PREDICTION OF CHEST WALL DISPLACEMENT AND HEART INJURY FROM IMPACT CHARACTERISTICS OF A NON-PENETRATING PROJECTILE

by

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ABSTRACT

Sternal injury, gross cardiac pathology and cardiac dysrhythmias following non-penetrating impact by a variety of impactors to the sternum of experimental animals are described. The biomechanical response of the chest wall to the impact are presented and a correlation between injury severity and chest wall displacement is demonstrated. A simple mathematical model is developed which predicts chest wall displacement when the mass, velocity and dimensions of the impactor are known. The model demonstrates the dependence of chest wall displacement on pre-impact kinetic energy, impactor diameter and target size.

INTRODUCTION

Thoracic injuries occupy a position second only to head injuries as a leading cause of death due to trauma. Clare et al (Ref 1) reviewed the available literature on experimental non-penetrating trauma to the chest by low mass, high velocity projectiles (15-150 $m.s^{-1}$) and were able to provide an estimate of the probability of death as a function of mass, velocity, effective diameter of the projectile and the weight and chest wall thickness of the animal. The lungs occupy the greatest proportion of the thoracic cage and damage to this organ was the main cause of death in the animals used in the literature that they reviewed.

The heart has received less attention in the experimental studies of non-penetrating injury. The high risk of death associated with cardiac injury is obvious and damage to the heart may involve gross or microscopic damage to the cardiac muscle with associated changes in the ability of the damaged area to conduct or initiate the inherently generated electrical signals responsible for co-ordinating contraction. It is possible to produce myocardial conducting system dysfunctions in the absence of obvious tissue destruction. The dysrhythmias are diverse and may range from transient alterations in the electrocardiogram indicative of, for examble, ischaemia, to ventricular fibrillation (VF), an arrhythmia likely to cause death unless skilled medical attention is available immediately. The capricious dysrhythmias make it difficult to obtain a correlation between the biomechanical response of the chest wall and the electrical dysfunctions. It may well prove possible to determine a significant correlation between myocardial damage and chest displacement. However, the relationship between cardiac contusions and the dysrhythmias is unpredictable and therefore significant correlation of dysrhythmias and chest mechanical response is not possible.

There has been little attempt in the past to understand the biomechanical mechanisms involved in cardiac contusion, even though the condition was first described by Akenside in 1764 (Ref 2). The occurrence of cardiac contusions has only recently been well described in clinical cases. The misconception

that non-lethal non-penetrating trauma to the heart was relatively rare is probably due to the fact that small myocardial contusions or traumatic pericardial lesions are usually well tolerated and the clinical findings difficult to recognise. More serious cardiac injuries are often seen in association with and overshadowed by extensive compound injuries and although rupture of a cardiac chamber may be obvious at post mortem, it is impossible for a pathologist to view an intact heart and decide that VF or other serious arrhythmia was responsible for a death. There are uncertainties therefore in the frequency of death from non-penetrating myocardial injury and any injury short of frank rupture may escape attention forever unless long term complications appear.

The response of the thorax to non-penetrating injury must be characterised not only in terms of the biological response of the target but also in the physical characteristics of the impact, and any attempt to define the relationship between the impact and its physiological and pathological effects must include the consideration of a large number of variables. The impactor must be defined in terms of its mass, velocity, dimensions and material properties. Similarly, the target characteristics are required: impact site, target mass, morphometry and suitability as an analogue to man - in particular whether its bio-mechanical, physiological and pathological response can be considered as representative of man, and whether scaling may be necessary.

Experimental techniques used to investigate cardiac contusion have ranged from the simple expedient of striking the exposed heart with a spatula (Ref 3) to the recent studies of Viano et al (Ref 4, 5) who attempted to correlate gross myocardial injury, myocardial conduction dysfunction and gross thoracic injury in pigs, to the mechanical impact response (applied force, sternal acceleration and chest deflection). A rigid impactor of mass 21 kg, diameter 15 cm, travelling at a constant velocity between 3 and 12 ms^{-1} was made to strike the thorax of an upright seated pig. Peak chest deflections were typically reached in about 20 ms and the characteristics of this type of injury are pertinent to car crash injuries.

This paper describes the biomechanical response of the chest wall to the non-penetrating impact of 3 smaller, faster impactors. Cardiac injuries are defined in terms of gross pathology and dysrhythmias and a correlation between the injury severity and the maximum transient chest wall displacement is determined. A mathematical model based on all the impact data is developed which relates the pre-impact kinetics of the impactor to the dynamic behaviour of the chest wall.

METHODS

The variables used in this study are defined in Table I. The non-penetrating impactor is defined by its mass, velocity and effective diameter. Each variable can significantly affect the type and severity of chest injury. Three different impactors were used to assess the dependence of chest injury on impactor characteristics:

Impactor A: Incompressible PVC cylinder, mass M = 0.14 kg, length 10 cm, effective diameter D = 3.7 cm.

Impactor B: Identical to A, with mass increased to M = 0.38 kg by steel insert.

Impactor C: Impactor A rotated through 90° about its short axis, with stabilising rod attached parallel to the axis of flight. Impactor mass M = 0.14 kg but effective diameter D = 10 cm.

Impactor C is in fact 10 cm x 3.7 cm. It is shown later that it behaves on impact as an impactor of diameter 10 cm when compared with A and B (D = 3.7 cm). The impactors were fired by compressed air at velocities between 20 $m.s^{-1}$ and 70 $m.s^{-1}$ against the mid-sternum of anaesthetised pigs (mass from 19 kg-86 kg) placed in a prone position. All impactors were free flying and delivered over 99% of their kinetic energy to the target. Impactor C was orientated to impact parallel to, and overlying, the body of the sternum. C was also used against the right lateral thorax. Table II summarises the impactor characteristics and the anatomical location of the impacts. The primary physical measurements were:

a. Velocity of impactor (V).

b. Displacement (P) of the wall of the thoracic cage v time.

c. Overpressure in the right ventricle of the heart v time (A only).

