A cardiac stimulating device provides demand pacing therapy (54), and is controlled by a physiological sensor (58), which provides a signal representing the mechanical contraction of the heart, such as various periods, and the onset thereof, of the cardiac cycle, the accompanying pressures, valve and heart wall movement, cardiac sounds, impedance changes, blood flow and electrical signals, etc.
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CARDIAC PACEMAKER WITH PHYSIOLOGICAL CONTROL AND METHOD

Field of the Invention

This invention relates generally to implantable cardiac stimulating devices, and particularly to implantable stimulating devices that include sensors for detecting mechanical cardiac contractions in a patient. More particularly, this invention is directed toward a demand-type implantable cardiac stimulating device and a method of stimulating the heart which uses a mechanical contraction detector to determine whether to inhibit the generation of stimulation pulses.

Background of the Invention

A pacemaker is an implantable medical device which delivers electrical stimulation pulses to cardiac tissue to relieve symptoms associated with bradycardia -- a condition in which a patient cannot maintain a physiologically acceptable heart rate. Early pacemakers delivered stimulation pulses at fixed intervals in order to maintain a predetermined heart rate, which was typically set at a rate deemed to be appropriate for the patient at rest. The predetermined rate was usually set at the time the pacemaker was implanted, and in more advanced devices, could be set remotely by a medical practitioner after implantation.

Early advances in pacemaker technology included the ability to sense the heart's natural electrical
cardiac activity (also known as the intracardiac electrogram (IEGM), or simply the electrogram (EGM)). The electrical cardiac signals include, for example, P-waves and R-waves corresponding to the depolarization of the atria and the ventricles, respectively.

The ability to sense the intracardiac electrogram (hereinafter referred to as "electrogram sensing") led to the development of "demand pacemakers," so named because they deliver stimulation pulses only as needed by the heart. Demand pacemakers are capable of detecting spontaneous electrical cardiac signals which occur within a predetermined time period (commonly referred to as the "alert period"). Immediately following a pacemaker stimulus or a sensed cardiac signal, an escape interval is started. The escape interval is comprised of a refractory period and an alert period. During the refractory period, the pacemaker ceases to be responsive to incoming signals. It is during the alert period that a naturally occurring electrical cardiac signal can be detected and a stimulation pulse will be inhibited. On the other hand, if no electrical cardiac signal is detected during the alert period, a demand pacemaker will generate a stimulation pulse at the end of the escape interval.

Demand pacemakers proved to be extremely beneficial in that they successfully reduced or eliminated seriously debilitating and potentially lethal effects of bradycardia in many patients. The demand pacemaker has the advantages of conserving current drain by allowing the patient's natural rhythm to occur and, more importantly, of potentially preventing the acceleration of an arrhythmia by not competing with the patient's natural rhythm.
Pacemakers that perform electrogram sensing for purposes of demand pacing present several drawbacks, particularly relating to signal processing, which have proven difficult to overcome. For example, it is extremely difficult to accurately sense the IEGM in the presence of noise or electromagnetic interference. Since the most common method of electrogram sensing includes using a comparator with a narrow bandpass filter (e.g., 40-80 Hz), a burst or pulse of high frequency noise or EMI will falsely inhibit the pacemaker (i.e., a burst of noise is effectively envelope detected, resembling a single pulse, thereby mimicking an R-wave).

Residual polarization effects, as a result of a stimulation pulse being delivered, also affect the electrogram sense amplifier's ability to sense properly. Residual polarization effects (commonly known as "afterpotentials") occur in the immediate vicinity of the pacing electrodes and depend largely on the implanted electrode's composition, size and surface area, in addition to, the energy content of the stimulation pulse. Indeed, most pacemakers enter a blanking period immediately after a stimulation pulse is delivered, during which time the sensing circuitry is deactivated for the specific purpose of avoiding undesirable sensing of afterpotentials. However, if the blanking interval is too short, or if the afterpotential is unduly large, the pacemaker will sense the afterpotential (also known as oversensing) and falsely inhibit the pacemaker.

Once implanted, a lead undergoes numerous changes at the point of contact with the cardiac tissue (also known as the site of injury). The body reacts to the lead electrode as it would any other foreign body by building up a capsule of fibrous, scar tissue around
the lead electrode, effectively encapsulating and, advantageously, anchoring it in place. However, due to the buildup of the fibrous capsule, changes in sensing thresholds can occur abruptly during the acute phase (that is, during at least the first month post-implant), and gradually during the chronic phase of the lead. If the electrogram sense amplifier does not have an adequate safety margin, the pacemaker will not sense a true cardiac signal (also known as undersensing).

Cross-sensing of electrical signals can also arise whenever a signal from one chamber is so large that it is sensed in the opposite chamber. For example, "far-field" R-waves originating from the ventricles are often observed on the atrial lead. Also, large output stimuli from one chamber may be cross-sensed by the other chamber. In either case, false inhibition may result.

