education, engineering and design, sporting-goods manufacture, the supply of basic materials for manufacture, and perhaps other disciplines and occupations. Subcommittees should be formed to study the forces involved in football, ice hockey, baseball, wrestling, basketball, soccer, lacrosse, and Rugby. The frequency, direction, deviation, and magnitude of blows to various body parts should be determined as accurately as possible for each sport. Because of the frequency of death associated with head and neck injuries, the frequency of disabling knee injuries, and the high frequency of ankle injuries, first consideration should be given to soft-tissue stress tolerance in the head and neck, the knee, the ankle, the shoulder, and the thigh (in that order). Tolerance of the neck to forces resulting from spearing (i.e., butt tackling or butt blocking) should be ascertained, with emphasis on head positions that tend to lead to lateral inclination, extension, and flexion. (The effect of the cervical collar in mitigating such injuries should also be studied.) The subcommittees would then be able to proceed to the questions of establishing the necessary standards for protective equipment. In so doing, they would have to consider:

(a) present practices, procedures, and standards, to ascertain common denominators and arrive at a uniformity of nomenclature (this would include contacting groups that have established

criteria based on physical evidence for use as a basis for developing standards; those engaged in research on and manufacture of protective equipment might be asked for unpublished data on pertinent tests and findings);

(b) investigation of new, superior materials and products that might be applicable to the development of protective equipment;

(c) review of the literature to uncover research and information that might be applied to the formation of standards;

(d) soliciting of the release of information developed under federal support for space exploration, so that related aspects could be determined and findings applied as appropriate by researchers and manufacturers in the area of protective equipment;

(e) identification and classification of research needs in specific areas; and

(f) other specific problems involved in protecting spectators of, as well as participants in, the sports under study.

(3) Because well-fitting mouthpieces are more effective in protecting the teeth and reducing the incidence of concussions due to blows on the jaw and are more acceptable to players from the standpoint of comfort, careful fitting is recommended. To further this goal, it is urged that contacts be established and maintained between state high-school athletic associations, colleges and other agencies sponsoring contact sports, state dental associations, and state health departments to see that such services are provided to high schools at reasonable cost. Programs of fitting mouthpieces might be considered by dental schools or local dental associations as community-service projects. The possible role of dental hygienists and technicians should be explored to provide additional sources of manpower for such programs.

(4) There are many factors involved in the relationship between the occurrence of ankle and knee injuries and the design, placement, and length of shoe cleats; the surfaces on which games are played; the conditioning and experience of the players; and taping or wrapping of ankles. The best available evidence indicates that the use of shorter cleats and/or the use of round or otherwise modified heel cleats will reduce the occurrence of serious ankle and knee injuries. Because

practices and games take place on a variety of surfaces and under many different conditions, it is recommended that the type of shoe cleats permissible be standardized for all conditions on the basis of the average requirement. The soccer cleat appears to satisfy requirements of safety and general utility. Further studies should be made to determine optimal materials, design, length, and placement of cleats. Because of the variety of new playing surfaces now available, it is advisable that guidelines be established for the use of different types of footwear that will promote safety to the greatest extent on each playing surface.

Protective Equipment for Football

(1) The top-grade football helmet, when properly fitted and maintained, is effective in protecting a player from head, face, and mouth injuries in the course of normal play, for which it was designed. A misguided faith in its effectiveness in other situations has resulted in the development of coaching techniques in which the helmeted head is used as an offensive and defensive weapon. "Spearing," the use of the protected head in blocking and tackling, exposes a player to serious hazard of neck and spine injury. No equipment is available that will fully protect the neck and the cervical spine from compression injuries. A careful study of the total problem, including possible rule changes, and enforcement of present rules that minimize the problem are needed.

(2) Face protectors have effectively reduced facial and dental injuries. The multiple-bar protector is far more efficient as a protective device than the single-bar mask. Manufacturers are invited to use new materials in making face masks and to improve the method of attachment in cooperation with helmet manufacturers.

(3) Well-designed and well-fitted mouthpieces have eliminated almost all dental injuries and reduced concussions. Care should be used in selecting only top-grade, fitted mouthpieces.

(4) Cantilever shoulder pads, when fitted properly, provide the best protection for the shoulders.

(5) Rib pads provide adequate protection when properly placed and secured.

(6) Hip pads provide adequate protection when worn properly. They should be kept high enough to cover the iliac crest, to prevent "hip pointers."

(7) Thigh pads and knee pads, like hip pads, are effective if kept in the proper positions. Because of the frequency of injuries related to improper positioning, study of design to maintain proper position is indicated.

(8) The present football shoe has been implicated in the incidence of knee injuries. Several studies are underway of the relationship between shoe cleats and knee injuries, of various types of cleats, and of the relationship between various types of cleats and injuries on various types of playing surface. The findings of these studies should be widely disseminated and studied for further possible actions.

(9) A study should be made of the relationship between ankle injuries and high- vs. low-cut shoes. Little information is now available on this problem. Ankle wrapping and taping, applied properly, have shown some effectiveness in reducing ankle injuries. Further study is indicated.

(10) Protection of the hand and forearm is adequate.

(11) The cervical roll is used as an additional safeguard for a player who has recovered from a neck injury. Further research is needed to determine how effective it is.

No one is naive enough to believe that resolving the problems of protective equipment in football is going to eliminate serious injuries from the game entirely. This is an area where we can make considerable progress; but to do so, we must be willing to invest much more time and money than have been put in so far. The reason that accident prevention is so difficult, as the National Safety Council has shown us, is that there are approximately 300 unsafe acts for each one that actually causes an accidental injury; there are 29 nondisabling injuries for each disabling one; and there are 100 disabling injuries for each accidental death. Thus, to prevent an accidental death, one must prevent about 900,000 unsafe acts. In football, we are dealing with what one might call "planned accidents," and our endeavor is to make these accidents happen in such a way that they do not produce disabling injuries or deaths. In addition to finding the equipment that will help us do this job, we must direct our attention even more seriously to safer ways of performing the basically unsafe acts of football.

That brings me back to the football games that are played essentially without protective equipment--Rugby, Gaelic football, and Australian rules football. Do these games not have injuries? Of course they do. Are the injuries more serious than those sustained in American football? On the whole, no, although many are similar. Do they have fatalities? Yes, but proportionally fewer. Outside of American football's static scrimmage line (as opposed to the constantly moving attack and defense) and allowing of interference in front of the ball-carrier, why should players in other football games not suffer more serious injuries in rugged contact sports, inasmuch as they do not wear protective equipment?

I would like to suggest one important difference: the spirit in which the games are played. The rules of American football fill several

hundred pages (including interpretations) in the most recent version. The rules of the other football games are contained in a very few pages each. The interpretation of how the other games shall be played is a responsibility mainly of the players, rather than the officials, who are present to assist the players, rather than regulate them. Tactics that are dangerous, unfair (although legal), and illegal are not resorted to deliberately by the players, because "it just isn't Rugby," for instance. If we could see a development of this attitude toward American football on the part of players and coaches, we might go a long way toward reducing our injury toll.

THE RELATIONSHIP OF TURF, PLAYING CONDITIONS, AND EQUIPMENT TO INJURIES

JACK ROCKWELL

PLAYING CONDITIONS

With regard to their relationship to injury, we must look at playing conditions from several different approaches.

Various playing conditions are brought about by temperature and climatic changes. Playing on a wet, soggy, slippery field is certainly going to contribute to injury. That is equally true of a field that is cement-hard, when a game is played under a hot sun with very high or very low humidity. But, unless we play all our games indoors, there is not a great deal that we can do about the weather.

One thing that particularly disturbs us is playing in extreme cold. How the Dallas and Green Bay players came through their game of December 1967 without suffering serious injury, I will never understand. Not only the players, but everyone else involved--coaches, commentators, spectators, and especially officials--suffered tremendous discomfort during that game. It would seem that some standard should be established whereby, if stated temperature, humidity, and wind factors were prevalent, a game would be rescheduled.

Other field conditions that we can and should control include uneven, badly prepared fields; skin infields; poor lighting; the placement of benches, chairs, and other obstructions too close to the field; improperly padded goalposts; poorly trained linesmen; and, last but far from least, the television and newspaper cameramen who all too often get into such positions that they can cause serious injury to the players, as well as themselves. It seems inconceivable that we should still be placing added risks of injury on our players by allowing such conditions. It must become a matter of individual team policy to correct them.

EQUIPMENT

It is difficult to differentiate between equipment-related injuries and those unrelated to equipment. The most common injury has to do with cleated shoes and ankle and knee injuries. With the evidence available at present, it seems that the wearing of short broad-tip cleats with disk or corrugated heels has helped appreciably in decreasing ankle and knee injuries. More research must be done in this area.

The NCAA recently adopted a rule that makes the wearing of any cleat longer than 3/4 in. illegal. In 1970, the steel-tipped cleat will also be banned. This is an excellent rule, because the "burred" steel-tipped cleat can be an extremely dangerous piece of equipment.

