

INJURY BIOMECHANICS RESEARCH
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BASEBALL RELATED CHEST IMPACT

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INTRODUCTION

A study conducted by the Consumer Product Safety Commission (CPSC) in 1981 on sports-related injuries to children between the ages of 5 and 14 years indicated that there had been more baseball-related fatalities in the 5-14 year old age group than for any other sport. It also indicated that there were more deaths from chest impacts with the ball than had been generally known. As a result, a follow-up study was done in 1984 on baseball related injuries and fatalities to children.

A summary of the findings showed that during the 1973-1983 period, 51 baseball-related deaths to children were reported to the CPSC. The most frequent type (23 cases) of baseball-related fatality was the result of chest impact from the ball. Of those fatalities, 11 occurred during organized games and the remainder in unorganized recreational play. These findings resulted in a compilation of all available documentation on each case, including police reports, autopsy reports and interviews with family or other individuals present at the time of the accident.

Based upon analysis of available data it appears that batters (primarily those who turn to bunt (25%) and pitchers (33%) are the most frequent victims

of chest impact deaths to children 5-14 years in organized play. This raises the question of whether some form of chest protection for batters and pitchers of organized league games would help to reduce such unnecessary deaths.

An examination of autopsy reports shows the incidence of fatalities occurring in children aged 5-14 without any apparent pre-existing heart disease or dysfunction. This presents a situation that requires further study in the hope of reducing the risk of fatal injury to young children.

MECHANISM OF DEATH AND HUMAN TOLERANCE CRITERIA

A literature review focusing on the physiological components of sudden cardiac death formed the basis for the investigation of the possible causes for this phenomenon.

Sudden death was attributed to cardiac concussion (commotio cordis) in the absence of actual rupture. This term was coined by Schlomka and Hinrichs (1932) to describe ventricular fibrillation resulting from direct pre-cordial trauma. Ventricular fibrillation describes a rapid chaotic asynchronous contracting or twitching of the heart muscle instead of its normal regular rhythm. It has frequently been found to be irreversible and consequently fatal. Cardiac standstill also cannot be excluded as a possible cause. In addition, blunt chest impact can initiate a series of arrhythmias (Lau, 1984 a & b) and physiological responses responsible for death.

In an excellent review of sudden cardiac death, Lown (1979) stated that the heart responds as a mechano-electrical transducer which converts electrical impulses into mechanical energy. Scherf and Bornemann (1960) have demonstrated the opposite effect showing the effectiveness of mechanical thumping (pre-cordial thump) to resuscitate the heart in ventricular standstill. The effect of sternal impact by a baseball, however, appears to be capable of producing the same type of dysfunction which disrupts normal cardiac pacemaker activity, that is, causing cardiac standstill or fibrillation.

The identification of critical factors in this sequence of events was a careful analysis of the 24 cases of baseball fatalities (including one lacrosse player fatality with a similar etiology) investigated by the CPSC. Other cases of non-penetrating chest injuries or non-baseball sports injuries (such as football and karate) were also included in our analysis because of common factors which appear to be relevant to the cause of death.

Possible Mechanisms

1. Mechanical impact to the myocardial conducting system may cause a shift in the ionic gradients and possible loss of cellular conductivity which are necessary for the heart to maintain a regular rhythm. The immediate effect may be an irreversible cardiac arrhythmia.
2. Impact of the myocardium can injure the contractile elements of the heart muscle either impairing contraction or initiating a beat that has its origin in some abnormal focus. Multiple occurrences of this type of cardiac activity can result in insufficient blood supply reaching the brain and death.
3. Damage to the heart muscle from impact can cause ventricular tachycardia due to reentry. Reentry refers to a premature impulse to an area of the heart muscle that has recently been activated and is not ready for reactivation. It has been shown that reentry may be a precursor to ventricular fibrillation.
4. Ischemia or a local reduction in blood flow from a mechanical impact can result in an accumulation of metabolic wastes and a simultaneous lack of oxygen. Ventricular arrhythmias can be precipitated by this sequence of events.
5. The autonomic nervous system and the hormone system can influence the inception of cardiac arrhythmias. Sympathetic and parasympathetic response may be anticipated as a response to a mechanical impact as a

simulated fright reflex. Sympathetic stimulation is believed to facilitate arrhythmogenesis.

6. Cardiac standstill as a result of the failure of the heart muscle to initiate an impulse may be caused by a severe blow to the chest. This sequence of events could occur if the impact resulted in a complete coronary artery occlusion.

Physiology

After an exhaustive review of all of the above mechanisms, the conclusion was reached that there is no one single physiological event that can be pinpointed as the key to sudden cardiac death under these circumstances. It was determined that a severe blunt impact to the upper torso could result in disruption of conduction in the heart causing cardiac arrhythmia followed by ventricular fibrillation or ventricular standstill.