Other shortcomings of electrogram sensing arise from the electrode configurations available. The most common electrode configurations comprise "unipolar-tip," "unipolar-ring" and "bipolar." Unipolar-tip sensing occurs from the distal tip electrode to the pacemaker case. Unipolar-ring sensing occurs from the distal ring electrode to the pacemaker case. Bipolar sensing occurs between the distal tip and the distal ring electrode. Although it is generally true that unipolar-tip IEGM signals are the largest in amplitude, this may not always be true. If the lead is poorly positioned or located near a myocardial infarct, unipolar-ring or bipolar sensing may have larger amplitudes. Another disadvantage of unipolar sensing (either from the tip or the ring) is that myopotential signals generated by the pectoral muscle can falsely inhibit the pacemaker. In such cases,
bipolar sensing is often chosen since it inherently has superior noise immunity to external interference signals, such as, myopotentials, EMI and noise. However, bipolar sensing can also suffer from low amplitude signals if positioned poorly or oriented perpendicular to the electrical wavefront (effectively nulling the difference signal).

Furthermore, arrhythmias, such as low amplitude fibrillation, are also difficult to sense electrically unless the implantable stimulation device incorporates automatic gain adjustment, or other types of automatic calibration and signal processing routines. But, again, EMI and noise can corrupt these automatic routines.

It is also known that pharmacological therapy, exercise and diurnal variations will also affect the amplitude of the IEGM. Pacemakers which perform electrogram sensing must therefore make certain accommodations (e.g., additional blanking/refractory circuitry, discharge circuitry, software checking, etc.) to overcome the difficulties described above.

What is needed, therefore, is an improved pacemaker that uses a reliable signal to determine whether or not to inhibit the pacemaker, without depending on the patient's IEGM.

**Summary of the Invention**

The disadvantages and limitations of the previously known pacemakers that perform electrogram sensing as described above are overcome by the present invention. With this invention, a mechanical contraction detector is provided which senses natural and evoked cardiac contractions and couples this information to control logic and timing circuitry.
within the implantable cardiac stimulating device to provide demand pacing therapy.

As used herein, the term "depolarization" refers to the propagation of electrical activity in the cardiac muscle cells. That is, during each cardiac cycle, an action potential is spontaneously generated in the S-A node, or may be artificially generated by a stimulation pulse. The action potential then propagates through both atria, the A-V bundle and into the ventricles. Thus, a P-wave is caused by the propagation of the action potential as the atria depolarize prior to contraction, and an R-wave is caused by the propagation of the action potential as the ventricles depolarize prior to contraction.

The physical or "mechanical contraction" of the cardiac muscle begins a few milliseconds after this action potential begins and continues to contract for a few milliseconds after the action potential ends. Thus, by employing a mechanical contraction detector, the present invention allows the implantable cardiac stimulating device to provide demand pacing therapy without the need for an electrogram sense amplifier. Alternatively, the mechanical contraction detector can be used as the primary sensor, with an electrogram sense amplifier as a secondary sensor or for purposes of redundancy or waveform analysis.

The functional result of the mechanical contraction-based demand pacing system of the present invention is similar to the prior art electrogram-based demand pacing system, but does not suffer from the problems of electrogram sensing. That is, the present invention delivers stimulation pulses to the patient's heart in the absence of mechanical contractions, and inhibits stimulation pulses in the presence of detected mechanical contractions. The
present invention includes single-chamber pacing (e.g., VVI or AAI modes), dual-chamber pacing (e.g., DDD, DDI mode, etc.), and atrial tracking modes (e.g., VDD mode).

In the preferred embodiment, the present invention comprises a cardiac wall motion sensor to detect the physical or mechanical contraction of the heart. In this embodiment, the present invention preferably includes an accelerometer placed on one of a patch lead, a myocardial lead or an endocardial lead. The output of the accelerometer can be used directly to detect cardiac wall motion, or can be processed to provide velocity (corresponding to contractility and sympathetic tone) or cardiac wall displacement (corresponding to blood flow and stroke volume).

Other direct mechanical sensors that may be used to detect cardiac contractions include, for example, stroke volume (impedance sensors), thermodilution, heart sounds, ventricular pressure, atrial pressure, or aortic pressure, etc.

Many of the advantages of the present invention stem from the use of a signal representative of mechanical contraction, instead of the patient's IEGM, to determine the true condition of the patient's heart. Thus, the mechanical contraction detectors used in accordance with the present invention are not susceptible to interference from pulse-induced afterpotentials, noise, electromagnetic interference, cross-sensing, undersensing, oversensing, drugs, exercise, arrhythmias or diurnal variations.

The present invention further includes a method of stimulating the heart using a mechanical contraction detector for detecting natural cardiac contractions and inhibiting the output of stimulation
pulses in the presence of a naturally occurring natural cardiac contractions.

Finally, all of the aforesaid advantages and objectives are achieved without incurring any substantial relative disadvantage. It will therefore be perceived that the advantages of the present invention result in a demand pacemaker providing a reliable inhibition mode based on a mechanical contraction sensor, thereby providing a higher quality of life for the patient, making the method of the present invention a highly desirable enhancement to implantable cardiac pacemaker therapy.

