Effect on Heart Rate, Aortic Flow and Left Ventricular Pressure Induced by Coupled Pacing*

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Within recent years, considerable use has been made of electric currents to control the rhythm of the heart. Pacemakers and defibrillators have become part of modern day therapy. Recently, Lopez et al. indicated that cardiac slowing could be achieved by the administration of long (150-250 msec.) two volt pulses or pairs of short pulses separated by 150-250 msec. Subsequently Chardack et al. and Braunwald et al. have also reported on the application of single pulses coupled to the preceding R wave.

The purpose of this paper is to report on the technic of slowing the heart by applying a single short duration electrical stimulus and to observe the effect of this form of pacing on left ventricular pressure and aortic blood flow. Since electrical excitability returns when the ventricle is still mechanically refractory, it is possible to stimulate the heart, thereby increasing the electrical refractory period without obtaining a mechanical contraction. Single stimuli are synchronized to follow the R wave of the electrocardiogram and are appropriately delayed to fall within the desired portion of the cardiac cycle. This method is referred to as "coupled pacing," and inherently incorporates the advantage of atrial contraction which is lost in the A-V dissociation produced by paired-pulse pacing.

Methods

An apparatus has been assembled which will trigger a pacemaker to produce an impulse at any desired period after the R wave. The pacemaker output, which is isolated, is delivered to electrodes implanted in the canine right ventricular wall (Fig. 1).

Eleven mongrel dogs weighing between 12 and 20 kilograms were anesthetized with sodium pentothal, 29 mg./kg. and maintained on a positive pressure respirator. Right lateral thoracotomy was performed, the pericardium was opened, and the pacemaker electrodes implanted in the right ventricular wall. A conventional electrocardiogram was recorded from the limb leads. Left ventricular pressure was recorded through polyethylene tubing connected to Statham transducers and ascending aortic flow was determined using a square-wave electromagnetic flowmeter.†† Electrocardiograms, left ventricular pressure and ascending aortic flow were recorded on a multichannel recorder.‡

The apparatus was operated in the following manner. The synchronizer was turned on and the delay was adjusted to fall in the T wave of the electrocardiogram by observing the delay pulse on the oscilloscope of the recorder. The pacemaker was then turned on and the output adjusted to three volts. Further minor adjustment of the delay of the synchronizer was made to achieve a left ventricular pressure response only from the normally conducted beat.

If the delay was too short, the pacemaker beat was nonconducted due to the refractory period of the preceding beat. On the other hand, if it was too long, a premature beat appeared on the descending limb of the pressure curve.

The application of coupled pacing to the animals in this experiment was well tolerated. There were no instances of induced tachycardias in this study. Close attention had to be paid to the period of delay so

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**Corbin Farnsworth Model DSYC-3, synchronizer and SCPT-4, oscilloscope.
†Corbin Farnsworth Model PXIB—this pacemaker is modified to respond to synchronizer and remain refractory for up to 500 msec.
††Carolina Medical Electronics Model SBI.
‡Electronics for Medicine Model DR8.
that the pacemaker beat did not cause a ventricular response.

RESULTS
The sinus rate is slowed to half by coupled pacing. In the process of slowing there was a concomitant increase in left ventricular pressure and mean aortic flow. Table 1 summarizes the experiments and Fig. 2 demonstrates these changes.

The average increase in left ventricular pressure was 29 mm.Hg (P<.025) and the per cent increase varied from 22 per cent to 51 per cent (average increase was 34.3 per cent, S.D. 12.2).

The average increase in mean aortic blood flow was 234 ml./min. (P<.005) and the per cent increase varied from 7.6 per cent to 40.8 per cent (average per cent increase was 21.4 per cent, S.D. 9.25).

DISCUSSION
These experiments show that synchronized appropriately delayed single pulses (coupled pacing) can effectively slow the mechanical rate of a rapid heart and significantly raise left ventricular pressure and aortic blood flow. Siebens et al. demonstrated that recovery of electrical excitability precedes recovery of mechanical activity, and in effect, these single pulses are delivered to the ventricle at that time when electrical excitability has returned, but mechanical contraction is absent. Thus, two electrical refractory periods are coupled, thereby increasing the total electrical refractory period of the heart and making

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*Sinus rhythm; **coupled pacing
the heart refractory to any stimuli for an additional period of time. The result is that the tachycardia is slowed.