A general equipment problem is the improper wearing or fitting or the complete lack of some equipment. It seems out of character for today's professional football player not to wear equipment properly, and even stranger for him not to wear some pieces at all. Players who spend months conditioning themselves to peak physical shape, who watch their diets, and who spend hours making sure that helmets, shoes, and uniforms fit properly will then refuse to wear mouthpieces and attempt to get on the field without wearing thigh guards or knee pads. Even improperly sized or fitted thigh guards or knee pads can cause serious injury, as the Cardinals found out this year. By allowing a 2-in. opening on the anterior thigh between the top of the knee pad and the bottom of the thigh guard, one of the players incurred a deep contusion, the result of a kick, which forced him out of action for 11 of the 14 league games.

It is essential for the safety of the players that they be provided with the best equipment available, that it fit properly, and that they wear it.

TURF

I mentioned earlier the climate conditions over which we had little control and field conditions over which we had at least some control. Now, with artificial turfs, we can overcome some of the factors that have before been uncontrollable.

Artificial turf is not a panacea, as both the major manufacturers might have us think. Some of the claims made for (and against) the turf are not only ridiculous--they are downright absurd! I have gathered statistics over a 2½-year period, but they are so inconclusive that I will not present them here. There is no doubt but that artificial turf will help to reduce the incidence of many of the injuries that we have been plagued with in the past. Of equal importance is the fact that it will provide a consistent surface, day in and day out, for practice, as well as for games in any type of climate.

Probably the most pressing problems facing the manufacturers of artificial turf at present are the development of an all-purpose shoe, the development of an underpadding of consistent resiliency for all sports, and the development of a surface acceptable to both baseball and football. All these problems are being worked on and will almost certainly be solved soon. With the nationwide acceptance of these products--at present, over 100 schools and teams have indicated enough interest to invite bids--it will be a matter of only a few years until we are all playing on artificial turf.

THE RELATIONSHIP OF FOOTBALL INJURIES TO PLAYING POSITIONS: STATISTICAL REVIEW OF INJURIES OVER 9 YEARS

JAMES A. NICHOLAS

This report is based on a continuing analysis of injuries to a professional AFL football team in 1960-1968. Obviously, injuries have many causes. It is not my intention here to explore causes, but rather to obtain statistical data in an effort to determine whether there is a relationship between position played and the incidence and severity of injuries.

The team used was the AFL New York franchise, the Titans from 1960 through 1962, and the Jets since then. The Titans team was made up of a great many rejects and free agents, and was put together rather hastily in 1960. By 1963, the franchise had been turned over to an efficient organization and had become known as the Jets; it culminated a 6-year building program by winning the Super Bowl on 12 January 1969.

Table 1 presents the statistics of the player pool studied. The total season roster over 9 years consisted of 435 or 445* playing positions, using 146 men. An average of 26 men returned from the previous season, and there were an average of 23 new faces per year, consisting of 11 men from taxi squads and 12 men picked up over the season by other means.

OCCURRENCE OF VARIOUS KINDS OF INJURIES

Table 2 analyzes the significant injuries in 1960-1968. These are defined as injuries because of which a player missed two consecutive games in a season. This figure has declined from 49% of the squad in 1960-1962 to 39% in 1963-1965 and to 25% in 1966-1968. In this series of 435 or 445* positions, covered by 246 players, 96 players were on offense, 97 were on defense, and 53 were on special teams. Sixty-eight percent of the personnel had injuries at one time or another that kept them out of two consecutive games. Of 246 different players, 166 were hurt significantly--70 of 96 (73%) offensive, 53 of 97 (55%) defensive, and 43 of 53 (81%) special-team. Table 3 shows that the preseason injury rate was 29%.

* See footnote *b*, Table 1.

TABLE 1
Player-pool statistics, 1960-1968[a]

Year	1960	1961	1962	1963	1964	1965	1966	1967	1968	Total[b]
1960	36/12									48
1961	25	11/10								46
1962	10	5	12/8							45
										139
1963	18	3	2	15/13						51
1964	6	1	2	12	13/10					44 or 49
1965	5	1	1	6	10	15/15				33
										148 or 153
1966	4	1	0	5	10	8	10/8			46 or 51
1967	3	1	0	5	8	5	7	11/13		53
1968	3	1	0	4	8	5	5	7	8/8	49
										148 or 153
										435 or 445

Total season roster 435 or 445[b]
Actual individual players 246
Average per year: 49

Returnees: 26
New faces: 23
Free agents: 11
Trades: 12

[a] 1960-1962, Titans; 1963-1968, Jets.
[b] Because of rule change, five players, other than taxi squad, in 1964 and 1966 were transferred to special reserve pool, to be activated only after minimal number of emergencies.

Table 4 illustrates the locations of the injuries (in both regular and exhibition seasons). By far the largest number of the total of 377 injuries were to the knee, 122 occurring, requiring 85 operations. The thigh, leg, ankle, and foot, labeled under one classification, constituted the second most common group, with 100 injuries. Injuries to the rest of the body accounted for about 40% of the total. One hundred seventeen operations were performed for the 377 injuries.

Tables 5, 6, and 7 illustrate the frequency of football injuries by regions and diagnosis. The most common locations in the upper extremity were the hands and fingers, including fractures and tendon ruptures. Acromioclavicular separation, rotator cuff tear, direct shoulder contusion, and recurrent dislocation of the shoulder were also common. Rib

fractures and tears of rib cartilages were much more common than were other injuries to the thorax or upper extremities, with concussion and low back injuries running a close second. The most common disabling injuries to the lower extremities, excluding the knee, were first- and second-degree ankle sprains, "pull" and contusions of the thigh, and fractures and sprains of the foot.

TABLE 2
Significant injuries in regular season, 1960-1968

Year	No. players[a]	No. players hurt			
		Total	By position		
			O	D	ST
1960	48	22	15	7	0
1961	46	20	13	5	2
1962	45	26	12	9	5
	139	68 (49%)	40	21	7
1963	51	21	10	7	4
1964	44 or 49	28	8	8	12
1965	53	10	3	4	3
	148 or 153	59 (39%)	21	19	19
1966	46 or 51	14	3	6	5
1967	53	16	5	4	7
1968	49	9	1	3	5
	148 or 153	39 (25%)	9	13	17
All positions:	435 or 445	166	70	53	43
Different players:	246	37% of positions, but 68% of personnel			

Conclusion: during the regular season, 37% of total squad injured sufficiently to lose two consecutive games; estimated preseason injury rate, 28%; but 70 of 96 players hurt on offense (73%), 53 of 97 players hurt on defense (55%), and 43 of 53 players hurt on special teams (81%).

[a] See footnote *b*, Table 1.

As seen in Table 7, there were 49 major ligamentous ruptures, which were classified as second- or third-degree tears, and 27 players had effusions associated with minor sprains. Thus, there were a total of 76 ligament injuries to the knee, of which 38 were operated on and in which cruciate and/or collateral ligament rupture was found; 11 others with such injuries were treated conservatively. Meniscus injury was the second most common finding, with 19 cases operated on. A miscellaneous group of injuries included loose bodies and degenerative

lesions, and 19 players with these injuries were operated on. A total of 76 knees were operated on; 38 of the ligament ruptures were repaired within 48 hr after injury. Nine players had subsequent late reconstructions.

TABLE 3
Injuries in exhibition season, 1960-1968

Year	No. players	No. players hurt
1960	69	18
1961	73	26
1962	79	21
	221	65
1963	103	22
1964	104	28
1965	78	18
	285	68
1966	76	28
1967	74	24
1968	72	26
	222	78
Totals	728	211 (29%)

Two-game loss rate: 75/729 (10%)

TABLE 4
Location of injuries, 1960-1968

I.	Head-neck-abdomen-spine-pelvis	59
	Most frequent site - rib cage 14	
	operations 3	
II.	Shoulder girdle to humerus	46
	Most frequent site - glenohumeral 22	
	operations 6	
III.	Elbow to hand	50
	Most frequent site - fingers 16	
	operations 7	
IV.	Thigh-leg-ankle-foot	100
	Most frequent site - thigh 35	
	operations 16	
V.	Knee	122
	Most frequent site - ligaments 76	
	operations 85	

Total injuries: 377

Resulting in surgery: 117

TABLE 5
Injuries to upper body and upper extremities, 1960-1968

	1960-1962	1963-1965	1966-1968	Total
Head, Neck, abdomen spine, pelvis				59
Concussion, jaw Fx (2)	4	2	3	9
Neck sprain, Fx, Disl, nerve	3	2	2	7
Rib Fx, tear of cartilage	2	4	8	14
Low back injury (contusion)	5	2	2	9
Disk	2	1	1	4
Congenital	2	1	1	4
Other	1	2	2	5
Abdominal-pelvic contusion	3	3	1	7
	22	17	20	
Shoulder girdle-humerus				46
Brachial plexus "pinch"	2	1	1	4
Contusion - shoulder	4	2	2	8
Rotator cuff tear	1	3	3	7
AC separation, 1^{o}, 2^{o},	2	4	2	8
3^{o}	1	1	1	3
Shoulder dislocation	4	2	1	7
recurrent	1	1	0	2
Miscellaneous	3	3	1	7
	18	17	11	
Upper extremities - below elbows				41
Dislocation - elbow	1	1	1	3
Fx-Dislocations-hand-digits	4	5	7	16
Fracture - forearm	0	1	2	3
Tendon rupture fingers	3	2	6	11
Other	4	2	2	8
	12	11	18	