The possibility that there is a vulnerable period in the cardiac cycle which would cause an impact to be more lethal than at another time was debated at length and generally agreed to be a possibility that could not be overlooked but it would be difficult to substantiate. Such an occurrence in the cardiac chain of events would be impossible to determine clinically and no evidence has been found experimentally that would support that hypothesis. Vagal overstimulation causing cardiac standstill and heart failure was proposed as a possible mechanism. There is substantial evidence in the literature to support many possible mechanisms for fatality due to a blunt chest impact. However, the precise mechanism in each case cannot be determined. All of the cases studied as well as those in the literature have striking similarities. 1) Evidence of pre-existing cardiac abnormalities was generally not present. 2) Damage to myocardial tissue from the impact was also absent or limited to very superficial contusions to the pericardium so that a fatal outcome would not be anticipated. 3) In addition, there are a

number of variables, particularly with respect to the location of the impact which certainly might suggest that no one single mechanism can be responsible.

Of the 24 CPSC case studies of fatalities, autopsies were performed in 18 cases. A number of factors stood out as being significant. In most cases, impacts were to the anterior chest wall. However, several impacts were to other areas of the torso, including the back and epigastrium indicating that a direct blow over the heart or sternum was not necessary to induce cardiac arrhythmia.

Administration of CPR

It was noted that in 13 of the CPSC cases, administration of CPR was immediate. Only one child was successfully resuscitated but he only survived for three days. In the remaining cases the sequence of events relating to the administration of CPR was not adequately documented. In any event, it was indicated by the cardiological consultants that the heart of a healthy individual in standstill should not be too difficult to restart with adequate CPR. However, one in fibrillation might not allow adequate induced pumping while in chaotic contraction and thus CPR might not be effective. It was concluded that the likelihood was high that at least some of the CPR attempts were adequate and timely but successful resuscitation was medically impossible.

The ultimate conclusion reached concerning the sudden death phenomenon in these cases was that although a precise mechanism could not be isolated the problem should be approached in terms of prevention and protection.

Human Tolerance to Direct Chest Impact

Although several possible mechanisms have been proposed, there is still a need to document the level of human tolerance to blunt chest impact. An injury criterion for adults have been proposed by Viano and Lau (1985) for frontal impacts to the chest by a 6-in diameter pendulum. It is based

on the percent compression of the chest (C) as well as on the velocity of the chest (V) as it responds to the impact. In fact, it is the maximum value of the product of V and C and is known as the V*C criterion or the viscous criterion. The proposed limit of V*C for a severe injury to the chest is a value between 1.3 to 1.8. This value appears to be valid for adults of all sizes, ranging from a 5th percentile female to a 95th percentile male. An assumption can therefore be made that the criterion for chest injury in children between the ages of 4 and 16 is the same. However, for this particular application of baseball impact, it is necessary to express this viscous criterion in terms of ball velocity and age of the child. However, injury data based on these parameters are not available. The next best alternative is to determine the response of the chest for children between the ages of 4 and 16 using a mathematical model proposed by Lobdell et al (1973). This model utilizes a set of springs and dampers to represent the rib cage and two masses to simulate the chest wall and the thoracic contents. Lobdell et al (1973) obtained numerical values for these parameters for the adult thorax and validated their model results against experimental data. For children, these values had to be scaled down based on available anthropometric data of children in that age range. Such data were taken from Snyder et al (1975). The method used to scale the parameters was to assume that the chest was a ring which is subjected to equal and opposite loads along a diameter of the ring. Using the newly determined values for a child's chest, the model was exercised to obtain chest response for different ball velocities ranging from 22 to 110 mph (10 to 50 m/s) and for children between the ages of 4 and 16. Model results include chest compression, chest velocity and sternal acceleration. From these results, the viscous criterion V*C was computed for children as a function of age and ball velocity. Figure 1 shows the tolerance band for

severe injury to the chest for children, based on the Lobdell model. The range of ball velocity is 66 to 110 mph (30 to 50 m/s).

It should be noted that the tolerance for injury proposed in Figure 2 is based on the results of a mathematical model and that experimental values are not available since it has not been possible to perform tests on human surrogates that are representative of children in this age range. However, the range of ball velocity appears to be realistic and there is an increase in velocity needed to cause injury to an older child. Experimental validation of this injury criterion is needed before it can be used as a standard for assessing chest injury due to baseball impact.