The following data were then derived from the above:

d. Deceleration of the impactor v time.

e. Time to peak deceleration, and its value.

f. Rate of application of energy to the chest wall.

g. Correlation between the overpressures in the heart and a-f above.

The primary physiological measurements were:

h. Electrocardiogram (Standard Lead II).

i. Instantaneous tidal air flow ($\dot{V}$).

j. Arterial blood pressure.

k. Arterial pH, P(CO2) and P(O2).

l. Morphometry of the pig.

Consideration of the physiological effects of the impact and a post mortem examination allowed an assessment of the severity of the skeletal fractures, gross myocardial damage and the cardiac dysrhythmias. Attempts were then made to correlate the injuries with the items a-g above.

Estimation of V, P and calculation of derived parameters The impactor velocity V and chest wall displacement P were determined using conventional high-speed cine photography at 5000 fps. The chest wall displacement under the impact

point was determined by observing the rear of the impactor, assuming that it did not tumble or distort and that the animal remained stationary. Peak displacement (Pmax) was typically reached in 3 ms after impact - the movement of the animal in this time was negligibly small. All film analyses, subsequent calculations and data storage were performed on a Hewlett Packard 9845B calculator, 9874A Digitizer and 9872A Plotter. The impactor kinetic energy was derived from the strike velocity. Plotting the location of the rear edge of the impactor (or stabilising rod for C) allowed chest wall displacement to be measured. An example of this primary data for pig 13/79 is shown in Fig 1 (solid line). A ninth order polynomial was fitted to the measurements of impactor travel. Numerical differentiation was used to calculate velocity (Fig 1 broken line), deceleration and rate of energy deposit for the impactor. The maximum transient chest wall displacement Pmax was estimated from the primary data of Fig 1. All chest deflections were also expressed as a fraction of the anteroposterior diameter (AP) at mid-sternal level. The lateral chest depth (LAT) at the point of impact was used for the strikes on the right lateral thorax. The variability in (AP) and (LAT) in animals of differing body weights was removed in this way and gave estimates of relative compression of the chest wall. For example, a Pmax of 8 cm in a 50 kg pig with (AP) = 24 cm gives Pmax/(AP) = 0.33 whereas an identical displacement in a smaller pig (W = 30 kg) and (AP) = 19 cm yields Pmax/(AP) = 0.42, which is likely to produce more severe injuries than the value 0.33.

Instrumentation Synchronisation Figure 2 outlines the layout of the instrumentation and the synchronisation of its various elements. Pushing "Start" on the "impact synchroniser" starts the high-speed cine camera. After 200 ms (nominally), the camera is running at 5000 fps and an event relay in it fires the gun. The incorporation of the "impact synchroniser" allowed the impact to occur at a predetermined point on the cardiac cycle. Preliminary experiments suggested that the heart was particularly vulnerable to acute VF if the impact occurred on the T-wave of the ECG - roughly coinciding with the ventricular repolarisation phase of the cardiac cycle. The "impact synchroniser" allows fibrillation vulnerability to be investigated but its main function in these trials was to minimise the probability of immediate VF which would otherwise obscure any gross myocardial damage which may be produced by synchronising the impact to occur randomly at any point on the cardiac cycle other than the T-wave. The synchroniser required an ECG from the animal and this was displayed on a Tektronix 466 scope with a pulse indicating the chosen point of impact on the ECG. This preselected trigger point initiates the camera run-up sequence on the cardiac cycle following the "Start" command from the sequencer.

Animal preparation and physiological instrumentation Pigs of the Large White strain within the weight range 19-86 kg were first sedated by an air/halothane mixture to permit venepuncture. Sodium pentobarbitone (SAGATAL) was administered to effect surgical anaesthesia and the animal was maintained by bolus intravenous administration when required. A patent airway was assured by tracheotomy and the animals were allowed to breath spontaneously. Arterial and venous catheters were introduced into the aorta and vena cava via the femoral vessels for the recording of arterial blood pressure and the administration of anaesthetic respectively. Arterial blood flow was recorded using a GAELTEC miniature Luer transducer. A lead II ECG was obtained using subcutaneous stainless steel electrodes. Instantaneous tidal air flow rate ($\dot{V}$) was measured using a Fleisch pneumotach and Hewlett Packard model 270 differential air

pressure transducer. Integration of $\dot{V}$ allowed an estimate to be made of expiratory tidal volume. All bioelectric signals were conditioned by a Hewlett Packard 7754A hot pen recorder with simultaneous transcription to an Ampex tape recorder.

The animal was suspended in the prone position approximately 60 cm above the muzzle of the compressed air gun in a stainless steel frame which supported the head and abdomen whilst allowing access to the thorax. Physiological recordings were undertaken for 30 minutes prior to impact and 60 minutes following it. The animal was sacrificed without it regaining consciousness from the anaesthesia and a post mortem examination performed. The injuries following the mid-sternal impacts were present in the subcutaneous tissues, the sternum and the heart. The right lateral impact kinetic data have been included solely to determine the validity of biomechanical models discussed later.

The categorisation of cardiac and sternal injury is shown in Table III. It must be strongly emphasised that the injury scales are arbitrary and do not imply a linear scaling of severity. Grade 2 in the cardiac injury scale is not twice as serious as Grade 1 nor half as serious as grade 4. These scales have not been used as groupings defined by the clinical assessment of injuries. Hearts are grouped purely on visible pathology. A very small myocardial bruise is classed the same grade (3) as a full thickness myocardial contusion involving a large portion of the ventricular surface area. The clinical prognoses of these injuries are, however, significantly different.

RESULTS

The results are presented in two parts:

A. Correlation of injury with chest compression.

B. Comparison of the chest displacement produced by the three impactor types to derive a model to predict chest displacement (Pmax or Pmax/(AP)) from known impact characteristics and animal morphometry (M, V, D, W and (AP)).