The current study, like the studies of Lopez et al.,1,2 Chardack et al.,3 and Braunwald et al.,4,5 shows that appropriately designed electrical stimuli are capable of slowing the mechanical rate of the heart. Paired pulse stimulation has also been shown to increase the myocardial contractile force.6 Unlike the paired pulse method of slowing the heart, this technic utilizes a short duration single electrical pulse (3 msec.) and incorporates the inherent advantage of atrial contraction. In addition, the heart rate during coupled pacing is reduced to one-half the heart rate during tachycardia, since the pacemaker best blocks the next auricular beat from activating the A-V junction, whereas with paired electrical stimuli an A-V dissociation is created, which in turn is reduced, thereby lowering the original heart rate, but not to one-half.

It is necessary to prevent firing of the pacemaker for a given period (300-500 msec.) after it has been triggered by the synchronizer. If this is not done, the QRS complex produced by the pacemaker after the pre-set delay may trigger the synchronizer and initiate another QRS complex, which in turn would repeat the cycle, thereby inducing a tachycardia. Such a sequence is readily broken up by insuring that the pacemaker remains non-responsive to stimuli for an adequate period after firing.

The results of this study demonstrate statistically significant increases in left ventricular pressure and aortic blood flow with coupled pacing. These observations suggest that coupled pacing may have clinical applications in certain tachycardias. While the technic has many apparent possibilities, it is at present an experimental procedure and it should be pointed out that there may be some hazards with technics of this order. Stimulation occurs in the vulnerable period of the heart, and under some situations, ventricular fibrillation can be induced. However, it is hoped that with further study in this area, means of avoiding this difficulty will become apparent.

**SUMMARY**

The mechanical contractile rate of the heart can be slowed by appropriately delayed single electrical stimuli applied to the ventricles. Slowing of the heart by this means leads to statistically significant increases in left ventricular pressure and ascending aortic blood flow. The technic of coupled pacing incorporates the inherent advantage of auricular contraction, not obtainable when the heart is slowed by paired
pulses. Coupled pacing may be applicable and beneficial in certain patients where a slower mechanical rate is desired.

**Resumen**

El ritmo de la contracción cardiaca puede ser retardado por estímulos eléctricos aplicados a los ventrículos. El retardo cardíaco obtenido de esta manera produce un aumento de la presión intraventricular y del flujo aórtico ascendente. La técnica de la regulación acoplada tiene la ventaja inerente de la contracción auricular, lo que no se obtiene cuando el corazón es retardado mediante pulsos pareados. La regulación acoplada puede ser aplicable y beneficiosa en casos en que se requiere un retardo mecánico del ritmo.

**Resumé**

Le taux de la contraction mécanique du coeur peut être ralenti par l'application aux ventricules de stimuli électriques retardés de manière convenable. Le ralentissement cardiaco par ce moyen amène une augmentation statistiquement significative de la pression ventriculaire gauche et du débit sanguin dans l'aorte ascendante. La technique de la stimulation couplée à l'avantage de la contraction auriculaire, que l'on obtient pas lorsque le coeur est ralenti par des doubles impulsions. La stimulation couplée peut s'appliquer de manière bénéfique à certains maladies pour lesquels on désire en taux plus lent.

**Zusammenfassung**

Die mechanische Kontraktrationsrate des Herzens kann herabgesetzt werden durch an die Ventrikel angesetzte entsprechende verzögerte elektrische Einzelreize. Eine Verlangsamung des Herzens kann auf diese Weise führt zu einem statistisch signifikant Anstieg des Druckes im linken Ven-

**Referencias**


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**ACUTE PULMONARY ATELECTASIS**

The authors describe the angiographic patterns in two cases of acute pulmonary atelectasis occurring during angiographic examination performed under general anesthesia with intubation. Contrary to what is known during chronic pulmonary atelectasis, the lung affected by acute atelec-

tasis is characterized by the presence of tortuous and dilated arterial vessels.


**EFFECT OF PLEURAL FLUID ON DIAPHRAGM**

Changes in the position and contour of the hemi-

diaphragm resulting from pleural fluid frequently explain the apparent lack of correlation between the roentgenographic appearance of the chest, the volume of fluid withdrawn by thoracentesis, and the clinical status of the patient. Severe depression, flattening and fixation or inversion and paradoxic excursions of the hemidiaphragm are believed to represent radiographic signs of impairment of pulmonary function beyond that usually occurring with large pleural effusions. These signs frequently cor-

relate better with the clinical status of the patient than the height to which the fluid has risen in the chest, and are believed to represent reliable radiographic indications for thoracentesis. A possible aberration of pulmonary function (pendulum breathing) resulting from paradoxic excursion of the in-

verted hemidiaphragm is discussed.