METHODOLOGY FOR EVALUATING CHEST PROTECTORS

Biomechanics

The model described above provided an insight regarding the response of the chest to blunt impact by a baseball. When the sternum is hit by such a projectile, only a small amount of the chest wall is set in motion by the impact. The amount of mass involved is of the same order of magnitude as that of the baseball (5 oz or 0.15 kg). Thus, the heart is slapped by the inner wall of the sternum at approximately 50% of the speed of the ball, resulting in ventricular standstill. To protect the heart, it is necessary to increase the effective mass of the chest wall which will in turn decrease the sternal velocity. The use of pads and other forms of protectors is a practical means of increasing this effective mass. By distributing the force of impact over a larger area of the chest, more of the chest wall is brought into play during impact, thus increasing the effective mass of the chest or sternum. A six year old child dummy was used as a test device to evaluate these protectors. The fact that the methodology needs to be both repeatable and reproducible mandates the use of a mechanical surrogate for children.

One of the aims of this study was to develop a test methodology for evaluating the protective capabilities of various chest protectors designed for baseball or hockey. The criterion used to determine their effectiveness was the increase in effective sternal mass as a result of the use of such protectors. However, some of the protective devices do not have a readily identifiable mass which can be assumed to participate in the impact. Catcher's vests used in baseball and goalie's vests used in hockey are examples of protectors for which it is not possible to estimate the amount of the vest participating in the impact. An alternate criterion is sternal acceleration as measured on the dummy chest. It is a measure of sternal motion and if the protective device can lower sternal acceleration significantly, it can be considered as being effective.

Test Methodology Runs

A series of tests at velocities of 50, 60 and 70 mph was carried out initially to simulate several conditions of impact expected in Little League type games. This speed range was thought to be reasonable since fast balls in major leagues were clocked at 90 to 105 mph. The dummy and the sternal accelerometer sustained damage at 70 mph and the speeds were reduced to 40, 50 and 60 mph. The principal criterion for a reliable test methodology was repeatability. That is, peak sternal and A-P spinal accelerations for three runs made at the same ball velocity should not differ by more than 10% from each other. The impact location was critical because the accelerometer was mounted on an aluminum bar behind the leather sternum and the peak acceleration was sensitive to changes in the point of impact.

Impact Tests on the Unprotected Chest

A series of these tests were made at each of the three ball velocities, namely, 40, 50 and 60 mph. These data were considered to be a set of baseline data to be used for comparison with data acquired using a variety of

chest protectors. The peak values are tabulated in Table 1 which also contains data from runs in which the chest was protected by some form of padding. There were a total of 13 impacts to the unprotected chest at the three baseline velocities.

Impact Test Using Chest Protectors

The chest protectors selected for this program were:

1. A standard catcher's chest protector worn by a child who died of cardiac arrhythmia during a baseball game.
2. A chest protector worn by goal tenders in an ice hockey game.
3. A specially designed sternum pad supplied to Wayne State University by Mr. Ray Sharpe of Thoradom, Inc.
4. Another specially designed breast protector of female baseball players supplied to Wayne State University by Mr. Byron Donzis of Medidyne, Inc. The vest is shown.
5. A plastic torso protector in the form of a plastic sheet, approximately 14 mm (0.55 in) thick. It was made by Thoradom, Inc.

The standard catcher's vest (Pad #1) provided protection in the form of horizontal ribs of light plastic foam stitched onto the vest. Its total weight was 397 gm (14 oz). The hockey goal tender's vest (Pad #2) was of a similar design but the foam material appeared to be slightly thicker and heavier. Its total weight was 340 gm (12 oz). The sternum pad (#3) above was designed to be worn with a special T-shirt which contained a pocket in the front of the sternum to hold the pad in place. It weighed approximately 67 gm (2 oz), was 150 mm (6 in) in diameter and 17 mm (0.7 in) thick. The Donzis cup (Pad #4) was made of a rigid plastic material and was designed to protect the breasts of female ball players. There were two cups sewn into each T-shirt. They were about 190 mm (7.5 in) wide, 127 mm (5 in) high and 38 mm (1.5 in) deep.

The plastic torso protector (Pad #5) was a sheet of light weight polyethylene foam weighing 313 gm (11 oz). It was 750 mm (30 in) long and 230 mm (9 in) high. It was designed to be worn around the chest for protection of the entire upper torso.

The test procedure adopted called for a series of impacts at the three nominal ball velocities of 40, 50 and 60 mph. There were at least two consecutive impacts against each protective pad or vest followed by or preceded by a pair of impacts against the unprotected sternum. The instrumentation used was identical to that described for the acquisition of baseline data.