A. Injury and its relationship to chest wall displacement

Sternal Injuries

Injuries to the sternum were classified as no fracture (NF), a fracture without displacement (F), and a fracture with displacement (D). The pig sternum consists of seven sternebrae, and the fractures tended to lie between the sternebrae across the less well ossified cartilaginous junctions. Ribs were occasionally dislocated at the sternocostal articulations with impactors A and B but the most common injury with these impactors was fracture and displacement of the body of the sternum. Figure 3 shows the relationship between relative chest wall displacement (Pmax/(AP)) and the sternal injury scale for impactors A and B. Greater chest wall displacements tend to result in more significant sternal injury (assessed by the NF, F and D injury scale). A Kendall Rank Correlation test demonstrated a highly significant correlation between Pmax/(AP) and sternal injury. Strikes with impactor C did not produce injuries to the body of the sternum because the impactor overlaid most of the sternum.

Injuries were confined to the sternocostal articulations and were either simple dislocations or rib displacements depending on the severity of the chest compression.

Cardiac Pathology

The relationship between severity of heart injury (as assessed by gross pathology and the injury scale to Table III) and the relative chest wall displacement Pmax/(AP) is shown in Fig 4. The figure uses data from impactors A and C on the mid-sternum but excludes any animal that succumbed to acute VF. Two pigs suffered cardiac rupture and could not be included because the projectile became unstable and Pmax could not be determined. The correlation between cardiac injury and Pmax/(AP) is also highly significant. The ruptures in the right ventricle occur predominantly at the right ventricular-interventricular septum border near the paraconal interventricular branch of the left coronary artery (the left anterior descending coronary artery). This is a relatively thin portion of myocardium - ruptures have been reported in this region in man when subjected to over-enthusiastic therapeutic cardiac compression. The myocardial, epi- and endocardial bruising was not confined to the portion of the right ventricle immediately adjacent to the sternum beneath the point of impact. Contusions were often seen in the interventricular septum and on the left ventricular walls. In some cases epicardial bruising was seen to follow the course of the left anterior descending coronary artery. The reason for this is unknown - transmission of pressure from the compressed ventricles down the coronary arteries may be involved.

Those animals struck by impactor B showed uncharacteristic localisations of the contusions. Myocardial contusions and ruptures occurred in the particularly thin area of myocardium at the RV-IVS border near the pulmonary infundibulum mentioned above. The contusions were generally small and there was no evidence of substantial bruising to other parts of the ventricular walls commonly seen with the lighter impactors. In three of the five animals, endocardial bruising was seen on the pulmonary muscles - an uncommon observation when the lighter impactors A and C were used. This different pattern of injury is difficult to explain. The heavier impactor tends to take longer to reach maximum chest wall displacement (4-5 ms) than the lighter impactors (2-3 ms), which may be related to the different injury pattern.

The Dysrhythmias

The cardiac dysrhythmias associated with each impact will not be presented in detail. These experiments have demonstrated that a quantitative relationship cannot be determined between the severity of the impact and the dysrhythmias produced (expressed as type and duration). The relationship is qualitative - on the whole, those animals suffering impacts of high energy exhibited more serious dysrhythmias than those impacted at low energy. It was also not possible to produce a quantitative relationship between gross myocardial injury and the type of dysrhythmia produced. There is no characteristic dysrhythmia of cardiac contusion. A period of ventricular standstill immediately following impact is often seen - the maximum duration in these experiments was 64 secs. S-T slurring and depression (indicative of ischaemia) is fairly common but on the whole the variety and variability of electrocardiogram changes produced does not suggest any particular dysrhythmia predominates.

Ventricular fibrillation (VF) was the most serious arrhythmia that occurred on impact. VF is a chaotic, random, asynchronous electrical activity of the ventricles which results in unco-ordinated ventricular contraction and prevents effective cardiac output. Death is almost inevitable unless active defibrillation is achieved by electrical discharge from a defibrillator. VF occurred in three ways following impact:

1. Subsequent to a ventricular or supraventricular tachycardia (and some other dysrhythmias) a discrete time after the blow. This was not very common.

2. It was seen following cardiac rupture but its significance was obviously diminished and its occurrence was probably a non-specific electrically unco-ordinated response to the gross ventricular injury.

3. Potentially the most serious occurrence was its acute initiation following the impact - termed acute "VF".

The tendency of the heart to fibrillate in an animal did not show any significant relationship to the biomechanical responses of the animal. It was noted however that of six pigs showing acute "VF", four had been struck coincident with the T-wave on the electrocardiogram. The T-wave occupied only 1/5 of the cardiac cycle and the disproportionately high incidence of acute VF associated with this wave suggests the presence of a "vulnerable period" for VF. Seven animals were struck coincident with the T-wave and four fibrillated. A Chi-squared test showed that the T-wave does indeed represent a statistically significant vulnerable period for acute VF following a blow to the chest. This is not surprising since the T-wave represents an electrically unstable heart - a ventricle that is uniform electrically cannot fibrillate. During the T-wave the myocardial cells are repolarising randomly - at any time some will be repolarised, others will be partially repolarised and the rest depolarised. The latter are refractory to stimulation. Conduction becomes fractionated and a stimulus occurring at such a time may provoke disordered electrical activity. Although this is the first time that a vulnerable period for acute VF following a blow to the chest has been described, it has been known for some time that electrical shocks of sufficient intensity to initiate VF are only effective if the electrical pulse falls within a "vulnerable period" roughly coincident with the T-wave (Ref 5).

It is not possible to determine if blows to the chest of man can initiate VF. It is known that a precordial thump can sometimes defibrillate a fibrillating patient - it could perhaps be argued that an impact to a healthy man could also produce VF. It is known from studies of VF vulnerability to electrical shock that the VF threshold can be lowered by anoxia, certain anaesthetics, acidosis, hypothermia, sympathetic nerve stimulation and circulating catecholamines. Any or all of these may occur in an anaesthetised animal and the experimental conditions of this trial may predispose to VF.

