USE OF ELECTROMAGNETIC COUNTERPOISE TO MEASURE CARDIAC FORCE

D.R. KULKARNI, P.V. DIWAN, L.D. TILLOO, P.A. PATIL AND M.K. BAGI

Department of Pharmacology,
J.N. Medical College, Belgaum-590010, Karnataka

Summary: To quantify the drug induced changes in cardiac force the methods in vogue use springs and strain-gauge coils, which apply counterforce to oppose cardiac force and thus stop systolic excursion of the lever.

The present report pertains to a new technique based on similar principle, utilising induced electromagnetic force as a means of counterpoising in study of contractility of isolated frog heart. The technique monitored changes in contractility produced by small doses of adrenaline, digoxin, acetylcholine and CaCl₂. The plots of estimated cardiac force (in V) against doses (log-units) of the drugs could be made to reveal dose response relation.

The technique uses components simple to fabricate and work with; it is reliable and sensitive.

Key words: cardiac force estimation
isometric tension
counterpoise afterloading
emagnetic force

INTRODUCTION

Myocardial contractility is usually expressed in terms of some index associated with conditions of the experiment rather than in any fundamental units which relate to the muscle itself (4). From amongst the commonly used indices, isometric tension more faithfully depicts the state of contractility (2). Strain-gauge coils, arches and tension transducers are sophisticated gadgets employed to measure isometric tension. These devices are expensive and often not easily available. Indeed, such a situation prompted us to fabricate and test the device described below, in which induced electromagnetic force is utilised for counterpoising cardiac force.

MATERIALS AND METHODS

Hearts isolated from frogs (Rana tigrina; 50-70 g weight) were perfused with Frog-Ringer at constant rate and pressure. All experiments were carried out at room temperature.

Myographic lever with an afterload screw was used to record isotonic heart beats. The lever was provided with a light-weight iron-shoe to over-hang the electromagnetic unit kept underneath.

A counterpoise unit was fabricated utilising an electromagnetic time-marker, a low voltage unit and a ‘dimmerstat’. Inclusion of a voltmeter permitted assessment of voltage at any given time. Fig 1. shows the entire set up.

*Preliminary results were presented at III Southern Regional Conference, I.P.S., November, 1976 and the technique demonstrated in ‘Pre-workshop in Research Methodology., 9th Annual Conference, I.P.S., Varanasi, December 1976
Fig. 1: Set-up of apparatus.

Note: Tension and shortening of A-B are recorded.

Setting-up of the preparation:

After the stabilisation of the heart over 20-30 min the after-load screw was positioned to permit fullest diastolic stretching for the given pressure and rate of perfusion. Then, the distance between the iron-shoe and the magnetic unit was adjusted to be narrow enough but never permitting actual contact between the two. The positions of the after-load screw and the magnetic unit so set were not disturbed till the end of the experiments.

Estimation of cardiac force:

After recording the isotonic contractions for a few min the electromagnet was activated and the intensity of generated magnetic force was swiftly increased by manipulating the current strength flowing through the magnet until the developed magnetic force was just enough to counter-balance the cardiac force and thus stop lever excursions. The voltage value was noted and magnet switched off. The procedure was repeated 3-4 times at five min intervals. The mean of the (voltage) readings provided an estimate of base-line (control period) cardiac force.

Testing of drugs:

Procedure I: After noting the base-line cardiac force, the contractile force was again estimated when adrenaline (1-20 ng), CaCl₂ (50-800 µg) or acetylcholine (5-80 ng) produced peak effects as assessed from isotonic records. A mean of three separate measurements was calculated.

Procedure II: After not giving drug-solution to the heart, the contractile force was measured and acetylcholine (Fig. 3) produced a reduction in the estimated cardiac force against hypodynamic condition of the heart. Since the heart was perfused with frog-saline, the degree of hypodynamic condition of the heart was observed. The principle adopted has been that is exactly equal to the maximum development and force-velocity relationship would influence a

Though the entire heart was driven home before starting the procedure, it was found to be wide apart depending on the drug-solution given in the hypodynamic condition of the heart.

In the present technique, the value of which reflects the inotropic effects of any drug used on the heart. A total or digoxin (in cardiac force). The procedure was performed to observe cardiotonic effects of the drug in the hypodynamic condition of the heart. It was possible to establish the inotropic effects of any drug on the heart. The principle adopted has been that is exactly equal to the maximum development and force-velocity relationship would influence a
After noting the control cardiac force, the electromagnetic counterpoise was allowed to act continuously and cardiotonics like adrenaline (5-20 ng) or digoxin (10-50 ng) were given and the degree of recovery of lever excursions was noted.

In either of the procedures the interval between switch-on and switch-off of the magnet was 30-60 sec. The passage of current over such a short period did not cause any change in either the temperature or the resistance of the coil.

The volume of drug-solutions administered did not exceed 0.25 ml; in the case of digoxin however, the volume was 2-4 ml. Before administering the drug a saline control was obtained by giving frog-saline in volumes corresponding to drug volume.

Since the heart was perfused at constant rate and pressure and since the after-load screw was driven home before starting the estimation procedures it is very unlikely that tension-length relationship would influence and vitiate the estimated values.

Though the entire heart is mounted the force estimated corresponds to the isometric tension developed by the segment of the cardiac tissue between two fixed points (A-B of Fig. 1).

**RESULTS**

**Procedure I:** A total of 42 experiments were carried out. Adrenaline (Fig. 2), CaCl₂ and acetylcholine (Fig. 3) produced typical effects which were clearly measurable. Plots of estimated cardiac force against log-doses showed linear relationship; the dose-response curves were found to lie wide apart depending upon the drug, its dose and sensitivity of the heart (Fig. 4).