TABLE 6
Injuries to the lower extremities, excluding the knee, 1960-1968

	1960-1962	1963-1965	1966-1968	Total
Thigh				
Contusion (charley horse) or fracture	4	5	5	14
1° and 2° strain (pull)	6	6	3	15
3° strain (major tear)	3	2	1	6
	13	13	9	35
Leg				
Fracture	1	2	0	3
Calf muscle tear (1°-2°)	3	2	2	7
Achilles tendon rupture all operative	2	1	0	3
	6	5	2	13
Ankle				
Loose bodies, chondral fracture	3	2	2	7
Talotibial-interosseus impingement	1	1	4	6
Instability	5	2	5	12
operated on	1	1	1	
	9	5	11	25
Foot				
Metatarsal joint sprains	2	4	1	7
Fractures	2	3	5	10
Other	4	4	2	10
	8	11	8	27
Totals	36	34	30	100

RELATIONSHIP OF POSITION, AGE, AND EXPERIENCE TO INJURY

Major injuries were defined as those which resulted in operations and/or in a loss of at least eight consecutive games during a season. In 1960-1968, as illustrated by Table 8, 22 players sustained such injuries on offense, 22 on defense, and 17 on the special teams. Ten of the 17 special-team injuries occurred on the kickoff or punting team, seven on the punt-return team, and none on the field-goal or extra-point team. One-third of the special-team injuries occurred in the 1964 season, and five offensive injuries occurred in 1967.

When one relates the position played to the age and experience of the player, it is evident that the younger players are more frequently injured and operated on (Table 9). Once the player has 2 years of experience, the operation rate drops, although the total-injury rate may continue. Of course, this may be due to many factors and should not be rigidly interpreted. (Strangely, only 20% of the injuries occurred on the home field.)

Table 9 illustrates that on the offensive team 15 of the 22 hurt were backs. Twelve of the 22 occurred during their first 2 years. Of the 22 defensive injuries, six were in backs and 16 in linemen; 17 of these 22 occurred during their first 2 years. On special teams, 14 of the 17 injuries occurred during their first 2 years. Thus, 43 of the 61 major injuries occurred in players with less than 2 full years of experience and 54 of 61 in players with less than 3 years of experience.

TABLE 7
Injuries to the knee, 1960-1968

Sprains	1960-1962	1963-1965	1966-1968	Total
Minor sprains	12	10	5	27
Major ligament tears	12	19	18	49
not operated on	4	5	2	11
Total ligament injuries	24	29	23	76
operated on	8	14	16	38
Cartilage				
Meniscus tears (isolated)	4	8	7	19
Medial	3	3	5	11
Lateral	1	2	2	5
Both	0	3	0	3
Injuries to Extensor Apparatus				
(Includes dislocations, fractures)	2	4	2	8
Degenerative lesions (operated)	9	5	5	19

Total knees operated on: 38 + 19 + 19 = 76
Plus 9 late reconstructions of ligaments

If one correlates the relationship of injury to the number of contacts per position, the figures are revealing. The total number of running plays in the 9 years of this team's existence was 3794; that number of times, 11 men on the offense moved with the snap. There were 4116 passing plays, making a total of 7910 offensive plays. Of the 70 offensive players who suffered significant injuries, 22 suffered major injuries. It was calculated (Table 10) that every 360 offensive plays produced a major injury, and every 113 plays, a significant injury. One major injury of an offensive player occurred in every five games, and a significant injury in every one and a half games.

The defensive figures were close. Some 4120 running plays and 3980 passing plays were defended against, for a total of 8100. Of the 53 defensive players who suffered significant injuries, 22 suffered major injuries. Every 368 defensive plays produced a major injury, and every 153 plays, a significant injury. One major injury of a defensive player occurred in every five games, and a significant injury in every two games.

Players on the special teams had a much higher morbidity rate per contact. They were involved in 1759 plays. Of the 43 special-team players who suffered significant injuries, 17 suffered major injuries. Every 103 special-team plays produced a major injury, and every 41 plays, a significant injury. One major injury of a special-team player occurred in every seven games, and a significant injury in every two games. The differences may be explained by the occurrence of fewer contacts in a game, but much higher injury rates. The chance of major injury on special teams, according to this study, was three times greater than on either offense or defense.

DISCUSSION

Exhibition statistics were kept separately and are difficult to analyze because of the tremendous turnover in personnel. In this series, there was no higher rate of significant injuries than in the general season rate, coming to some 29% in only five preseason games per year. It should be recognized that many men came to camp with injury problems that were compounded, and these figures require a great deal more analysis, because they are not yet valid for interpretation (nor do they properly come within the scope of this paper).

In evaluating injuries, many factors come into play: field conditions, equipment, player activities, types of contact, and temperature and humidity (their exact nature requires further elucidation, which is now in progress with computers). It should be emphasized that, to obtain adequate data in such studies, injury reports must be standardized. When different teams report injury, there is an inherent error; this is where the great problem lies, unless nomenclature, the characteristics of the observer, and techniques of reporting are standardized, as in this paper. Careful notes are necessary as to whether an injury

is early or late, or compounded on previous injuries. There must be a good description of combinations of injuries and their severity. One must decide whether a particular injury was new, a reinjury, or a cumulative injury. Even players themselves have difficulty recognizing and describing how they got hurt.

TABLE 8
Major football injuries by position, 1960-1968

Year	Total	Offense	Defense	Special teams	Kickoff/ punting	Kickoff return and punt return	Field goal and extra point
1960	3	1	2	0	0/0	0	0
1961	5	3	1	1	0/0	1	0
1962	5	3	2	0	0/0	0	0
	13	7	5	1	0	1	0
1963	7	1	4	2	0/1	1	0
1964	9	4		5	2/0	3	0
1965	9	3	4	2	1/0	1	0
	25	8	8	9	4	5	0
1966	8	2	4	2	0/1	1	0
1967	10	5	3	2	2/0	0	0
1968	5	0	2	3	1/2	0	0
	23	7	9	7	6	1	0
Totals	61	22	22	17	10	7	0

The player himself is one of the most important factors in injury. For example, on the current New York Jets team are 12 (of 40) men who have never been operated on and 28 men who have had a total of 36 operations. What makes these 12 men injury-free is an important subject for investigation; there seems to be a relationship between loose-jointedness and ligamentous injuries, and between tight structures and muscle pulls, backache, and tendon rupture. The position of the player, his familiarity and experience, his size, and his past history (including previous injuries) are important, as are contacts, asymmetric strength of extremities, coordination, balance, speed, and ability to roll, which all figure in the prevention of injury. The problem of protection in a given play--for example, whether a quarterback is exposed to pocket breakdown--is important in the problem of injury and requires further study. The offseason condition of the player, the use of drugs, and functional aspects are difficult to evaluate but may certainly play a role in proneness to injury.

TABLE 9
Relationship of position, age, and experience to injury, 1960-1968

	Offense			Defense			Specialty	
	Back	Line	Total	Back	Line[a]	Total	teams	Total
1st year	7	1	8	4	10	14	10	32
2nd year	2	2	4	1	2	3	4	11
3rd year	3	2	5	1	3	4	2	11
4th year	2	0	2	0	0	0	0	2
over 4 years	1	2	3	0	1	1	1	5
	15	7		6	16			61
Totals	22			22			17	

[a] Including linebackers.

Specific positions:

QB	5	DE	6
HB	7	DT	5
FB	2	OLB	3
FL	1	MLB	2
SE	1	QB	3
OT	1	S	3
OG	3		
C	2		
	22		22

From this study, it is obvious that knee injury is the most serious problem in terms of career-ending potential and that the younger the player, the more likely he is to be hurt. That may be because he is more frequently placed on special teams and because he is less experienced. Moreover, it is evident that some positions have a greater predilection to some kinds of injury, with a high morbidity that requires further analysis. Perhaps even coaching methods and the use of personnel play roles in injuries.

A statistical analysis of football teams over a sufficient period would be useful in revealing the true relationship of injury to position. Once a relationship is established, a different approach in protective technique and equipment might help to eliminate the risk of injury. However, it is unwise to make dogmatic statements or predictions, because the data at hand are inadequate. This is the first statistical survey of a team's injuries covering 9 years and using standard techniques of nomenclature, examination, and reporting by the same physician to reduce bias. It is suggested that, in any computerized study of many teams, nomenclature and the techniques of history-taking and examination be uniform to avoid any possibility of error.

TABLE 10
Relationship of injury to position

	Major injuries	Total injuries	Total running plays	Total passing plays	Total plays	Total runs kickoff	Total runback	Extra point	Field goal	Total
Offense	22	70	3794	4116	7910					
Ratio	1:360	1:113								
Defense	22	53	4120	3980	8100					
Ratio	1:368	1:153								
Specialty teams	17	43				569	606	333	251	1759
Ratio	1:103	1:41								

THE RELATIONSHIP OF GAME RULES TO INJURIES

MARK DUNCAN

What is the purpose of rules in football? They are intended to prevent one team from taking advantage of another through physical acts or through producing involuntary reactions with unsportsmanlike conduct. We know what the physical acts are--clipping, holding, etc. But they are not the acts that cause our main problems. It is the unsportsmanlike conduct that causes involuntary reactions on the part of an offensive team that really create havoc.