B. The relationship between impact characteristics and chest wall displacement

For any particular anatomical site of impact and animal species, the chest compression is likely to be a function of the mass of the impactor (M), its velocity (V), its diameter (D), and the morphometry of the animal target (W

and (AP)). These variables must be combined to form a mathematical expression which relates the response of the chest wall, Pmax, to the impact. The mathematical model incorporating M, V, D, W and (AP) must possess four characteristics:

1. It must be a relatively simple expression.

2. It must provide a good fit to the data points of a particular impactor to a curve (W and V varying).

3. The data from all impactors should lie on a common curve (M and D varying).

4. It must satisfy known boundary conditions (eg Pmax = 0 when V = 0).

If the results from all impactors can be made to fall on the same curve then it may be possible to predict chest compression for an impactor from kinetic data alone. Internal injuries can probably be predicted if chest compression is known.

For a given impactor, the smaller the weight of the animal the greater the relative chest wall displacement. Similarly, the application of the force over a larger area will result in a lower relative displacement. A model defining the relationship between chest wall displacement and pre-impact kinetics must satisfy the criteria above. There are two conflicting requirements - accuracy and simplicity. Biological systems tend to be complicated and therefore an over-simplified model may not predict effects with sufficient accuracy. In order to produce a highly accurate model, the mathematical expressions may become complicated, cumbersome, difficult to understand and possibly incapable of widespread applicability. The models outlined below are a compromise between accuracy and simplicity. Two models are available:

1. Relating pre-impact kinetics to the relative chest wall displacement Pmax/(AP).

2. Relating pre-impact kinetics to the absolute chest wall displacement Pmax.

The models were formulated using all the mid-sternal impact data of impactors A, B and C.

Model 1: Pmax/(AP) related to pre-impact kinetics

It was found that the axes giving the best grouping within each set of data and the best correlation between all the sets (impactors A, B and C) was:

$$\text{Pmax}/(\text{AP}) \text{ v } E/WD$$

Figure 5 shows the combined mid-sternal impact data from all impactors. The data are well grouped despite differing effective diameters, impactor masses, strike velocities and animal weights. The data were plotted using a Hewlett Packard 9845B calculator and curves were generated which gave the best visual

fit. The curve was constrained to pass through the origin because zero impact energy obviously produces zero chest compression. Figure 5 includes the data from the right lateral data for comparison.

The equation of the best curve to the mid-sternal data is:

$$y = 0.4(1 - \exp(-0.95x)) \qquad 1$$

where $y = Pmax/(AP)$ and $x = E/WD$

where E = impactor kinetic energy (J), W = animal weight (kg), D = effective impactor diameter (cm) and Pmax/(AP) = relative maximum chest wall displacement.

Transforming this equation into its linear form allowed calculation of the correlation coefficient, r, of the fit to the data. Linearising equation 1 gives:

$$\log (0.4 - Pmax/(AP)) = \log 0.4 - 0.95 (E/WD)$$

with a correlation coefficient of r = 0.95. As indicated above, Fig 5 shows the right lateral data of impactor C plotted on the same axes with (LAT) replacing (AP). The model tends to predict lower values of Pmax than those found in practice based on the mid-sternal impacts.

Model 2: Pmax related to pre-impact kinetics

The best relationship between absolute chest wall displacement, Pmax, and pre-impact kinetics was:

$$Pmax \text{ v } E/D \text{ (AP)}$$

Figure 6 shows the mid-sternal impact data plotted on these axes. The curve giving the best visual fit to the combined results was:

$$y = 10.3(1 - \exp(-0.6x)) \qquad 2$$

where $y = Pmax$ and $x = E/D(AP)$

The units are as in equation 1. This curve is drawn in Fig 6 and has a correlation coefficient r = 0.93. The right lateral data is included for comparison and the fit to the curve is encouraging.

An important conclusion is that chest wall displacement (and consequently thoracic injury) is kinetic energy dependent. Neither the momentum nor velocity correlated with chest displacement. The premise that chest displacement is inversely related to impactor diameter is a simple assumption but appears to be valid. The other assumption that displacement is inversely related to animal mass and chest diameter is also correct - the range of mass in this study is from 19-86 kg.

Experiments are planned to widen the scope of the models. The impactors masses and diameters used in this study were limited but the impactor velocity and animal mass varied considerably (Table II). The use of

equations 1 and 2 to predict chest compression for impactor masses and diameters outside this range cannot be justified at this stage although the kinetic energy dependence and the inverse relationship between chest displacement and impact diameter would probably still hold.

CONCLUSIONS

1. Sternal fracture and gross myocardial injuries show a significant correlation to the degree of chest displacement when pigs are subjected to non-penetrating mid-sternal impacts.

2. The type and duration of cardiac dysrhythmias following impact cannot be quantitatively correlated with any of the measured biomechanical parameters. The relationship is purely qualitative.

3. Acute ventricular fibrillation can be initiated by a blow to the mid-sternum. There appears to be a vulnerable period associated with the T-wave of the ECG during which the heart is more susceptible to fibrillation.

4. Pressure changes within the right ventricle of the heart show a positive correlation with chest displacement.

5. Two mathematical models have been derived which predict chest wall displacement following mid-sternal impact if the mass (M), velocity (V), impactor diameter (D) and animal weight (W) or A-P chest diameter (AP) is known. The first predicts relative chest wall displacement (Pmax/(AP)); the second, absolute chest wall displacement (Pmax).

REFERENCES

1. CLARE V.R. et al. Blunt trauma data correlation. Edgewood Arsenal Technical Report No EB-TR-75016, 1975.

2. AKENSIDE M. Account of blow upon the heart and its effects. Phil Trans Roy Soc London (Biol), 353, 1764.

3. BECK C.S. Contusions of the heart JAMA, 104: 109-114, 1935.

4. VIANO D.C., WARNER C.Y. Thoracic impact response of live porcine subjects. Paper 760823, Proc 20th Stapp Car Crash Conf, Warrendale, PA, USA. 1976.