Test Results

The test results are summarized in Figure 3 which is a plot of sternal acceleration against ball velocity for the unprotected chest (Pad #0) and for the 5 pads used in this study (#1 - 5). It can be seen from this figure that the unprotected chest sustained the highest sternal acceleration at each of the ball velocities tested. The catcher's vest and the goalie's vest were minimally effective in reducing sternal acceleration while there was a significant reduction in acceleration of the sternum when the Donzis cup (Pad #4) or the Sharpe pad (Pad #3) were used. The Thoradom vest (Pad #5) afforded only minimal protection. It was possible to estimate the net increase in effective mass of the sternum for Pad #3 and 4 because their mass could be determined accurately. Figure 4 shows the net increase in effective mass of the sternum as a result of the use of Pad #3. The increase was almost 300 gm (twice the mass of the baseball) at 40 mph but it dropped rapidly with increasing ball velocity to about 30 gm (20% of the mass of the baseball) at 60 mph. The Donzis cup (Pad #4) was less effective at 40 mph but its protective ability did not drop as sharply as the Sharpe pad, as shown in Figure 5.

CONCLUSIONS

Case reports describing the incidence of sudden death in children due to an impact to the chest by a baseball resulted in the initiation of a literature search and case study to determine the cause of such occurrences and possible preventive measures that might be taken. It was found that no one physiological event could be identified as the sole mechanism of death. However, numerous factors initiated by the impact were found to lead to ventricular fibrillation which is frequently fatal.

Human tolerance to chest impact can be expressed in terms of a viscous injury criterion. Injury threshold values were determined for children based on animal and cadaver study data scaled down for a child's chest. From this data a test methodology was developed to evaluate chest protectors for their ability to reduce forces to the chest to an acceptable level based on the size of the child. The protection afforded by various pads and vests can be attributed to the added mass of the pad and the added mass of the chest wall involved in the impact due to load spreading by the pad or vest. However, the increase in effective sternal mass drops off with increasing ball velocity for all pads tested. There is a need to evaluate the performance of these and other pads at higher ball velocities.

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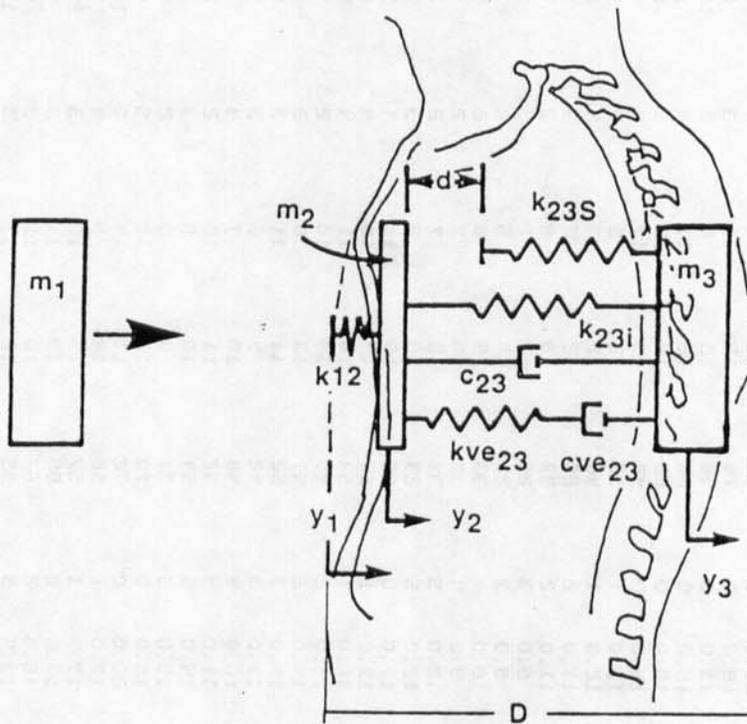
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TABLE 1