Football coaches have their players finely trained and well drilled on starting counts. An offensive player on the line of scrimmage is concentrating on the quarterback, listening to his terms. Suddenly, a defensive player on the line of scrimmage shouts "Go." If I do that in a room, heads in the audience will jerk. When it happens on a football field, there is chaos; they are not supposed to go. It does not have to be "go." It can be almost anything, and these people explode.

Another kind of situation that has caused rule changes, such as about 3 years ago, involves the little safety man standing back and sticking up his hands to signal for a fair catch. The man covering the kick, when he sees that, knows that the man is protected; he cannot hit him. But now the little safety man has a free shot at a man to keep him from downing the ball before it goes over the end line. Here a rule was changed to prevent injury, of which we had several before we got the rule changed.

We are very proud of our rules in professional football. They are probably superior to any other code of rules. Some things in professional football make it different from college football. Take the case of the personal foul. In college football, a personal foul is often not penalized. In professional football, every personal foul is penalized. If a college quarterback throws a touchdown pass but is roughed on the play, he must refuse the touchdown if he wants to accept the penalty for roughing. That is ridiculous, because he never wants to refuse a touchdown, and the defense therefore has a free shot at the passer. In professional football, every personal foul is penalized; in the case mentioned, it would be penalized on the next kick. We have

no rules that I can think of that allow one player to take advantage of another, through any act. Each player is protected against the other.

Silly as it might seem at times, players are becoming more and more conscious of rough play on the field. Fewer players are now taking free shots. In professional football, we have relatively little spearing, as college football has.

I would like to sketch some of the history of rules changes in football. From 1895 until about 1917, there was very little change in football. In 1917, the federal government said that something had to be done about football from the standpoint of rules or it would abolish the game. At about that time, the NCAA rules committee was established, and the first real code of football was instituted.

It was not until 1942 that headgear was made mandatory in professional football. At the same time, hose was made mandatory, to prevent injuries, cuts, infections from cuts, etc. Even if professional football players did not have to wear hose, I think they would, because it is a good holder for their shin guards, and most professional football players wear shin guards.

It was not until about 1947 that plastic headgear was approved. Football was very slow in adopting plastic headgear and, after that, tried to develop a foam-rubber casing that would go over the top of the helmet, to protect the opponent.

In 1949, professional football banned the aluminum cleat.

In 1955, the rule change was made that probably helped professional football more than any other one factor, from the standpoint of injury. The ball carrier, once on the ground, was prevented from getting up and advancing. Before then, a man could be tackled or knocked down, and could then crawl, get up, and run; if he tried to get up and run, many people were jumping all over him. The rule now is that, if any part of the ball carrier's body, other than his hands and feet, touches the ground, if he has been contacted by an opponent, the ball is dead.

In 1956, grabbing the facemask of any player except the ball carrier was outlawed and given a 15-yard penalty. In 1957, the ball carrier was included. At each year's professional-football rules meetings, someone recommends that we drop the penalty of 15 yards for grabbing the facemask. Such recommendations have so far always been defeated.

In 1965, we added a sixth official. Surprisingly, there was a decline in penalties in the first year with six officials. We feel that this was another means of preventing injury.

Also in 1965, professional football established the single-standard goalpost. This helped us by 50%; at least there was a smaller chance that someone would run into a goalpost.

We think free substitution also prevents injury, because it helps to prevent fatigue. One rule involved in free substitution in professional football is that a player must cross the sideline on the side of the field on which his team is seated. Once, when Baltimore was playing Chicago in Chicago, a Baltimore player went off the field on the Chicago side and ran into trouble. The rule now is that a player has to cross the sideline on the same side as that on which his team is seated.

We are greatly concerned about the handling of injured players, from the standpoint of doctors and trainers coming onto the field. In several situations, we have received a great deal of criticism involving the treatment of injured players on the field as it affects playing time--timeouts, etc. We must come up with some kind of fair rule for this sort of thing, and I am sure that we will.

We in professional football are very fortunate in that, as opposed to colleges and high schools, we have a preseason, during which we can experiment on many different phases of our game. It gives us a good opportunity to experiment with rule changes and other aspects of football. And the primary goal of such experimentation is to keep the players from being injured.

BIOMECHANICAL ANALYSIS OF FOOTBALL INJURIES

VICTOR H. FRANKEL

There are three basic areas of biomechanics as applied to the musculoskeletal system: physical biology concerns the determination of the physical properties of biologic tissues, substructures, and functional units necessary to an understanding of the musculoskeletal system; human engineering deals with the man-environment interface, including preventive engineering with regard to sports injury; and engineering research and development have as their output such tangible products as prosthetics, orthotics, and implants. On the basis of the methodology derived from this organizational concept of biomechanics, an outline for the biomechanical analysis of football injuries has been developed. Information in three areas--injury occurrence, kinematics, and functional loading--is necessary.

INJURY OCCURRENCE

A careful statistical analysis is necessary to establish the frequencies and anatomic sites of injuries. The determination of exact anatomic sites may prove difficult, owing to the similarity of symptoms arising from different structures. Obviously, the analysis can be no better than the input data, which must be prepared in a simple, easily scored form. Once a pattern of injuries has been established, refinement of anatomic diagnosis and coding may take place, before the development of a prospective study. If the examiner does not have a thorough knowledge of what to look for, subtle injuries to the musculoskeletal system may be overlooked.

It may be possible to develop tests of human characteristics that depend on a functional analysis of the musculoskeletal system. Such characteristics as reflex time, agility, force-velocity relationships for muscle, and proprioception need to be considered. Easily applied tests must be developed to demonstrate changes in these important characteristics that may be related to age or deconditioning. Perhaps an injury-prone player could be identified through a battery of such tests. A change in a player's capabilities might be identified. It is certainly of interest to identify what "goes" when the legs go! Tests of this nature are under development at the Biomechanics Laboratory of Case Western Reserve University for the study of elderly patients with fractures of the neck of the femur.

The physical conditions under which the game is played certainly play a large role in injury production. The physical conditions are determined by the playing field and the equipment. The playing field is studied through the science of soil mechanics. The time-dependent viscoelastic properties of the turf and soil are important, as are the energy-absorbing characteristics. Wetness and temperature may change the load-deformation curve and energy-absorbing characteristics of the field. It would seem important to develop a turf that has a large energy-absorbing capacity but that permits energy absorption without the development of high frictional forces. At the same time, the turf must have dimensional stability in the horizontal plane to prevent ground reaction in running and turning.

Equipment is designed to absorb the energy of contact blows and to distribute it in a manner that will prevent damage to the body. The efficiency of the equipment in doing that is studied by noting its elastic properties, energy-absorbing capabilities, and functional behavior. The elastic properties of the material and structure are determined through loading tests and measurement of deformations. The data may be reported as a stress-strain curve or as a load-deflection curve. A stress-strain curve is useful, in that the modulus of elasticity (an indicator of stiffness) may be read from it. The energy put into the system during the load test for any given load or stress may be found by integrating the area under the load-deflection curve. This allows the designer to pick the appropriate material from which to form the item of equipment. Such a substance as foam rubber will absorb energy by undergoing compression but will develop a low resistance in the process. Metal, when absorbing energy, will deform very little but will develop a high resistance. A compromise between deflection and force must be made, and the material picked must meet the requirements imposed by the compromise.

The functional behavior of equipment needs careful assessment. A helmet may protect a player's head but allow his neck to hyperextend. An ankle support may protect his tibiotalar joint but restrict the motion necessary for proprioception.

KINEMATICS OF INJURY

Kinematics, the science of motion, allows one to characterize motion scientifically and mathematically. The basic data that must be used are displacements and time. Linear and angular displacements are found by a number of techniques. Perhaps the oldest is the use of multiply triggered cameras, which led to the development of the motion-picture camera. Game films can be studied, not only from the standpoint of the play but from the standpoint of injury production. Stroboscopic photographs are a refinement of the motion-picture technique. Points or lines drawn on a figure may be identified in stroboscopic pictures. Linear and angular displacements of the lines are noted. If a time

scale is provided, via either the film speed or the strobe light frequency, the time between individual displacements may be measured. Velocity, the change in position (displacement) divided by time, may be determined from the photographic data.

Velocity increase (acceleration) and decrease (deceleration) may be similarly identified. Linear and angular accelerations of the body and its extremities may be found, and decelerations produced during contact may be calculated. Another derivative of displacement is the jerk, the time rate of change of acceleration. This needs to be studied in relation to the injury problem, inasmuch as large accelerations and decelerations may be tolerated by the body if they are spread over long periods. An understanding of this phenomenon can be gained by catching a hard ball in bare hands; after the first few tries, the hands are pulled in the direction of the ball as it is caught, thus spreading out the time during which the ball is decelerated. The accelerations may be determined directly through the use of accelerometers, as has been done for helmets. However, it is difficult to mount accelerometers on the limbs, because the skin and soft parts move so much.