**Procedure II:** Digoxin produced a recovery (Fig. 5) of lever excursions (i.e. an increase in cardiac force). The procedure enabled the digoxin-inotropism to be demonstrated without a hypodynamic condition of the heart. In all the 10 experiments (Procedure II) it was possible to observe cardiotonic effects of the drug in doses less than 50 ng. However, only in few experiments it was possible to establish dose-response relationship.

**DISCUSSION**

The inotropic effects of any drug is best described in terms of its influence on isometric tension development and force-velocity relations (3). The present technique monitors the former. The principle adopted has been that an after-load which just stops isotonic shortening represents isometric tension developed at that moment. It is well established that the imposed after-load that is exactly equal to the maximum tension (isometric) that the muscle is capable of developing prevents segment shortening (3,4).

In the present technique electromagnetic force has been used as an imposed after-load, the value of which reflects the isometric tension developed at the time of counterpoise. Since
Fig. 2: Cardiogram showing effects of adrenaline by Procedure 1 (see text).
Upstroke-systole, C-control, E-Adrenaline, V-Voltage.
*Note:
(i) Gaps in isotonic tracings indicate application of magnetic counterpoise to stop isotonic shortening.
(ii) Voltage values reflect magnetic force equalling cardiac force; these values increased with adrenaline dose.

Fig. 3: Cardiogram showing effect of acetylcholine by Procedure 1 (see text).
C-control, Ach-acetylcholine.
*Note: Voltage reflecting magnetic force decreases with increase in Ach dose.

DOSE RESPONSE CURVES (DOSE VS VOLTAGE)
(EACH POINT IS MEAN OF 3 READINGS)

VOLTAGE REFLECTING E.M.F.

DOSE (LOG SCALE)

Fig. 4: Few prototype dose response curves for acetylcholine, adrenaline and CaCl₂.
EMF = Generated electromagnetic force. n = number of experiments.

As long as the electromagnetic force has been given as:
E.M.F. = \frac{2\pi nC}{I(r)}
the electromagnetic force has been given just sufficient to prevent shortening. For measuring the isometric tension.

In procedure I, an estimate as at the peak effect of drugs given is proportional to changes produced by drugs in the ship between log-doses and voltage CaCl₂ and the negative inotropic effect.
We concluded therefore, that the
In procedure I, an estimate of isometric tension was made during the control period as well as at the peak effect of drugs given in different doses. The changes in voltage values reflected changes produced by drugs in the development of isometric tension. There was linear relationship between log-doses and voltage values (Fig. 4) for positive inotropic drugs like adrenaline and CaCl₂ and the negative inotropic drug, acetylcholine. Further, the doses were in nanograms. We concluded therefore, that the method is reliable and sensitive.

The electromagnetic force has been used as imposed (counterpoising) after-load, Po (i.e. the after-load just sufficient to prevent shortening) should be expressed in terms of magnetic flux (force) given as:

$$ E.M.F. = \frac{2 \pi nC}{10r} $$

where, $E.M.F.$ = Electromagnetic force.

$n$ = number of turns in the coil.

$C$ = Current strength.

$r$ = radius of the coil.

As long as the electromagnetic unit remains the same, changes in $E.M.F.$ will be proportional to changes in $C$. Further, magnetic unit and its coil temperature remaining the same the resistance ($R$) of the coil also remains the same and therefore, the current strength ($C$) will be proportional to voltage ($V$), because, $C = V/R$. So, one can assume that when the same electromagnetic unit is used, Po can be expressed either in terms of $E.M.F.$, current strength or voltage. In our experiments we have taken voltage values as estimate of $E.M.F.$ and hence that of isometric tension.

In procedure I, an estimate of isometric tension was made during the control period as well as at the peak effect of drugs given in different doses. The changes in voltage values reflected changes produced by drugs in the development of isometric tension. There was linear relationship between log-doses and voltage values (Fig. 4) for positive inotropic drugs like adrenaline and CaCl₂ and the negative inotropic drug, acetylcholine. Further, the doses were in nanograms. We concluded therefore, that the method is reliable and sensitive.
In procedure II, the unloaded isotonically contracting myocardium was subjected to EM
counterpoise to abolish control systolic force. This counterpoise was allowed to act continuously
for 30-60 sec during which period positive inotropic drugs, adrenaline or digoxin was given and
percent recovery of segment shortening noted. This procedure does not involve counterpoise
to measure tension developed subsequent to drug administration; and hence, can not provide
an estimate of isometric tension developed after drug application. It is possible, however, to
have this, if nullifying counterpoise is applied at the peak recovery of segment shortening. But
such procedure will require the magnet being kept on for a longer time (i.e. more than 30-60 sec).
With the magnetic coil we have used, such procedure lead to increase in coil temperature and
hence in coil resistance. Therefore, in the present study (procedure II) only the percent recovery
has been noted.

Excursion amplitude of isotonic contractions of actively beating normal heart does not
increase much under the influence of digitalis (5). But digitalis-action on failing heart is much
more impressive than on normal one (1). Hence, to demonstrate digitalis effect one employs heart
rendered hypodynamic by perfusing Ringer containing less amount of CaCl₂ or Ringer having
added barbiturates (1). In the present experiments with partial counterpoise (procedure II)
actively beating normal heart also responded by increased force of contraction to digitalis adminis-
tration.

The device is not meant as a substitute for more precise and accurate gadgets but is inex-
 pense, easily fabricable and fairly dependable for routine work.

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