CPSC CONTRACT NO. CPSC-P-84-1170 BASEBALL IMPACT DATA TABULATION OF PEAK DATA 05/23/85

GRP	BALL	VELOCITY (MPH)	A-P (+)	P-A (-)	ACCEL (G)	CH. VEL. (m/s)	CH. DEFL. (mm)	PAD NO.	A-P (+)	P-A (-)	S-I (+)	I-S (-)	L-R (+)	R-L (-)
36	1	37.9	1800	1200	1200	10.0	13.0	0	48	9	10	3	12	8
37	2	46.9	2000	1500	1500	11.0	15.5	0	63	16	10	3	15	6
38	3	56.3	2300	1600	1600	12.0	18.0	0	73	18	15	3	18	11
39	1	39.0	1800	1300	1300	6.0	13.0	0	46	10	8	3	14	6
40	2	47.0	2000	1400	1400	10.0	15.0	0	60	14	9	2	15	9
41	3	56.2	2200	1700	1700	12.0	18.0	0	74	18	11	3	16	7
42	1	40.6	1900	1200	1200	10.0	13.0	0	48	12	8	3	11	8
43	2	47.8	2000	1500	1500	11.0	16.0	0	60	15	11	4	16	8
44	3	58.5	1950	1500	1500	6.5	18.0	0	68	18	10	5	16	7
45	1	40.9	1700	1300	1300	9.0	14.0	0	42	9	11	4	14	4
46	1	40.9	1200	900	900	8.0	13.0	1	36	12	9	4	14	4
47	1	40.9	1300	1000	1000	8.0	12.0	1	40	12	9	4	16	4
48	1	40.8	800	640	640	7.0	11.0	2	29	12	8	3	6	4
49	1	41.1	1000	800	800	7.0	11.0	2	33	9	8	2	5	3
50	1	41.1	750	520	520	4.0	9.0	3	28	8	7	2	4	2
51	1	40.5	690	400	400	4.0	8.0	3	27	8	6	3	3	3
52	1	40.5	920	600	600	5.0	8.0	4	33	6	6	2	3	3
53	1	40.6	850	520	520	5.0	8.0	4	35	8	7	2	4	3
54	1	42.0	1050	950	950	8.0	12.0	5	34	9	7	2	4	2
55	1	42.4	950	750	750	6.0	12.0	5	29	9	8	2	2	3
56	2	49.9	2200	1600	1600	12.0	17.0	0	65	16	11	4	2	2
57	2	49.9	1800	1300	1300	10.0	13.0	1	54	14	10	4	3	3
58	2	49.9	1800	1300	1300	11.0	16.0	1	54	16	11	4	6	5
59	2	50.5	1200	900	900	8.0	14.0	2	40	14	10	2	5	3
60	2	49.4	1300	1000	1000	9.5	14.0	2	47	13	8	3	4	4
61	2	50.1	1100	850	850	7.0	11.0	3	52	11	11	3	8	4
62	2	49.9	950	750	750	7.0	11.0	3	42	9	6	3	7	4
63	2	49.7	1000	800	800	7.0	10.0	4	48	10	7	4	7	4
64	2	49.6	950	700	700	6.0	10.0	4	42	11	10	2	6	3
65	2	49.4	1450	1050	1050	8.0	14.0	5	44	10	9	3	6	4
66	2	49.4	1600	1200	1200	10.0	15.0	5	51	10	8	2	4	7
67	3	56.6	1900	1800	1800	10.0	19.0	0	70	17	12	5	8	6
68	3	57.4	2500	1900	1900	13.0	20.0	0	72	17	12	5	8	6
69	3	56.9	2000	1300	1300	11.0	17.0	1	58	14	14	6	10	8
70	3	58.0	1900	1300	1300	10.0	17.5	1	57	13	10	4	5	4
71	3	57.3	2000	1200	1200	11.0	17.0	2	59	18	11	3	24	13
72	3	58.3	1800	1200	1200	11.0	16.5	2	67	10	11	3	5	5
73	3	58.0	1200	900	900	8.0	13.5	3	55	12	11	3	6	14
74	3	58.1	1100	800	800	7.5	13.0	3	54	12	12	4	13	6
75	3	58.3	1000	800	800	6.5	12.0	4	50	7	11	2	8	5
76	3	57.2	1200	900	900	8.0	13.0	4	50	9	11	3	9	3
77	3	57.2	1800	1100	1100	10.5	17.0	5	54	11	12	3	11	7
78	3	58.0	1500	1000	1000	9.5	16.5	5	50	11	10	4	7	5

GRP. NO. - BALL VELOCITY GROUP - 40, 50 OR 60 MPH
 PAD NO. - TYPE OF PADDING USED
 A-P - FROM ANTERIOR TO POSTERIOR
 S-I - FROM SUPERIOR TO INFERIOR
 L-R - FROM LEFT TO RIGHT
 PAD NO. 1 - CATCHER'S VEST (FATALITY)
 PAD NO. 2 - HOCKEY GOALIE'S VEST (GBP/UR)
 PAD NO. 3 - SHARPE SHIRT INSERT (ROUND PAD)
 PAD NO. 4 - DONZIS FEMALE PROTECTOR (PLASTIC CUP)
 PAD NO. 5 - THORADOM TORSO PROTECTOR (GREEN SHEET)



$m_2 = 0.45 \text{ kg}$	$k_{12} = 281 \text{ kN/m}$	$c_{23} = 0.52 \text{ kN-s/m, compression}$
$m_3 = 27.20 \text{ kg}$	$k_{23i} = 26.3 \text{ kN/m}$	$c_{23} = 1.23 \text{ kN-s/m, extension}$
	$k_{23S} = 52.6 \text{ kN/m, } d = 38.1 \text{ mm}$	$c_{ve23} = 0.18 \text{ kN-s/m}$
	$k_{ve23} = 13.2 \text{ kN/m}$	

Figure 1 Lumped-mass representation of the chest structure with parameter values for the adult male (Taken from Viano and Lau, 1985 - may be found in Appendix E)