5. VIANO D.C., KROELL C.K. and WARNER C.Y. Comparative impact response of living and sacrificed procine siblings. Paper 770930, Proc 21st Stapp Car Crash Conf. 1977.

TABLE I LIST OF SYMBOLS AND THEIR UNITS

Symbol	Definition	Units
M	Mass of the impactor	kg
D	Effective diameter of the impactor	cm
V	Velocity of the impactor	$m.s^{-1}$
E	Kinetic energy of the impactor	Joules (J)
W	Mass of the pig	kg
(AP)	Chest depth of the pig measured anteroposteriorly at mid-sternum	cm
(LAT)	Chest depth of the pig measured laterally at the point of impact	cm
P	Displacement of chest wall	cm
Pmax	Maximum transient chest wall displacement	cm

TABLE II IMPACTOR TYPE AND KINETICS

Impactor Type	Impact Site	Mass (M) kg	Effective Diameter (D) cm	Velocity Range (V) $m.s^{-1}$	Animal Mass Range (W) kg
A	Mid-sternum	0.14	3.7	35 - 72	25 - 86
B	Mid-sternum	0.38	3.7	20 - 34	26 - 44.
C	Mid-sternum	0.14	10.0	50 - 64	19 - 40
C	Right lateral thorax	0.14	10.0	26 - 47	25 - 43

TABLE III INJURY GRADES FOR THE CATEGORISATION OF CARDIAC AND OF STERNAL INJURY

Organ	Grade	Gross Pathology
HEART	1	No visible damage
	2	Epicardial or endocardial bruising only
	3	Myocardial bruising
	4	Rupture of a cardiac chamber
	5	Animal succumbed to acute ventricular fibrillation Pathology not assessed
STERNUM	NF	Not fractured
	F	Fractured but not displaced
	D	Fractured and displaced

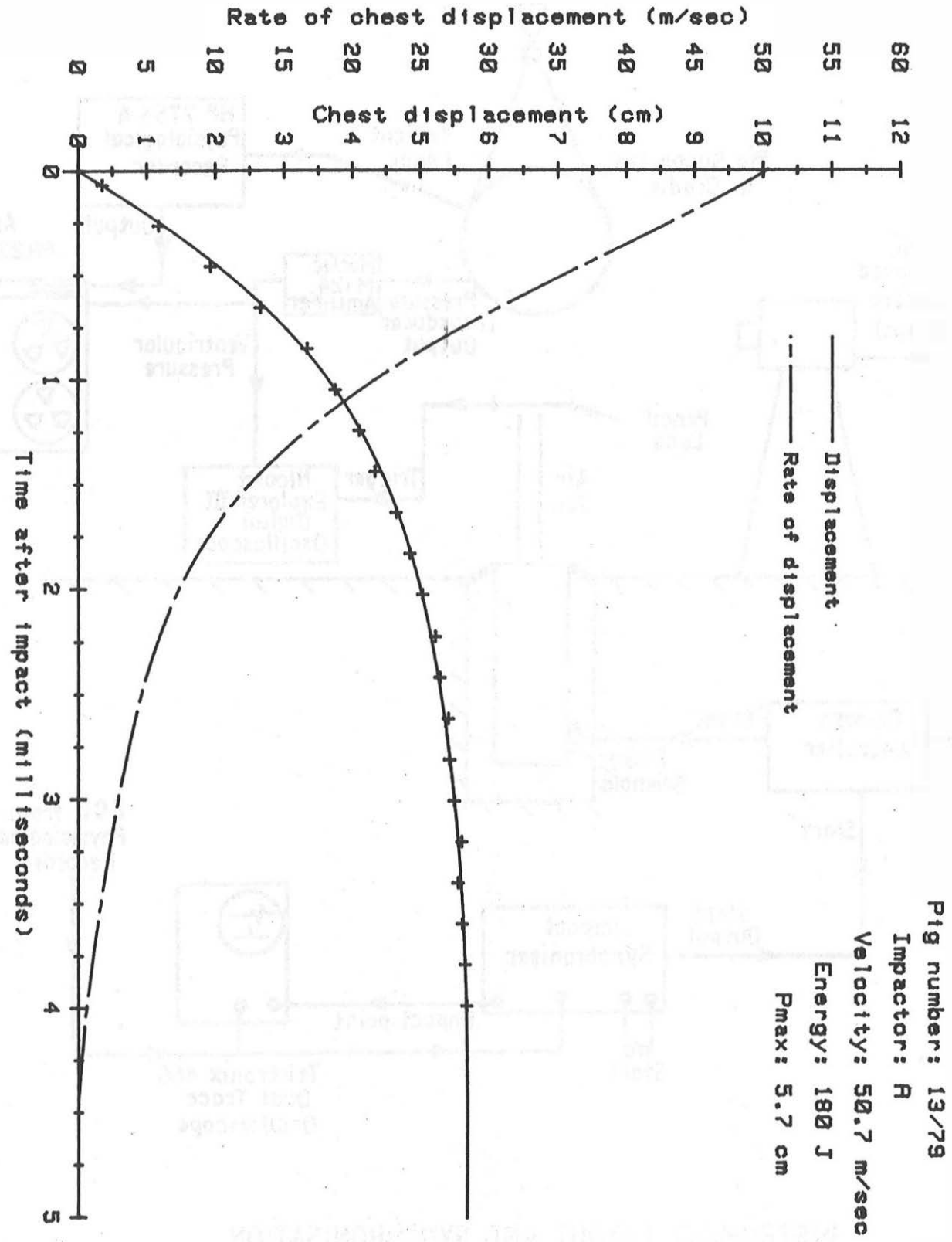
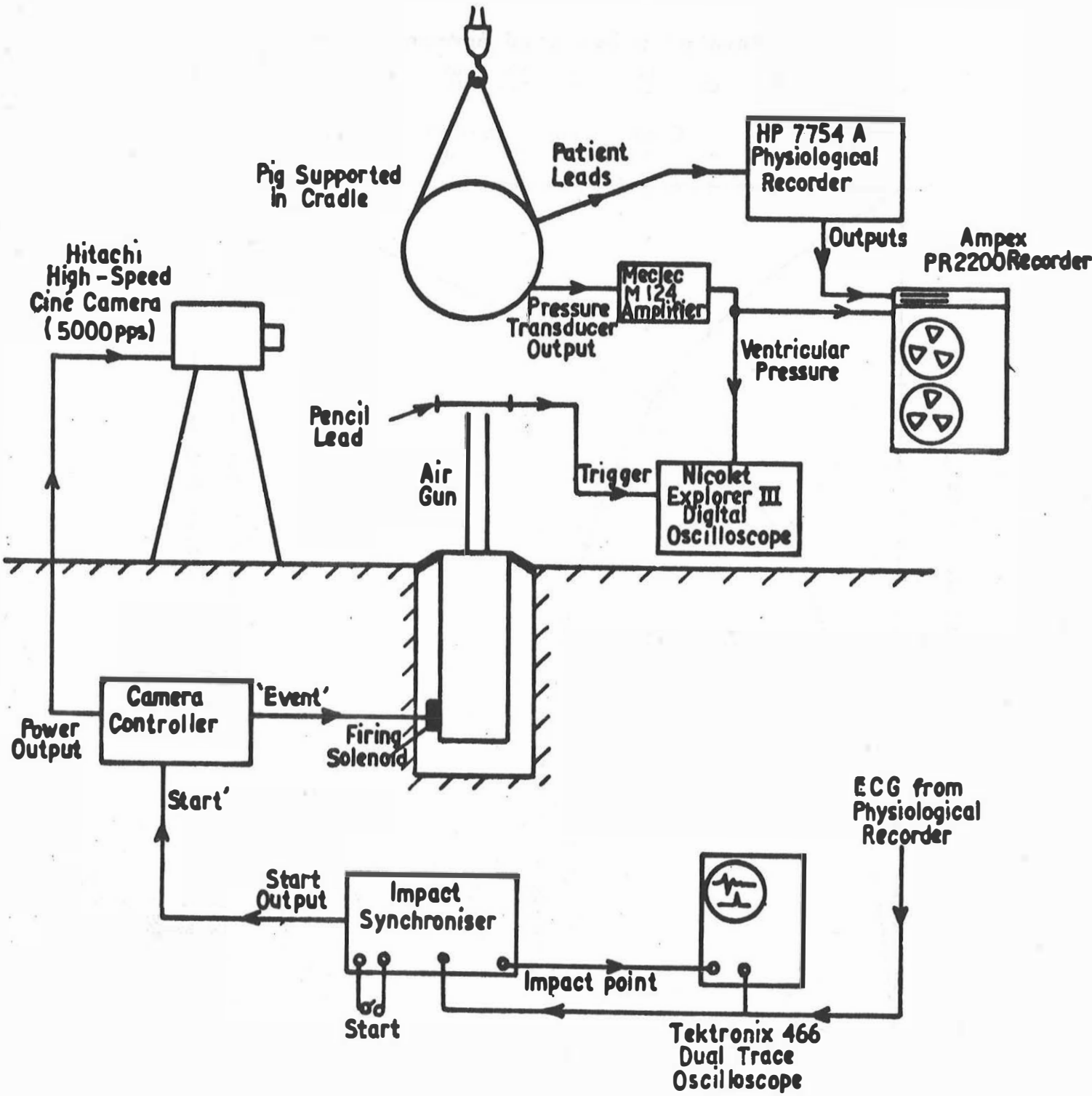