**Brief Description of the Drawings**

The above and other advantages of the present invention will be apparent upon consideration of the following detailed description, taken in conjunction with the accompanying drawings, in which like reference characters refer to like parts throughout, and in which:

**Fig. 1** is a block diagram of an implantable cardiac stimulating device that is capable of providing demand pacing therapy based on mechanical contraction in accordance with the principles of the present invention;

**Fig. 2** shows a variety of locations for a mechanical contraction detector in combination with a variety of leads in contact with a patient's heart;

**Fig. 3** depicts a simplified flow diagram for controlling the calibration of the adaptive bandpass filter;

**Fig. 4** illustrates how intrinsic and evoked R-waves are observed on a surface electrocardiogram (ECG), on an intracardiac ventricular electrogram (VTR
EGM), and at the output of the cardiac wall motion sensor, along with basic timing diagrams; and

Fig. 5 is an illustration of the relationship of the surface electrocardiogram (ECG) with other physiological events that occur during the cardiac cycle.

**Detailed Description of the Preferred Embodiment**

The following description is of the best mode presently contemplated for practicing the invention. This description is not to be taken in a limiting sense but is made merely for the purpose of describing the general principles of the invention. The scope of the invention should be ascertained with reference to the issued claims.

In Fig. 1, a block diagram is shown representing an implantable cardiac stimulating device 50 which performs mechanical contraction detection in accordance with the principles of the present invention. The implantable cardiac stimulating device 50, as described below, is a combined pacing and cardioverting/defibrillator device capable of providing demand pacing therapy as well as higher energy therapies, such as cardioversion and defibrillation shocks. However, mechanical contraction-based demand pacing can be easily implemented in a simpler device, such as a dedicated demand pacemaker, in view of the description below.

The implantable cardiac stimulating device 50 delivers therapeutic electrical stimulation to a patient's heart (not shown) through a pacing lead 52 and a shocking lead 62, proximal ends of which are connected to the implantable cardiac stimulating device 50, and distal ends being in contact with a
selected region of cardiac tissue (not shown). In the embodiment shown in Fig. 1, at least one pacing lead 52 is used to deliver stimulation pulses generated by a pulse generator 54 (which may be conventional) in accordance with instructions provided by a control logic and timing circuitry 56. Although only one pacing lead 52 is shown, it is understood that one of skill in the art could adapt the implantable stimulation device 50 to have a second lead in the atrium for dual-chamber demand pacing. Furthermore, the shocking lead 62 may be a single lead with two spaced-apart endocardial electrodes, or may take the form of two shocking leads attached to the myocardium.

In one mode of operation, the implantable cardiac stimulating device 50 operates as a demand pacemaker, in that the delivery of a stimulation pulse provided by the pulse generator 54 may be inhibited by a spontaneous cardiac contraction which occurs during an alert period determined by the control logic and timing circuitry 56. The mechanical contraction detector 58 provides a control signal to the control logic and timing circuitry 56, indicative of whether a mechanical cardiac contraction has occurred. The control logic and timing circuitry 56 determines if a sensed mechanical contraction occurred within the alert period, and if so, inhibits the pulse generator 54 from generating a stimulation pulse. Otherwise, stimulation pulses continue to be administered at the programmed rate.

In the preferred embodiment, the mechanical contraction detector 58 is a cardiac wall motion sensor. Although the mechanical contraction detector 58 is shown in Fig. 1 as a separate element (e.g., a separate "sensor only" lead), in the preferred
embodiment, the mechanical contraction detector 58 is mechanically coupled to the patient's heart by a lead that is also used to administer therapeutic electrical stimulation (e.g., either the pacing lead 52 or the shocking lead 62). Furthermore, while the pacing lead 52 and the shocking lead 62 are shown as physically separate leads, their respective electrodes may also be provided by a single lead (not shown) which includes a pacing, sensing, and shocking electrodes, as well as, a mechanical contraction detector. One advantage of using such leads is that the lead which contains the cardiac wall motion sensor may contain at least one (and perhaps all) of the electrodes that are used to deliver stimulation pulses, thereby advantageously reducing the number of leads that need to be implanted in the patient's body.

Leads containing cardiac wall motion sensors that are particularly well-suited to be used in the present invention are described in commonly assigned, copending U.S. Patent Application Serial No. 08/091,636, filed 7/14/93, entitled "IMPLANTABLE LEADS INCORPORATING CARDIAC WALL MOTION SENSORS AND METHOD OF FABRICATION AND A SYSTEM AND METHOD FOR DETECTING CARDIAC ARRHYTHMIAS USING A CARDIAC WALL MOTION SENSOR SIGNAL," which is hereby incorporated by reference in its entirety. That patent application discloses an accelerometer-based cardiac wall motion sensor in a variety of implantable leads, including endocardial leads, myocardial active-fixation leads, and epicardial patch electrodes.

In Fig. 2, a variety of locations for a cardiac wall motion sensor are shown in combination with a variety of leads in contact with a patient's heart 78. For example, in a single-chamber pacing system (such as a VVI or an AAI pacing system), a mechanical
contraction detector 58 (Fig. 1) could be employed at the distal end 82 of a ventricular endocardial lead 80, the distal end 92 of an atrial endocardial lead 90, or at the distal ends 98, 99 of an epicardial lead, such as, patch leads 94 or 96, respectively. In a dual-chamber pacing system, the preferred location would be the distal ends 82 and 92 of the ventricular and atrial leads 80 and 90, respectively.