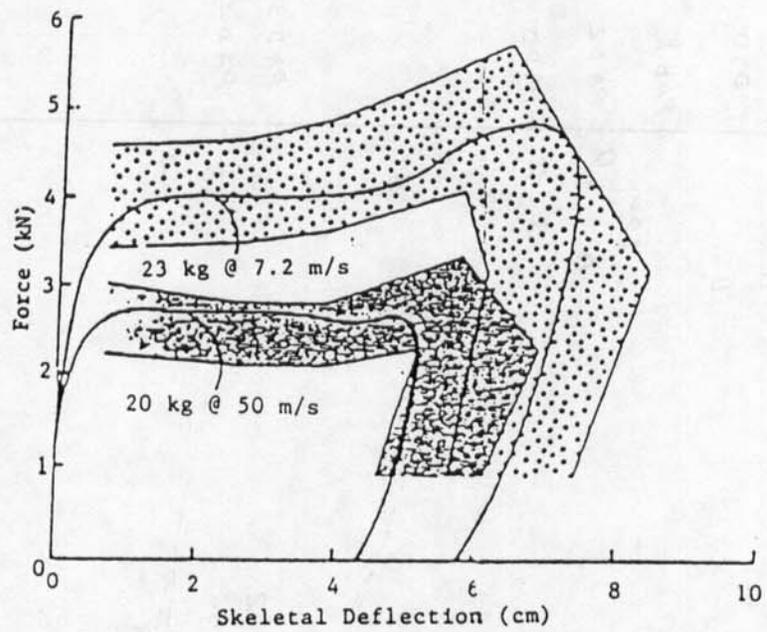
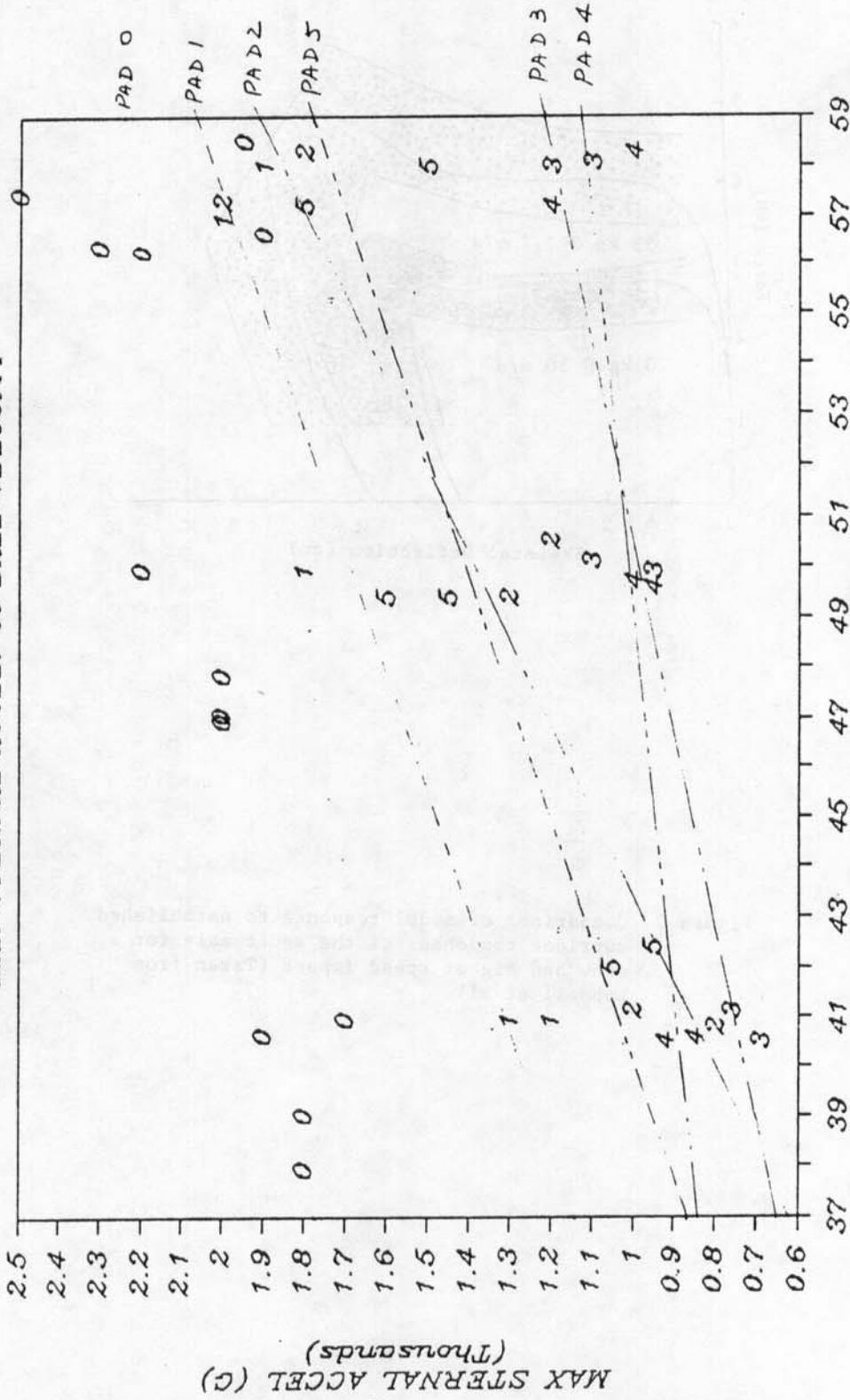