Fig.1 Typical displacement results



INSTRUMENT LAYOUT AND SYNCHRONISATION

FIG.2.

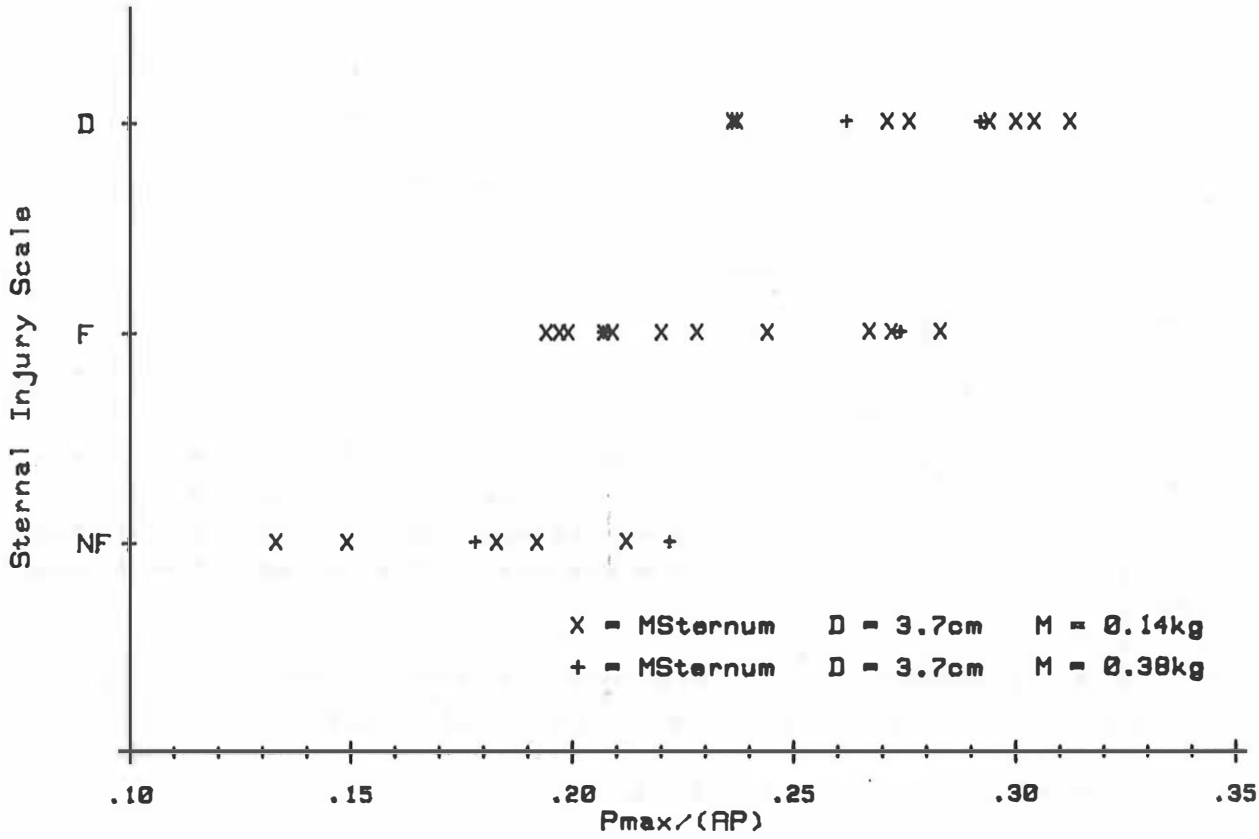


Fig.3 Relationship between Sternal Injury and Pmax/(AP)

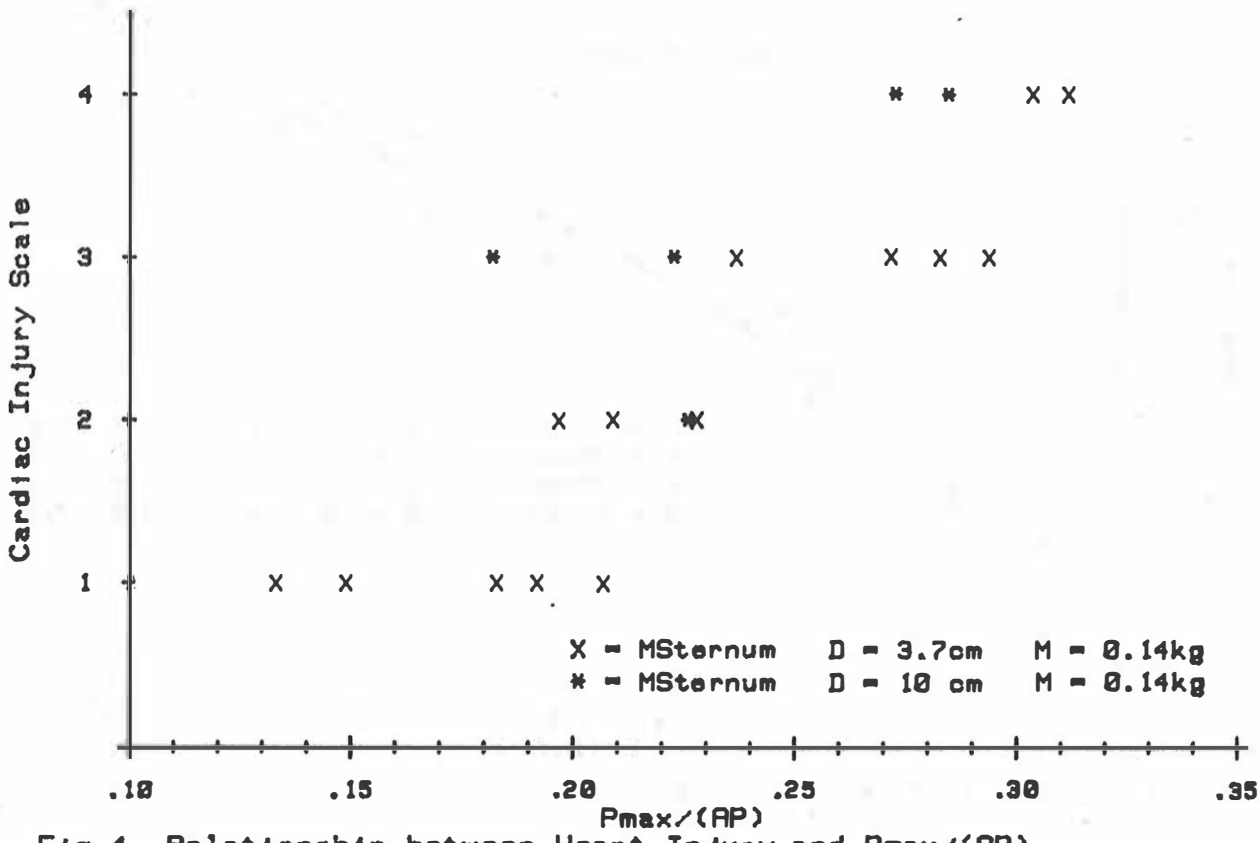


Fig.4 Relationship between Heart Injury and Pmax/(AP)