One or more mechanical contraction detectors could also be located on a lead so that both the atrial and the ventricular physical muscle contractions can be detected. For example, in an atrial tracking pacing system (i.e., a VDD pacemaker) a single lead is desirable to minimize the number of leads implanted. Although it is believed that a mechanical contraction detector 58 located at the distal end 82 of the ventricular lead 80 could pick up atrial contractions, it may be desirable to move the mechanical contraction detector 58 below (or near) the A-V valve, e.g., at location 84. Alternatively, it may be necessary to place two mechanical contraction detectors on the single ventricular lead, e.g., by placing one detector in the atrium at location 86 and one detector in the ventricle at location 82 or 84.

It is also understood that conventional electrogram sensing could be added as a redundant sensor or for waveform analysis. For example, ring electrodes could be added to the distal ends 82 or 92 of the ventricular or atrial lead, respectively, or a pair of electrodes could be located floating in the atrium at the proximal end (e.g., at location 86) of the ventricular lead 80.

It is also understood that electrogram sensing could be used in combination with a mechanical contraction sensor. For example, ring electrodes could
be located in one chamber of the heart, either the ventricle or the atrium, and a mechanical contraction detector 58 could be used in the other chamber.

Indeed, a great variety of lead configurations may be used in accordance with the principles of the present invention, so as to not diminish the flexibility that a medical practitioner normally has when selecting leads that meet the needs of a particular patient.

As mentioned above, other mechanical contraction detectors that may be used to detect physical cardiac contractions include, for example, stroke volume, thermodilution, heart sounds, ventricular pressure, atrial pressure, or aortic pressure, etc. These other sensors will be discussed below in conjunction with FIG. 5.

In the preferred embodiment shown in Fig. 1, signals from the mechanical contraction detector 58 are received by a preamplifier and bandpass filter 60 within the implantable cardiac stimulating device 50. Preferably, the bandpass filter is an adaptive bandpass filter with is tunable to different center frequencies and/or bandwidths so that noise and other artifact signals, such as valve sounds, can be eliminated. During a calibration phase (described in detail below in conjunction with Fig. 3) the preamplifier and bandpass filter 60 are tuned by the control logic and timing circuitry 56 to select relatively large amplitude, high slew rate signals that are associated with coherent cardiac contractions, and to reject signals caused by patient movements.

The preamplifier and bandpass filter 60 may further process the signals using conventional techniques, such as noise filtering, averaging,
integrating or double-integrating. The integral and double-integral of, for example, cardiac wall motion accelerations correspond to velocity and cardiac displacement, as described in commonly assigned, copending U.S. Patent Application Serial No. 08/154,800, filed 11/16/93, entitled "SYSTEM AND METHOD FOR DERIVING HEMODYNAMIC SIGNALS FROM A CARDIAC WALL MOTION SENSOR SIGNAL," which is hereby incorporated by reference in its entirety.

The filtered signals are then provided as the input to a detector circuit 62 and the resulting output indicative of cardiac contractile activity is provided to the control logic and timing circuitry 56. The detector circuit 62 could be a simple threshold detector or, as in the preferred embodiment, it could be a sample-and-hold circuit followed by an analog-to-digital converter and a comparator (not shown), as is known in the art. The control logic and timing circuitry 56 executes a program (the program code being stored in the memory 64) to perform demand pacing based on the mechanical contraction detector 58.

The control program of Fig. 3 illustrates a simplified flowchart to automatically calibrate the bandpass filter 60 (shown in Fig. 1). In the preferred embodiment, the bandpass filter 60 will be calibrated using an "evoked" contraction, based on the assumption that an intrinsic contraction has approximately the same response. To ensure capture, the calibration routine paces at the maximum amplitude which, in most cases will cause capture (barring, of course, any severe arrhythmia, e.g., fibrillation). The advantage of using the evoked contraction is that the system will calibrate to signals representative of cardiac contractions caused by the application of the
maximum amplitude stimulation pulse, and not, for example, body motion.

The calibration routine begins at start 100, which is followed by step 102, wherein the pulse generator 54 is adjusted to generate stimulation pulses having a predetermined maximum amplitude (e.g., 5 volts). The predetermined maximum amplitude is stored as a parameter in the memory 64. At the step 104, the pulse generator 54 generates a stimulation pulse having the maximum amplitude at a rate which is programmed to be faster than the patient's intrinsic heart rate. Both the number of pulses and the pulse rate may be stored as parameters in the memory 64. Since the rate is set to be faster than the patient's natural rhythm, a contraction should be detected by the mechanical contraction detector 58 soon after the stimulation pulse is delivered. At the step 106, the control logic and timing circuitry 56 tunes the preamplifier and bandpass filter 60 so that an expected excursion appears in the signal provided by the mechanical contraction detector 58 within a predetermined period of time following each maximum amplitude pacing pulse delivered at the step 104.