Because it is necessary to have an understanding of the bodies, velocities, and accelerations in three planes, we are currently developing a television scanning system that uses two cameras at right angles to each other. Data are automatically processed to indicate velocities and accelerations.

The configuration of a body at the time of injury and the areas exposed to contact may be determined through photo-optical techniques. Simultaneous viewing of the play from sideline and endzone cameras would yield useful data.

FUNCTIONAL LOADING

Internal and external forces act on the body during a traumatic incident. The internal forces result from muscle action, joint reaction, and stretching of ligaments and capsules during function. For such an activity as punting, the quadriceps must produce a force three times body weight to produce the required acceleration at the knee. The joint reaction will be nearly equal in magnitude to this force and opposite in direction. These equal and opposite forces are necessary for the production of a couple that causes the knee to accelerate forward. Large forces may develop similarly, owing to stretching of ligaments. When a player bends forward, the posterior longitudinal ligament and other posterior ligaments are stretched and produce a load on the intervertebral disks.

The internal forces are determined from the acceleration data obtained from kinematic studies and the body dimensions and other characteristics. Because force is equal to the product of mass and acceleration, if the mass and acceleration are known, the force can be found. Similarly,

torque is equal to the product of the polar moment of inertia and acceleration. Both the mass and the polar moment of inertia of the limbs and trunks are easily found. From a knowledge of the forces and torques based on free-body analysis, the joint reactions may be found.

External forces may be produced by body contact, gravitational accelerations, and the ground reaction. These forces may be studied with the aid of a force plate, which measures the forces and moments of the ground reaction. A force plate could be constructed that would be an integral part of the field, and the ground reaction associated with running, cutting, and blocking maneuvers found.

All these factors lead to an understanding of the traumatic loading of the injury site. This knowledge is necessary if injuries are to be prevented. It is often possible for a keen observer to develop an understanding of injury mechanics by experience. He is in the same position as the physician who studies a limp and attributes it correctly to a paralysis of the tibialis anterior. When he prepares a brace prescription, he again uses his judgment as to how strong to make the dorsiflexion spring. It is possible, however, with the biomechanical techniques briefly described here, to arrive at an exact knowledge of the accelerations and decelerations of the ankle produced by the muscle and to replace this function exactly. So, too, should it be possible to analyze the player, the equipment, and the field and to determine scientifically the factors leading to and preventing injuries.

COMPUTERIZATION OF INJURY DATA

VERGIL N. SLEE

Dr. Ryan has recommended the development of a registry of sports-injury data, and Dr. Kraus discussed the importance of the epidemiologic method in studying events and getting clues for further studies. I would like to discuss some aspects of the establishment of a computer system for handling large numbers of reports about football injuries--that is, setting up the injury registry that Dr. Ryan envisioned and laying the groundwork for much of the epidemiologic research considered by Dr. Kraus.

It is entirely feasible to develop a large-scale comprehensive injury-reporting system. I speak, not from experience in handling injury data, but from experience in handling large amounts of data from hospitals. Our organization operates a system (the Professional Activity Study) that receives approximately 10,000,000 medical-record summaries each year from over 1200 hospitals scattered across the continent in the United States and Canada (and some foreign countries). Operating this system has taught us some things that would be important if one were trying to set up an ongoing, continuous system for collecting sports-injury data.

There are two ways to get data. One is to make a special study--to go out and pick up data and then come home and study them. That is not what we are talking about. We are talking about a system in which injury data are accumulated constantly at a central point, deposited, and kept ready for rapid retrieval and analysis by computer.

It seems to me that such a development could have three general uses. The first would be a registry. One could search for individual cases with specific requirements that someone is interested in. Any factor that is recorded in the system could be used for indexing. One would not put all the details available on a given episode into the central computer system; it would sink under its own weight. Rather, one would put in selected data about the event, and the indexing would permit backtracking to the home team for the detailed records of individual cases. One could then search for details (which, it is hoped, would have been recorded) when necessary for special studies. To illustrate

this registry function, 4 or 5 years ago, we had an experience in mobilizing a search through hospital records for specific patients who might have been injured by a particular drug. With an elapsed time of 48 hr, we had figured out what to look for and searched 3,500,000 records by computer. We located approximately 300 possible cases that fit the criteria. That was a registry function. Most registry functions, incidentally, would not have to involve computer runs, because one regularly prints out lists of cases in various arrangements, and many registry searches are done by eyeball. There is no more point to sending a man on a boy's errand than to sending a boy on a man's errand. One of the problems we are having today is that people think that computers always do their jobs by being called into play when the crisis occurs. That is the hard way to do it. You can usually anticipate the types of things you are going to want and the types of searches; then it is a lot cheaper and simpler to get ready in advance. It is easier, for instance, to pull a dictionary off the shelf and find the meaning of a word than to use a computer for that type of problem.

A second use for a computer system would be to produce descriptive statistics on a routine basis. I would visualize an essential part of a computer injury-data system as feeding back to the contributing teams a periodic, regular flow of information--statistics on kinds of injuries, age distributions, and the circumstances under which injuries occurred. Of course, if one fed back data only on the individual team, the system would not be nearly as useful as it could be. It could be enormously improved by feeding back comparisons, so that a team could see how it stood in comparison with others. This could be done confidentially; there are techniques that could be used if the teams wanted to keep secrets from one another, and comparisons would still add a great element of interest and value.

A third major use for such a system would be to carry out nonroutine studies, doing epidemiology of the sort that has been mentioned throughout this workshop. No one would be able to predict all the tables that should be produced or the correlations that should be examined in any set of data. The way to get around that is to devise the system so that such studies can be done on demand. If one wanted, for example, a more detailed age breakdown of the players than is in the routine tabulations, it would be simple to go back to the data and produce it.

The system that I would design would have a central file of information, whose content would be reports of injuries. These would not be in great detail. Some reasonable degree of detail could be achieved to balance the wishes of the researchers against the various problems of getting forms filled out and the cost of handling information. One must get enough so that it is worth while, but not ask for so much that no one will collect it. The information would come from the many contributing teams in such a standardized form that comparisons would be valid. It would be retained by electronic memory devices, and the computer would be the retrieval mechanism.

There is one critical assumption behind the proposal of such a system: that the teams contributing data have or would create good routine basic record systems. There would always be a need to be able to go back to the teams and get greater detail on specific cases for more exhaustive studies. Each team must have a system to make this possible. Furthermore, each team must have a system for keeping baseline data. It should be possible to find out how many players on a given team wore low and how many high shoes and when, how many players actually wore protective gear, when new devices were introduced, etc. Those items are typical of the baseline data that must be available if one is going to be able to interpret the statistics and carry out epidemiologic studies. Careful thought must be given to the data that each team should maintain routinely, both on injuries and on activities--data that would be available on demand as necessary for studies.

Some of the things that must be considered in designing a computer system are described briefly in the following paragraphs.

Output

I have already mentioned three kinds of output: the registry, routine statistical descriptions, and epidemiologic studies.

Input

I have alluded to input. The input must be reasonable--it must be in a form that people are willing to contribute and that is not too time-consuming. It must not call for too many decisions on the part of the person filling out the form. (Of course, I see information coming into the system by having a form filled out.)

The design of the input piece of paper is very tricky and deserves a lot of work. The form has to be kept simple if it is going to be successful, and its questions must have as little ambiguity as possible. Furthermore, there should be only one kind of form. A system that calls for different forms for head injury and for knee injury would make things so complicated that it could not be easily managed.

It would have to be decided which cases were to get into the system There are various definitions of a reportable injury, based on number of days off, number of days not suited, etc. I think it would be better in the long run if all cases went in, rather than selected ones, although many would be minor injuries. I do not think we know whether serious injuries are worse copies of minor injuries or an entirely different breed. We might learn a good deal if we had data about the so-called minor injuries. There may be different families of accidents that could be detected that way.

I have argued for a simple document carrying a skeleton of information, and I would hold to that argument. But it would be essential that the form have room for additional data at the discretion of the team or some investigator who wants to turn on selected questions for limited periods. Hitchhiking extra data on a basic system is by far the easiest way to get it.

Coding has to be used for recording data. We do not have the money or technology yet to handle material in English narrative. A standard injury nomenclature has already been developed, and certainly much of the work toward coding is already done.

To get an injury data bank going would require the effort of a design team made up of a wide array of specialists: in sports medicine (both physicians and trainers), coaching, playing, systems analysis, data processing, terminology, and statistics.

Control

Once the system is running, there are critical "little" things that have to be planned for. One is keeping track of the system itself--the papers coming in and other details. It is easy to think that you are going to send a supply of forms out to teams and have them come back in; but you will find quickly that instruction manuals will have to be written. There will have to be someone at the receiving end to look at the forms and see that they are filled out legibly and correctly. Someone has to get on the telephone and ask what the originator meant. Some forms will have to be sent back because they contain only blank spaces. If Hoboken said that they sent in 200 forms, you have to be sure that you got 200. All these elements in data processing we call "control."