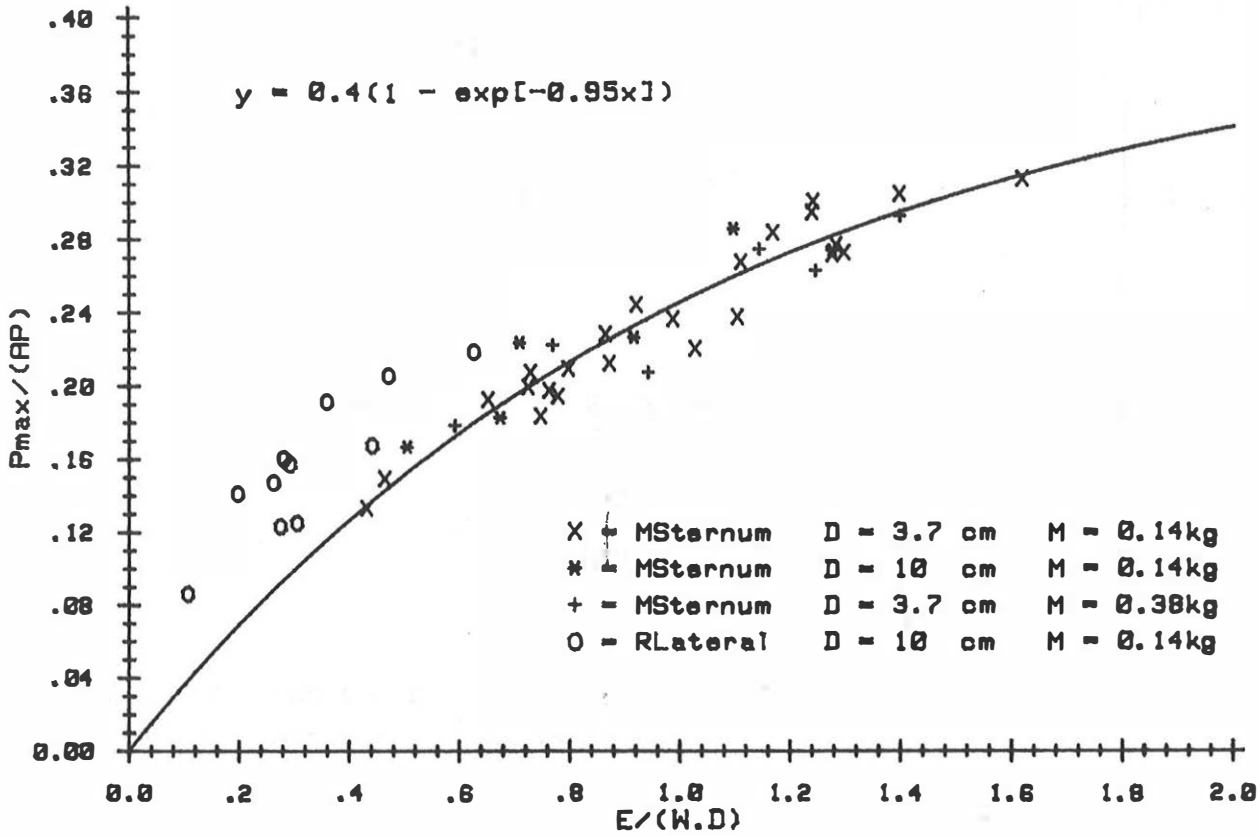


Fig.5 Model 1: Pmax/(AP) v E/(W.D)

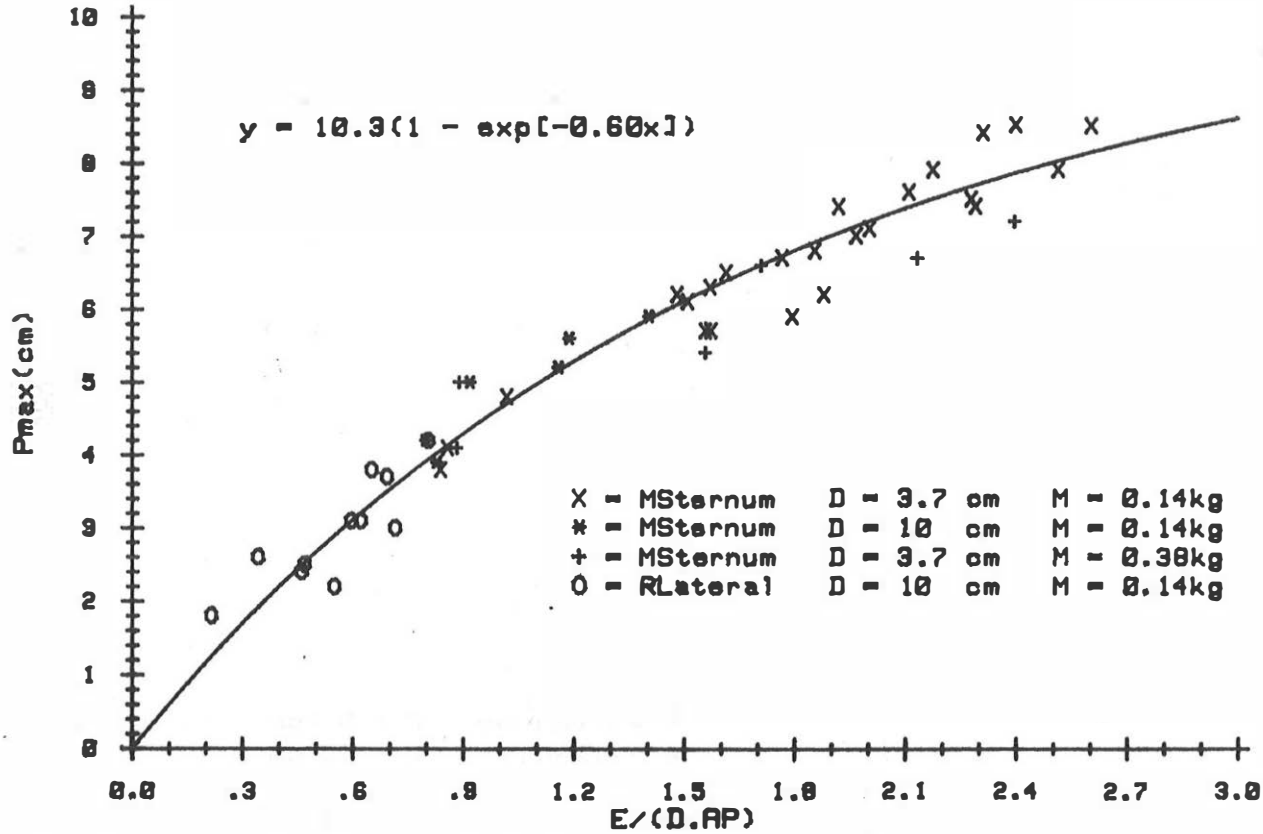


Fig.6 Model 2: Pmax v E/(D.AP)