At the test 108, the control logic and timing circuitry 56 determines whether the calibration phase has been completed. Under most circumstances, sixty maximum amplitude stimulation pulses should be sufficient to properly calibrate the preamplifier and bandpass filter 60; however, this number can be stored as a parameter in the memory 64, and can be adjusted by the medical practitioner as needed. Until the calibration phase is completed, the program repeatedly loops back to the step 104, where the control logic and timing circuitry 56 causes the pulse generator 104
to generate another maximum amplitude stimulation pulse.

Since the undesirable sensing of pulse-induced after-potentials are not sensed by the mechanical contraction detector, it is an added feature of the present invention that the mechanical contraction detector 58 can detect both evoked (stimulated) contractions and intrinsic (spontaneous) contractions. For a complete description of an autocapture system using, for example, a cardiac wall motion sensor, see commonly assigned, copending U.S. Patent Application Serial No. 05,152,659, filed 11/15/93, entitled "CARDIAC WALL MOTION-BASED AUTOMATIC CAPTURE VERIFICATION SYSTEM AND METHOD," which patent is incorporated herein by reference in its entirety.

Furthermore, the implantable cardiac stimulating device does not have to enter a blanking or a refractory period due to the amplifiers saturating in response to a stimulation pulse, as is done in EGM-based demand pacing. Thus, as explained above, the mechanical contraction-based demand pacemaker does not have to make special accommodations (e.g., separate sensing leads, circuitry to remove polarization or specialized sensing circuitry) in order to discern an evoked R-wave over the pulse-induced afterpotential.

In the preferred embodiment shown in Fig. 1, the implantable cardiac stimulating device 50, as mentioned above, may be capable of providing higher energy shock therapies to interrupt more severe cardiac arrhythmias. For example, cardioversion shocks may be administered to convert ventricular tachycardia (VT), and defibrillation shocks may be administered to convert ventricular fibrillation (VF). Like pacing therapy, higher energy shock therapies are administered under the control of the control logic
and timing circuitry 56. Advantageously, the control logic and timing circuitry 56 may receive a signal indicative of whether the patient is experiencing a severe arrhythmia (e.g., VT or VF) based on the rate of the signals detected by the mechanical contraction detector 58, without relying on the IEGM signal. If such an arrhythmia is detected, the control logic and timing circuitry 56 causes the high energy shock generator 70 (which may be conventional) to generate a therapeutic shock of an appropriate energy content to convert the particular type of arrhythmia detected. These higher energy shocks are generated and delivered to the patient's heart through at least one shocking lead 62.

In fact, the present invention can discriminate between arrhythmias based on the behavior of the mechanical contraction detector 58. Discrimination of cardiac arrhythmias base on, for example, cardiac wall motion sensor signals is also disclosed in the above-incorporated U.S. Patent Application No. 08/154,800. The '800 patent application discloses a variety of hemodynamic signals that can be derived from a cardiac wall accelerometer sensor, including contractility, sympathetic tone, blood flow, fluid displacement and stroke volume. In addition, the signals provided by the accelerometer-based cardiac wall motion sensors may be used by the control logic and timing circuitry 56 as a substitute for, or in combination with, the patient's IEGM for detecting hemodynamically stable and unstable tachycardias, in addition to detecting fibrillation.

The manner by which the implantable cardiac stimulating device 50 delivers pacing therapy and higher energy shock therapies is controlled by the control logic and timing circuitry 56 in accordance
with parameters stored in a memory 64. Many of these parameters are known in the art (e.g., escape interval, refractory period, cardioversion shock energy, defibrillation shock energy, etc.), and they may be programmed by a medical practitioner using a programming unit 72 that communicates with the control logic and timing circuitry 56 through a telemetry circuit 66.

The implementation of the control logic and timing circuitry 56, as is well known in the art, could be a microprocessor, a state machine or dedicated logic circuitry. For example, U.S. Patent Nos. 4,390,022 and 4,404,972, both disclose a microprocessor based implantable stimulation system, including high energy shocking systems. U.S. Patent Nos. 4,712,555 and 3,595,242 illustrate state machines and dedicated circuitry, respectively, for controlling a demand pacemaker. U.S. Patent Nos. 4,390,022; 4,404,972; 4,712,555; and 3,595,242 are all incorporated herein by reference in their entirety.

In Fig. 4, the manner by which the control logic and timing circuitry 56 evaluates the signal provided by the mechanical contraction detector 58 to control the implantable stimulation device may be fully appreciated by reference to the waveforms shown.

As illustrated in Fig. 4, a surface ECG waveform 200, a ventricular electrogram (VTR EGM) waveform 300, and a cardiac wall motion signal 400 are shown synchronously in time.

The surface electrocardiogram (ECG) waveform 200 depicts two intrinsic beats, followed by two evoked, or stimulated, beats. The intrinsic beats are comprised of a P-wave 202 and an R-wave 204. The waveforms in Fig. 4 assume that an atrial stimulation lead has been implanted in the right atrium to provide
dual-chamber pacing. Thus, the evoked beats are comprised of an atrial stimulation pulse 220, followed by an evoked P-wave 222, and a ventricular stimulation pulse 224 followed by an evoked R-wave 226.