Figure 2 Comparison of model response to established corridor responses of the adult male for a slow and higher speed impact (Taken from Lobdell et al)

CPSC BASEBALL IMPACT DATA

MAX STERNAL ACCEL. vs BALL VELOCITY

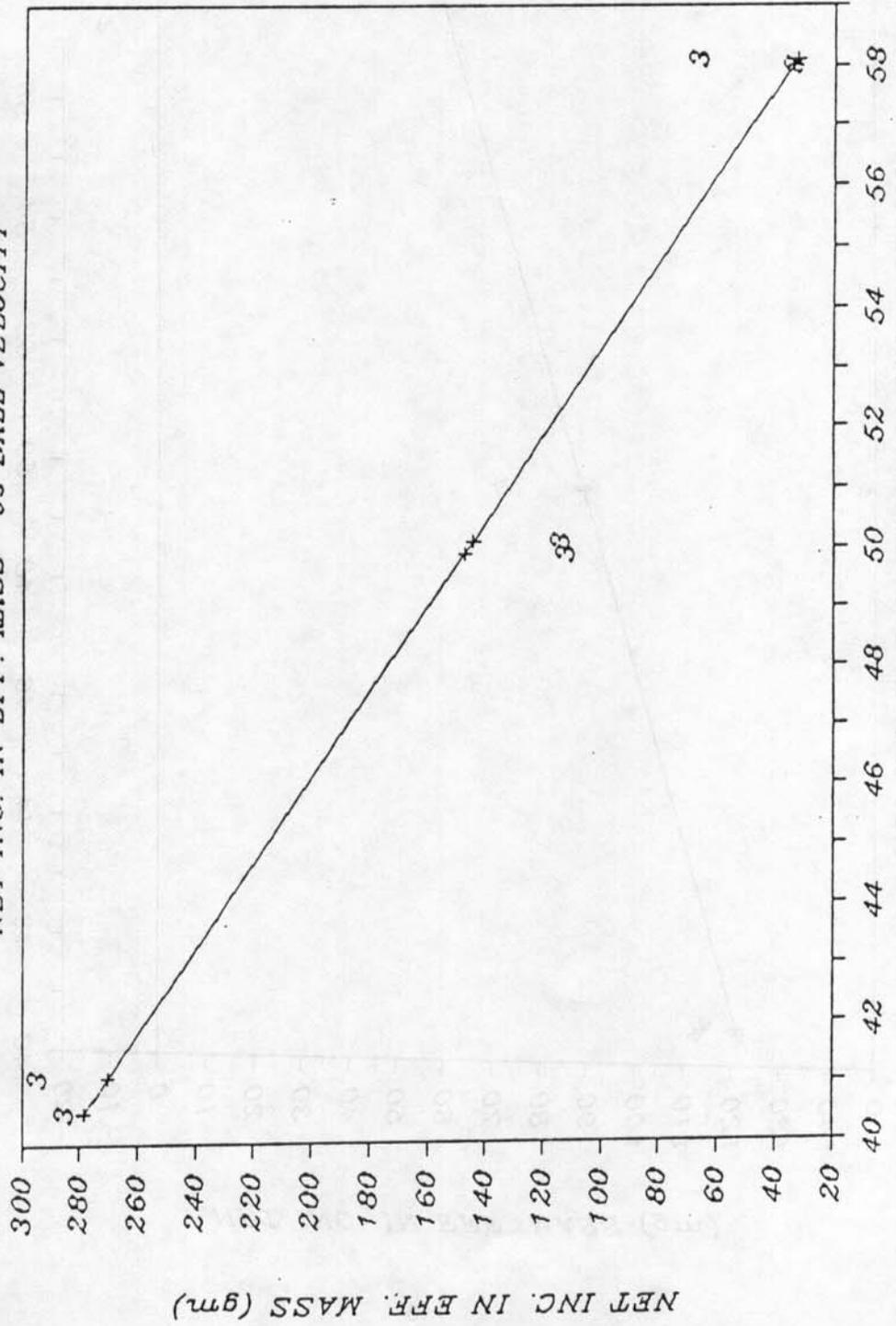


MAX BALL VELOCITY (mph)
 — 05/23/85

Figure 3 Summary of test results with sternal acceleration plotted against ball velocity.