Liaison

Control is closely related to "liaison." You have to be ready when someone contributing data says: "I sent some wrong information; here is a correction. I hope it is in time to change the record." Liaison between the data center and the contributing units is not a technical concern, it is not a computer problem, and it is not concerned with wires. It is people talking to people, going out and instructing them, educating them, and keeping them happy and functioning, so that the system runs. I do not see any goals in the injury data-bank idea or needs that would justify a system involving high-speed electronic transmission of data. The U. S. mail would be perfectly adequate. It is a dependable transmission system of satisfactory speed.

Legal Problems

Legal problems would have to be worked out. Contractual arrangements between the data center and the participating teams would be needed. Contracts would concern such subjects as cost, what the teams send in, what they get out, and how confidential it must be kept.

Access

Research policies would have to be developed as to who could get at the data bank and how. This data bank would have more information in it than the teams could ever find time to use or think of questions for. It should be available to those who want to do research, and research policies would then be essential. Can noncontributors get at it or does one have to be a member of one of the participating teams? How do you finance service for researchers? How do you keep identities confidential? What policies govern publication? There will also be the problems of linking between the researcher and the data bank, negotiating costs of retrieval with him, telling him how the system is set up and what its limitations are, and so on.

Education of Users

A system like this creates another set of problems, which are not usually anticipated. Someone is soon going to say: "You are sending me a staggering amount of paper with numbers on it; how do I use it?" One may as well face at the start the necessity for educating the contributors and other users as to what these numbers mean and what conclusions can be drawn from them.

One thing that sometimes stops people from getting into a game like this is the wisecrack about computers: "GIGO"--"garbage in, garbage out." That is often used to mean: "Let's not do anything, because we do not have good information to work with." That is not the way to go at it. William Kincaid, Associate Director of our organization, says that the motto should not be GIGO but "GIFO"--"garbage in, fertilizer out." That emphasizes the key to making something like this go. You must take advantage of the fact that the data are not as good as the contributors would like. Be sure they find out that, if they had kept good records, if they had filled out the forms accurately, if they had used proper nomenclature, they could have the answer that they wanted. Start with the data the way they are and use the GIFO principle, and the data will be better each time around.

Usability

Now for two final points that are keys to making an information system like this work. First, remember that the most important person in the whole system is the contributor of the data--not the computer specialists or the statisticians. If you want to have good data, the system has to be doing something useful for him, something that he wants to have done. I do not see much future for any data system that is a one-way flow of information into Washington or Houston or wherever, just to satisfy someone in an ivory tower who wants to make statistics. Even if the most important long-range goal is research on football injuries, the system will fail unless primary concern is given to the contributors--trainers, owners, coaches, physicians, and players--so that they get regular and useful feedback of their own data from the system. For best results, this must be supplemented by comparisons to stimulate and challenge the data contributors. Second, in its input, an information system presents basically a clerical task. It is not a doctor's job, a trainer's job, or a coach's job to fill out reporting forms. It is a clerical job. One should plan the task that way and give it to clerical personnel; then the system will run.

Conclusion

Establishing a computerized data bank is not an insurmountable task. It could be designed and operated within a reasonable budget. And great benefits should accrue.

A STATISTICAL APPROACH TO THE PREDICTION OF KNEE INJURIES IN COLLEGE FOOTBALL PLAYERS

EWEN M. CLARK, GEORGE O. THOMASSON, and EARL BLEKKING

The purpose of this study is to inquire into the possibility of identifying football players with increased susceptibility to knee injury. A well-known coach of a professional team said that one of the things he liked most about a particular one of his players was that he did not get injured. Is there, in fact, a type of player who does not get injured? Can he be recognized? And, if so, can a probability scale be constructed to indicate the types of injury to which he is susceptible? With the aid of the computer and advanced statistical methods, many unexpected relationships have been found; for example, there is a close relationship between blood uric acid level and the desire to succeed in the academic field.[5] It seems not unreasonable to hypothesize that it would be possible to recognize the "noninjury" trait in athletes by physiologic and biochemical characteristics.

If the status of a player's knee were viewed as moving along a time continuum, constantly being subjected to and affected by the stresses, strains, and trauma of the game and practice situation, and if it were possible to recognize the factors responsible for the knee status at a particular point on the continuum and evaluate that status accurately, then, by projection, future changes in the knee status could be predicted. If in such an analysis one person's knee development differs widely from that of his peers, this surely must be accounted for by some dramatic change, probably an injury.

Consider this in another way. Assume that the variables--such as body configuration, position played, exposure to or frequency of playing, and physiologic and biochemical profiles--have been responsible for arriving at a current and unstable knee status, and that another person, with different values for the same variables, has a stable knee; then it is possible to classify all players using this set of variables into a degree of risk, and so be able to project a scale of probability of future injury to the knee.

Traditionally, the knee status is evaluated by examining its stability, as indicated by the integrity of the medial, lateral, and cruciate ligaments. If these ligaments are so weak as to render the joint

unstable, then it is correctly assumed that the person has an increased risk of injury. There are, however, many people for whom it is impossible to reach a firm conclusion on this basis, in that, although they are not entirely normal in this respect, neither are they classifiable as having an unstable joint.

The University of Florida Student Health Service performs a specific service for the Athletic Department in giving the athletes preseason physical examinations.[2] Data are collected in five broad classifications: (1) anthropometric, (2) physical, (3) biochemical, (4) exposure, and (5) medical. In each of these five main groups, we have a wide selection of variables that are measured. The anthropometric characteristics include height; weight; length of arm and thigh; circumference of arm, biceps, wrist, chest, waist, calf, ankle, and gluteus maximus; bicromial diameter; anterior/posterior diameter (bicromial); xiphoid diameter; umbilical diameter; thigh diameter; width of chest and iliac crest; and arm, scapular, chest, thorax, and abdomen skinfolds. The physical, or physiologic, variables are based on the electrocardiogram (P-R, QRS, and Q-T intervals), the spirometer (vital capacity, forced expiratory volume, and maximal breathing capacity), and hemoglobin measurement. The biochemical characteristics used include blood cholesterol, uric acid, sugar, and urea; spot test of blood for mononucleosis; urine sugar, albumin, and cells; and urine culture. The data collected from this examination were subjected to statistical investigation to see whether it were possible to differentiate between those with weak knees and those with normal knees, using measurements other than the knee examination itself.

As part of the preseason physical, we include an evaluation of the knee and classify each player into one of four groups, as follows:

Normal: On relaxation of the quadriceps and hamstring muscle groups, no movement is detectable within the joint capsule.

Questionable: On relaxation, slight movement is detected within the joint capsule, due to weakness of one or all of the medial, lateral, and cruciate ligaments.

Weak: On relaxation, sufficient movement is possible within the joint capsule that, on return to the normal position, the opposing bone surfaces could be detected knocking together.

Unstable: With both quadriceps and hamstring muscles in contraction, there is movement within the joint capsule.

This may seem a rather unsophisticated classification. However, previous experience in examining large numbers of high-school and university athletes has indicated that it affords a reasonable and desirable method of recording the results of the examination. Of the 87 athletes examined by these clinical criteria, 40 had normal knees, 16 had weak knees, and 31 had questionable knees.

We used statistical methods to determine which variables had the greatest discriminatory ability between players with clinically weak knees and players with clinically normal knees. The main statistical analysis in this program consists of multivariate discriminant-function analysis. This technique examines the variables that act as discriminators between two *a priori* groups. Basically, it calculates the F value at each stage in the program for each variable and then uses only those which are statistically significant.

If we take t measurements, x_1, x_2, . . ., x_1, on two samples of size N_1 and N_2 and the investigator seeks to determine whether the two samples are different on the basis of t measurements, one approach might be to consider each of the measured variates separately and to perform t ordinary t tests between the samples. This approach may be criticized as lacking efficiency in two respects: (1) it ignores the interrelationships among variables, and (2) it does not allow for an assessment of the relative power of each of the t variates in determining sample differences. The general problem as a discriminant function was developed by Fisher[4] and, in terms of a generalized distance function D^2, by Mahalanobis.[7]

We will assume that we have t measurements on two groups of individuals, of sample sizes N_1 and N_2. We seek some linear combination of the variables that will maximize the "between"-group difference relative to the "within"-group differences. The multivariate samples will then be reduced to the univariate case, and maximal distinction between the two groups will be afforded as their scale values are computed on the single-discriminant variate. It is conventional to test the significance of the difference between the two groups, which we can do by using the D^2 statistic to calculate the conventional F ratio with t and (N_1-N_2-t-1) degrees of freedom. If the F test is significant at a suitable level, it is reasonable to use prediction equations that will allow for the optimal assignment of sampling observations to group 1 or group 2. We use the discriminant weights for this purpose and develop a discriminant equation for group 1 and a second equation for group 2. In future observations with scores on the t variates, we would calculate the discriminant value using the discriminant weights applied to the observational values, and then assign the individual observation to group 1 or to group 2, depending on whether the calculated discriminant value is closer to the score for group 1 or group 2.

The discrimination afforded by that procedure is maximal with the variables in the immediate problem. The deletion or addition of any variable from the analysis would reduce or increase, respectively, the distance function and the consequent efficiency of the prediction equations. Various tests for the significant effects of deleting or adding variables in the analysis are given by Rao.[8] The calculated U statistic is used to calculate a corresponding F value, to test again the significance of the difference between the two groups.