The ventricular electrogram waveform (VTR EGM) 300 is included for illustration purposes to show what an intracardiac electrogram sense amplifier of the prior art would see. It should be noted that P-waves are typically not seen on the ventricular electrogram.

The waveform 400 illustrates the output of the preferred mechanical contraction detector, i.e., a cardiac wall motion sensor. Using a cardiac wall motion sensor located, for example, in the distal end of a ventricular lead, it may possible to detect both the atrial contraction (e.g., at excursion 402) and the ventricular contraction (e.g., at excursion 404). (This would have the advantage of permitting an atrial tracking mode with a single pass lead, thereby eliminating a lead in the atrium.) In the event that atrial contractions could not be sensed in all patients on a single ventricular lead or that atrial stimulation was required, the present invention could be adapted to include a mechanical contraction detector, such as the cardiac wall motion sensor, into an atrial lead.

Below the waveform 400 are timing intervals generated by the control logic and timing circuitry 56 to control the A-V delay 500 and the atrial escape interval 600. Briefly, the A-V delay is the desired conduction time between the atrium and the ventricle. An R-wave which occurs during the A-V delay will inhibit a stimulation pulse in the ventricle. In general, a solid dot and the end of an interval is used to denote a sensed contraction (e.g., at 502) and an arrow denotes a timed-out event without sensing a.
cardiac contraction (e.g., 504). The atrial escape interval 600 comprises a refractory period 602, 612, 622 (during which time the tissue is deemed incapable of stimulation) and an alert period 604, 614 or 624 (during which time the mechanical contraction detector is enabled).

In operation, a P-wave 202 occurs during the alert period 604 of the atrial escape interval 600 (denoted by a solid dot at 606) and elicits an excursion 402 from the cardiac wall motion sensor, thereby initiating an A-V delay. An R-wave 204 occurs during the A-V delay (denoted by the solid dot at 502) and elicits an excursion 404 from the cardiac wall motion sensor, thereby resetting the atrial escape interval 600 at 610. (For illustration purposes, the resetting of the atrial escape interval 600 is shown to occur at the very onset of the excursion 404, however it may be necessary to adjust the timing slightly based on the actual level of detection and the delay in detecting the excursion.) Next, the atrial escape interval 600 times-out (denoted by the arrow at 608) without sensing another intrinsic P-wave. If an atrial pacing lead has been implanted, a stimulation pulse 220 could be provided in the atrium which elicits the excursion 422 from the cardiac wall motion sensor, indicative of an evoked atrial contraction. The A-V delay times-out (denoted by the arrow at excursion 504) without seeing an R-wave. Therefore, a stimulation pulse 224 is provided in the ventricle which captures the heart (at 226 and 326) and is detected by excursion 426 of the cardiac wall motion sensor.

To some extent the present invention may be able to detect heart sounds. It is believed that excursion 404 also corresponds to the first cardiac heart sound,
excursion 406 corresponds to the second heart sound, and excursion 408 corresponds to the third heart sound. In one embodiment, it may be desirable to filter out some of these heart sounds (406 and 408) with the preamplifier & bandpass filter 60 (Fig. 1), so that only the contractile signal is available as an input to the detector 62 (Fig. 1). Alternatively, it may be desirable to extend the refractory period 602 (Fig. 3) so that the heart sounds (406 and 408) can be ignored and to enhance detection of the atrial excursion 402 (or 422).

In FIG. 5, the surface electrogram is shown in its relationship with other physiological signals that may be used for the mechanical contraction detector 58. It can be seen from Fig. 5 that any mechanical sensor which can detect the occurrence of a true ventricular contraction, such as, a sensor which can detect systole, the onset of systole (e.g., the period of isometric contraction), or the period of ejection (e.g., the period when blood is ejected from the ventricles) would be suitable.

For example, in one embodiment, it may be desirable to detect the closure of the A-V valve, since the closure of the A-V valve closely corresponds to the R-wave and the onset of systole. Closure of the A-V valve can be achieved by detecting the first heart sound using an acoustic sensor or with a cardiac motion sensor which detects cardiac wall accelerations, as described above, or by using an impedance measuring sensor to detect a change in impedance as the A-V valve closes (or when the aortic valve opens). It can also be seen in Fig. 4 that a sensor which can detect a sudden increase in ventricular, atrial or aortic pressure could also be used to detect the onset of systole. Also, the peak
atrial pressure could be used to detect the onset of systole.

During the portion of systole (contraction) known as the period of ejection, left ventricular pressure forces the semilunar valves (i.e., the aortic and the pulmonary valves) to open and blood rushes out of the ventricles. Thus, any mechanical sensors which could detect the blood flow out of the ventricles also be a reliable detector of ventricular contractions. For example, blood flow sensors that may be used in the present invention would include stroke volume (e.g., impedance), thermodilution, cardiac wall displacement, cardiac wall accelerations or cardiac wall velocity sensors.

Measurement of stroke volume using impedance for purposes of modulating the rate of a pacemaker is known in the art, see for example U.S. Patent Nos. 4,535,774 (Olsen) or 4,686,987 (Salo), which patents are hereby incorporated by reference.