CPSC BASEBALL IMPACT DATA

NET INC. IN EFF. MASS vs BALL VELOCITY



BALL VELOCITY (mph)
+
LINEAR REGRESSION

— 06/03/85

Figure 4 Net increase in effective mass of the sternum as a result of the use of Pad #3.

CPSC BASEBALL IMPACT DATA

NET INC. IN EFF. MASS vs BALL VELOCITY

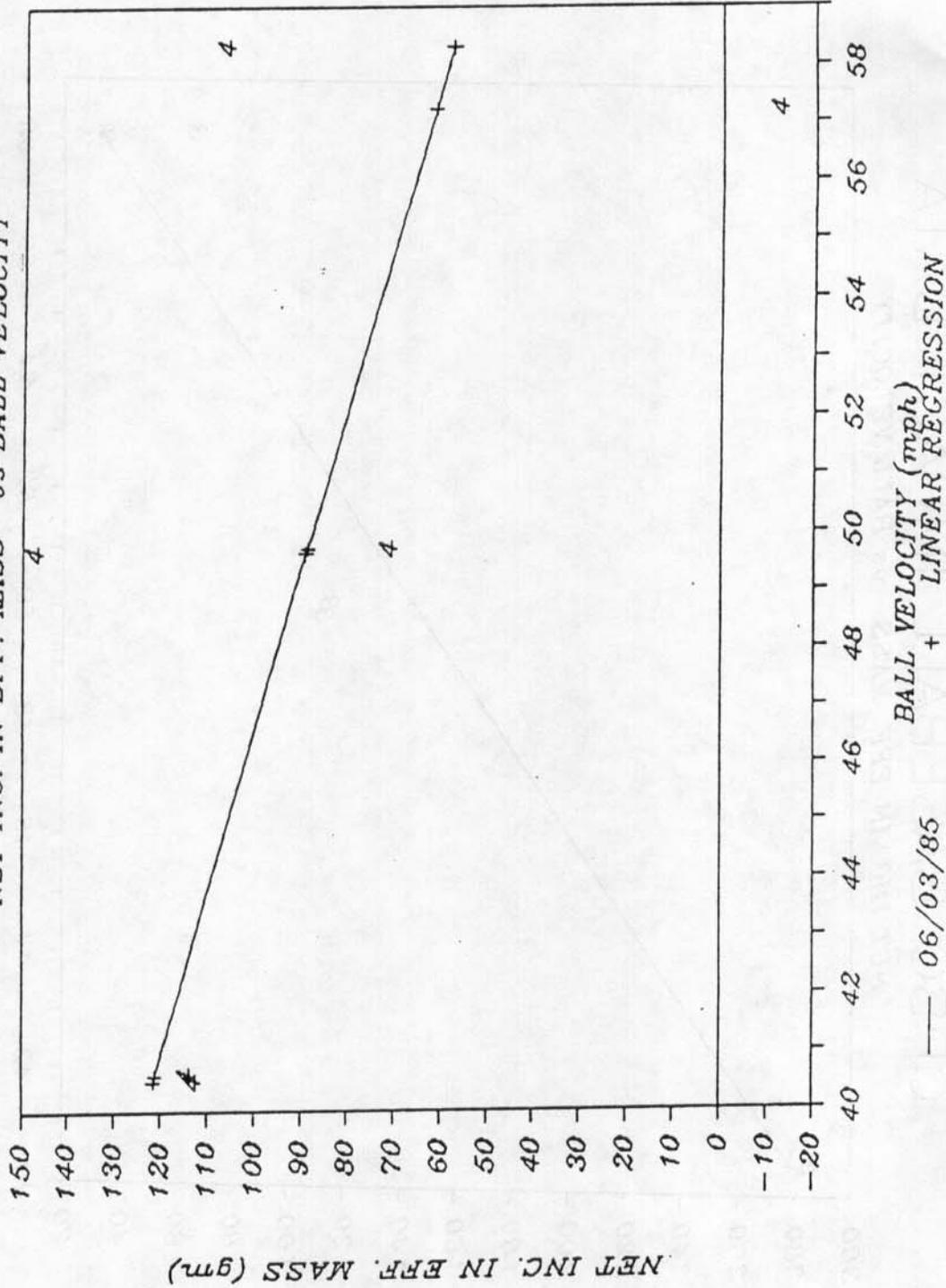


Figure 5 Effective mass of the sternum with the use of the Donzis cup.

06/03/85 BALL VELOCITY (mph) + LINEAR REGRESSION