The more general form of the discriminant problem arises when we have several (g) groups of individuals, events, things, etc., and we seek a determination of the differences among the groups on the basis of t measurements.*

Our results can be illustrated best by going through the various steps of the program, looking at the p value at each step. If we chart on a graph (Fig. 1), the p value against step number, we find a parabolic curve. It can be seen that the p value becomes highly significant at about step 10 and then loses its significance again at about step 24. Its maximal significance, when it has a value of less than 0.001, occurs somewhere between steps 14 and 20. It is at this point in the program that maximal separation between the two clinical groups will occur.

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* Various discriminant programs are available for the performance of these calculations.[3] A discriminant-analysis program is available as part of the IBM 360 scientific subroutine package, presented in programer's manual H20-0205-2, and as part of the BMD Library (Biomedical Computer Programs) developed by the Health Sciences Computing Facility, Department of Preventive Medicine and Public Health, School of Medicine, University of California, Los Angeles. The techniques used in this are discussed fully by Anderson.[1] Included in this library are programs BMD04M, Discriminant Analysis for Several Groups; BMD05M, Discriminant Analysis for Several Groups; and BMD07M, Stepwise Discriminant Analysis. Program BMD07M was used in the computations of the present study. In this program, in general, variables not yet entered into the discriminant equation are considered at each step or stage and the one with the largest F value is entered. The consequent calculations are based on the use of the variables found in each stage by this selection procedure. As the program passes through the stepwise discriminant stages, a moving picture of the action, influence, and interaction of each variable with respect to the discrimination function can be obtained. If the levels of significance obtained from the F values at each stage are graphed, we can obtain some evaluation of the optimal number of variables to consider in a discriminant analysis as applied to the specific problem. A chart of the ranking of variables at each stage, according to F value, is also useful for analysis. *A posteriori* probabilities for each individual observation can also be calculated, aiding in the analysis. Program BMD07M also permits the option of classifying additional observations into the most closely related group. The statistical relationships found should be viewed as being statistical, but can perhaps indicate areas for investigation to determine whether causal relationships may possibly exist.

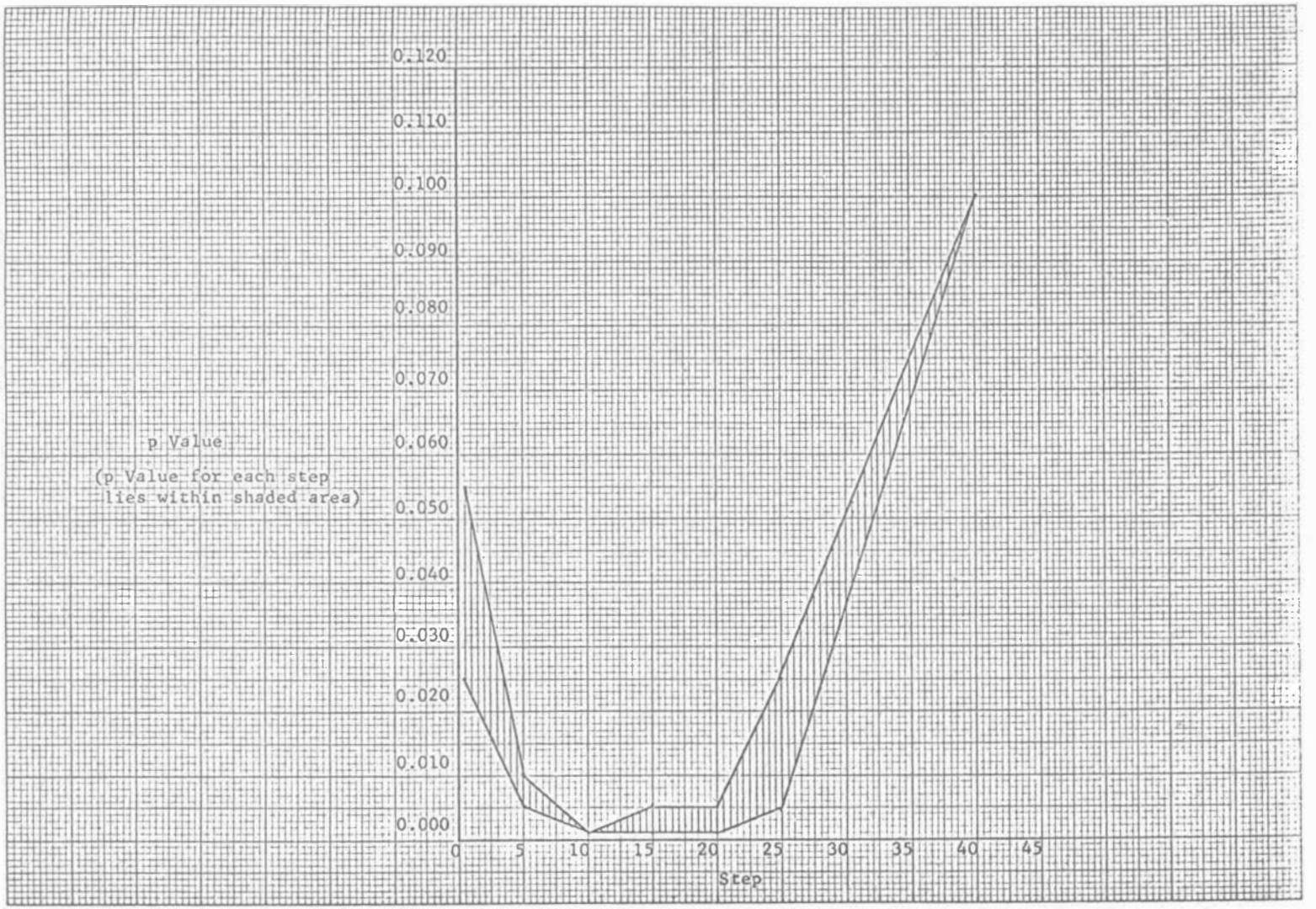

FIGURE 1
Significance (p value) of discrimination at each program step.

We should now look at the variables that are operating in this series of steps and categorize them in their order of F value as discriminators.

Figure 2 shows that variable 31 has a fairly high F value, and during the steps of greatest significance it reaches the number 1 position. Variables 49, 12, 55, 33, and 6 behave in the manner indicated on the graph. These variables are, respectively, abdominal skinfold, blood urea nitrogen, wrist circumference, ECG P-R interval, vital capacity, and age.

Table 1 shows that the direction of scalar movement of these variables will discriminate between normal and weak knees. Thus, a thin abdominal skinfold will predict a smaller probability of knee injury. According to the table, if you play football at the University of Florida, it is better to be thin, have low blood urea nitrogen, have a small wrist, have a long ECG P-R interval, have a low vital capacity, and be a freshman. A colleague has remarked: "Who's going to use such a small, undernourished player as that, anyway?" But it is necessary to bear in mind that these measurements are proportional to football players, and not to the general population. Also, the difference within each variable is small in itself. It is these factors in combination, not the individual measurements, that are important.

TABLE 1
Variables that optimize separation of group with weak knees from group with normal knees

	Variable	Normal	Weak
31	Abdominal skinfold	Thin	Thick
49	Blood urea nitrogen	Low	High
12	Wrist circumference	Small	Large
55	ECG P-R interval	Long	Short
33	Vital capacity	Low	High
6	Age	Low	High

It would be appropriate to speculate on why these particular variables are apparently such good discriminators.

Age:

Age is the least difficult to explain. In this context, age is a measure of exposure to risk. Players who were 18 years old had less trouble than 19-year-olds. Most freshman football players are 18. Perhaps when they try for the varsity team at the age of 19, this is the breaking point, both literally and metaphorically, for the knee ligaments.

Wrist Circumference:

At first, wrist circumference seems a paradoxic finding, but on closer inspection perhaps it is not. Two factors must be considered. Wrist circumference is the only measurement taken that reflects only bone structure, indicating that thinner-boned players are safer. We could find no reference in the literature to any work that indicates that larger-boned people have a proportional increase in ligamentous tissue; so they might have less support of this kind than they require. Another consideration is that a large joint surface rotating around the instant center would increase the leverage exerted at the circumference of the rotation, and so allow ligaments to be torn more easily. It has also been suggested by an anatomist* that, with a smaller weightbearing area, there will be a much increased load per unit area during weightbearing, which might contribute substantially to the instability of the joint.

Abdominal Skinfold:

At the University of Florida, skinfold measurements are used in association with the Ponderal Index as an index of athletes' conditioning status. Contrary to other workers, we have found that the abdominal skinfold site correlates[6] very closely with the percentage of lean body mass--much better than any other site, including the currently acceptable triceps area. It seems generally agreed that a well-conditioned and fit athlete is less susceptible to injury than others.

Blood Urea Nitrogen:

There is evidence that heat stroke is accompanied by a rise in the blood urea nitrogen level.[9] For this to reach pathologic levels, severe heat stroke has to be present, but minor variation may occur in those who are poorly acclimatized to heat. This can be a major problem in Florida, where the football season starts with temperatures in the upper 90's. We have shown that there is an increased incidence of injury in poorly heat-acclimatized athletes.