Implantable pressure sensors for treating a malfunctioning heart are also well known in the art, see for example, (Cohen) U.S. Patent No. 4,899,751, which patent is incorporated herein by reference.

Measurement of cardiac velocity, cardiac displacement, cardiac accelerations are described in the above-incorporated by reference U.S. Application Serial No. 08,154,800, filed 11/16/93.

Piezoelectric pressure sensors for detecting the opening and closing of heart valves, stroke volume, and ejection time for purposes of modulating the rate of a pacemaker are also known in the art, see for example, U.S. Patent Nos. 4,600,017 (Schroeppele) or 4,802,481 (Schroeppele) which patents are hereby incorporated by reference.
Piezoresistive pressure transducers are also known for determining ventricular pressure, duration of contraction, ejection time, blood volume and the detection of the pulmonary valve opening and closing, see for example U.S. Patent No. 4,730,619, which patent is incorporated herein by reference.

Isovolumic contraction time is also known to be used for modulating the rate of a pacemaker. Isovolumic contraction time is determined by detecting the onset of mechanical activation and the onset of ejection, as shown in U.S. Patent No. 5,168,869 (Chirife), which patent is incorporated herein by reference.

Thermodilution sensors are well known in the catheter art, see for example, U.S. Patent No. 3,995,623 (Blake et al.), which patent is hereby incorporated by reference.

It can be seen from Fig. 5 that mechanical sensors which could detect the opening/closing of the semilunar valves would also be a reliable detector of ventricular contractions, i.e., either the aortic valve opening and closing or the A-V valve closing and opening. It can also be seen from Fig. 5 that the first and second heart sounds correspond to the opening and closing of the aortic and A-V valves, thereby providing an indication of blood flowing during the period of ejection. As can be seen in Fig. 4, the first and second heart sounds 404 and 406, respectively, are available from the cardiac wall motion sensor of the present invention.

A peak ventricular or aortic pressure sensor could also be used to detect the blood flow. In these embodiments, some compensation would be required in the timing circuitry 56 for the associated delay time in detecting a peak value.
As mentioned above, the limitations of prior art IEGM-based inhibition systems are overcome by the present invention. Many of the advantages of the present invention are achieved through the use of a mechanical contraction detector which is responsive to contractile activity of the patient's heart. The mechanical contraction detector provides a true indication of physical cardiac contractions, which may be processed to determine if a particular stimulation pulse or a naturally conducted cardiac signal evoked a cardiac contraction.

Thus, a mechanical contraction-based demand pacing system is provided. One skilled in the art will appreciate that the present invention can be practiced by other than the described embodiments, which are presented for purposes of illustration and not of limitation, and the present invention is limited only by the claims which follow.
CLAIMS

What is Claimed is:

1. An implantable cardiac stimulating device for generating stimulation pulses to cardiac tissue through an implantable stimulation lead, the device comprising:
   pulse generating means for generating the stimulation pulses;
   mechanical detection means for detecting a physical cardiac muscle contraction; and
   control means, responsive to the mechanical detection means, for inhibiting the pulse generating means from generating a stimulation pulse when a cardiac muscle contraction is detected.

2. The implantable cardiac stimulating device of Claim 1, wherein the mechanical detection means further comprises:
   means for detecting the physical cardiac muscle contraction which occurs as a result of an electrical depolarization of the cardiac muscle tissue.

3. The implantable cardiac stimulating device of Claim 1, wherein the mechanical detection means comprises:
   means for detecting the physical cardiac muscle contraction on a beat-by-beat basis.
4. The implantable cardiac stimulating device of Claim 1, wherein the mechanical detection means comprises:

means for detecting a period of the patient's cardiac cycle corresponding to ventricular contraction.

5. The implantable cardiac stimulating device of Claim 4, wherein the means for detecting ventricular contraction comprises:

means for detecting a period of the patient's cardiac cycle corresponding to the onset of ventricular systole.

6. The implantable cardiac stimulating device of Claim 5, wherein the means for detecting the onset of ventricular systole comprises:

means for detecting a period of the patient's cardiac cycle corresponding to the period of isometric contraction.

7. The implantable cardiac stimulating device of Claim 5, wherein the means for detecting the onset of ventricular systole comprises:

means for detecting a sudden rise in the ventricular pressure.

8. The implantable cardiac stimulating device of Claim 5, wherein the means for detecting the onset of ventricular systole comprises:

means for detecting a sudden rise in the atrial pressure.
9. The implantable cardiac stimulating device of Claim 5, wherein the means for detecting the onset of ventricular systole comprises:

means for detecting a sudden rise in the aortic pressure.

10. The implantable cardiac stimulating device of Claim 5, wherein the means for detecting the onset of ventricular systole comprises:

means for detecting closure of the heart's A-V valve.

11. The implantable cardiac stimulating device of Claim 10, wherein the means for detecting the closure of the heart's A-V valve comprises:

impedance measuring means for detecting a change in impedance corresponding to the heart's A-V valve closing.

12. The implantable cardiac stimulating device of Claim 10, wherein the means for detecting the closure of the heart's A-V valve comprises:

means for detecting a first heart sound.