Vital Capacity:

It is difficult to hypothesize why vital capacity should be of importance, but there is evidence that pulmonary function is a very powerful discriminant in the characteristics of players of different positions. In a parallel study in which we were able to have the computer classify, with remarkable accuracy, the playing position of players, three of 15 of the variables were related to pulmonary function. One may postulate that this is a reflection of the undesirability of a player's being used in a particular position, i.e., one for which he is physiologically unsuitable and therefore at greater risk of injury.

* J. J. Bernstein.

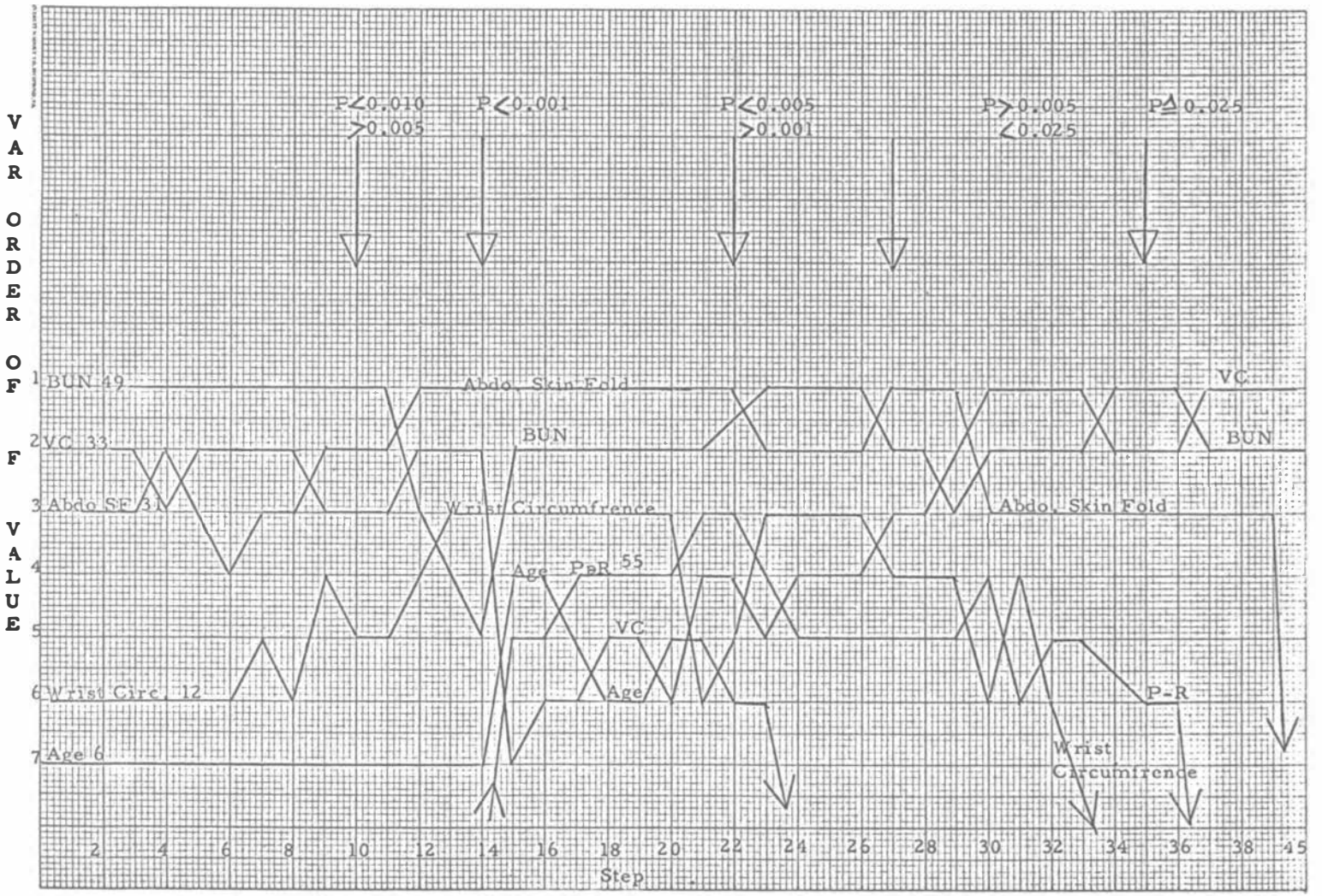

FIGURE 2
Schematic representation of changing relative importance of significant discriminating variables. These changes are caused by new variables entered into program at each step.

ECG P-R Interval:

The P-R interval is normally found to be higher in healthy subjects. It is also increased in those who are heavy and older and in those with lower heart rates. Bradycardia is a characteristic finding in well-conditioned athletes.

The difference in injury rates in those groups indicated that our technique can be used to supplement clinical knee evaluations.

Figure 3 summarizes the injury rates in both the clinical and computer-classified groups. The injury rate in the clinically normal knees was 15%, and in the clinically weak knees, 50%. Of players of the clinically questionable group classified by the computer as normal, 21% were injured, and of those classified by the computer as weak, 42% were injured. The chi square test applied to these groups gives a p value of less than 0.03. Although this is not a perfect result, it does indicate that the computer can separate the clinically questionable group into high- and low-risk groups with some accuracy. This indicates that the technique is of considerable value as a discriminator in this context, but that further refinement could increase the accuracy.

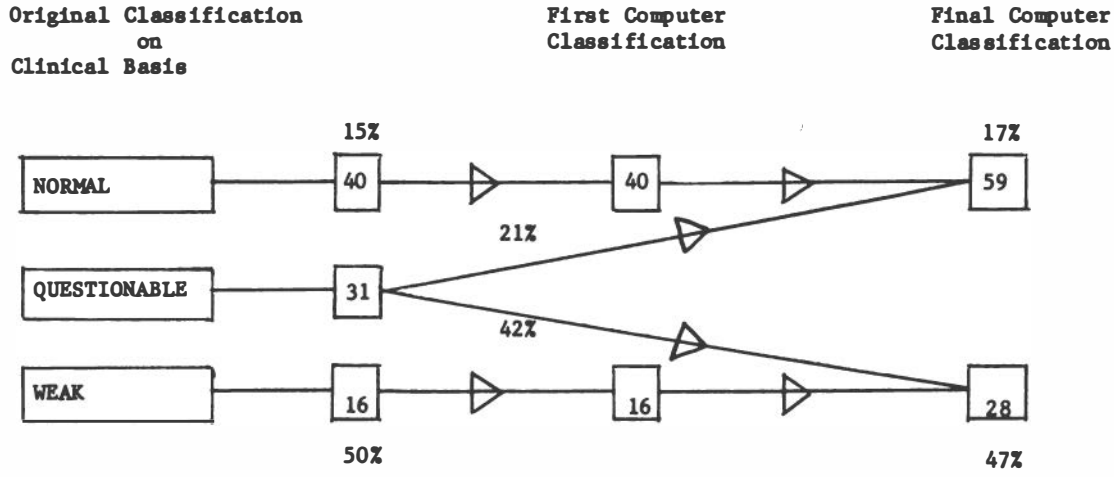

FIGURE 3
Summary of computer program classification. In blocks, numbers in each group; percentages are of knee injuries in each group.

There is need for further study in this field, with the inclusion of other variables, and for a larger study of this technique with a wider age group, from the high-school to the professional level. As a greater volume of information becomes available, a more accurate system can be designed, which will be of value to coaches, trainers, and the players themselves.

In summary, it is possible to improve our prediction of which people are at considerably higher risk of knee injury. The results of this program can be of practical value if they lead to the institution of specific conditioning programs for strengthening the muscles and ligaments that support the knee, general fitness conditioning, and greater attention to heat acclimatization. The team physician treating the relatively minor knee injury of a player known to be at high risk could protect him from playing for those extra 2 or 3 days, which may indeed be critical to his future.

This study can serve as a pilot study. In future studies, the following are indicated:

(1) larger samples,

(2) diversity of sample sources,

(3) refined measurements to approach better or more distributions,

(4) comparison over long periods or long-term follow-up studies, and

(5) the inclusion of variables in other psychologic and physiologic areas.

Applications of factor analysis to determine the areas of greatest influence and to assist in the refinement of test instruments are also indicated. It is hoped that this would result in the eventual optimization of a test instrument with the characteristics of brevity, economy, simplicity, and accuracy.

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CONCLUDING REMARKS

FRED C. REYNOLDS

I am not going to try to summarize everything in the meeting. But I do want to say that, in planning this program, we tried to bring together knowledgeable people in the field and have them discuss some of the problems related to athletic endeavor, in the hope of establishing a baseline of known facts and indications, learning where the deficit in knowledge is, and stimulating continued research in an effort to avoid athletic injuries. I believe that this workshop has satisfied our objectives.

But if we stop here, we really will not have accomplished the larger task. This workshop should be the opening. I would hope that professional football would be willing to make another grant--perhaps even a yearly grant--to allow meetings like this to be held in the future. Perhaps in future meetings, it would be well to limit discussion to particular fields. If there is sufficient interest in continuing this type of program, and if the football people feel that they are not in a position to make continued grants for conferences, perhaps we might interest some other group.

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