13. The implantable cardiac stimulating device of Claim 12, wherein the means for detecting the first heart sound comprises:

a cardiac wall accelerometer in contact with ventricular tissue.
14. The implantable cardiac stimulating device of Claim 10, wherein the means for detecting the closure of the A-V valve comprises:

pressure sensing means for detecting a peak pressure in the atrium.

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15. The implantable cardiac stimulating device of Claim 14, wherein the means for detecting the onset of systole comprises:

means for detecting the heart's aortic valve opening.

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16. The implantable cardiac stimulating device of Claim 15, wherein the means for detecting the opening of the heart's aortic valve comprises:

means for detecting a change in impedance corresponding to the heart's aortic valve opening.

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17. The implantable cardiac stimulating device of Claim 4, wherein the means for detecting ventricular contraction comprises:

means for detecting a period of the patient's cardiac cycle corresponding to the period of ejection of blood from the ventricles.

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18. The implantable cardiac stimulating device of Claim 17, wherein the means for detecting the period of ejection comprises:

means for detecting blood flow out of the ventricles.
19. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting blood flow comprises stroke volume detection means.

20. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting blood flow comprises a thermodilution sensor.

21. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting blood flow comprises:

   means for detecting a first and a second heart sound.

22. The implantable cardiac stimulating device of Claim 21, wherein the means for detecting the first and second heart sounds comprises:

   means for detecting cardiac wall accelerations corresponding to the first and second heart sounds.

23. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting blood flow comprises:

   means for detecting cardiac wall displacement.

24. The implantable cardiac stimulating device of Claim 23, wherein the means for detecting cardiac wall displacement comprises:

   a cardiac wall accelerometer coupled to the implantable stimulation lead; and
means for twice integrating the output signal of the cardiac wall accelerometer to produce as an output a signal indicative of cardiac wall displacement.

25. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting blood flow comprises:

pressure sensing means for detecting a peak pressure in the aorta.

26. The implantable cardiac stimulating device of Claim 18, wherein the means for detecting the blood flow comprises:

pressure sensing means for detecting a peak pressure in the ventricle.

27. The implantable cardiac stimulating device of Claim 4, wherein the means for detecting ventricular contraction comprises:

means for detecting a change of contractility of the heart tissue.

28. The implantable cardiac stimulating device of Claim 27, wherein the contractility detecting means comprises:

means for detecting a change in cardiac wall velocity.

29. The implantable cardiac stimulating device of Claim 28, wherein the means for detecting cardiac wall velocity comprises:
a cardiac wall accelerometer having an output signal indicative of cardiac wall accelerations; and means for integrating the output signal of the cardiac wall accelerometer to produce as an output a signal representative of cardiac wall velocity.

30. An implantable cardiac stimulating device for generating stimulation pulses to cardiac tissue, the device comprising:

timing means for controlling a plurality of time intervals, the plurality of time intervals including at least a first interval; mechanical detection means for detecting a first type of physical cardiac muscle contraction in a first chamber of the heart; pulse generating means for generating a stimulation pulse in the first chamber of the heart in the absence of the first type of physical cardiac muscle contractions during the first interval; and control means, responsive to the mechanical detection means, for inhibiting the pulse generating means from generating a stimulation pulse in the first chamber of the heart in the presence of the first type of physical cardiac muscle contraction during the first interval.

31. The implantable cardiac stimulating device of Claim 30, wherein the implantable cardiac stimulating device includes at least one implantable stimulation lead, wherein:
the mechanical detection means comprises at least one mechanical contraction sensor located in a distal end of the at least one implantable stimulation lead.

32. The implantable cardiac stimulating device of Claim 30, further comprising:

- electrogram sensing means for sensing electrical cardiac signals in the first chamber of the heart corresponding to an electrical depolarization of the cardiac muscle tissue;
- determining means for verifying the presence of both electrical depolarization and physical cardiac muscle contractions in the first chamber of the heart; and
- wherein the control means includes means for inhibiting the pulse generating means from generating a stimulation pulse in the first chamber of the heart when the determining means verifies the presence of both electrical depolarization and physical cardiac muscle contractions during the first interval.

33. The implantable cardiac stimulating device of Claim 30, wherein:

- the timing means includes means for controlling a second interval;
- the mechanical detection means includes means for detecting a second type of physical cardiac muscle contraction in a second chamber of the heart;
- the pulse generating means includes means for generating stimulation pulses in the second chamber of the heart in the absence of the second
type of physical cardiac muscle contractions during the second interval; and

the control means includes means for inhibiting the pulse generating means from generating a stimulation pulse in the second chamber of the heart in the presence of the second type of physical cardiac muscle contraction during the second interval.

34. The implantable cardiac stimulating device of Claim 33, wherein the implantable cardiac stimulating device includes at least a first and a second implantable stimulation lead, wherein the mechanical detection means comprises:

a first mechanical sensor located within the first implantable stimulation lead implanted in the atrium; and

a second mechanical sensor located within the second implantable stimulation lead implanted in the ventricle.

35. The implantable cardiac stimulating device of Claim 33, further comprising:

electrogram sensing means for sensing electrical cardiac signals in at least one of the first and second chamber of the heart corresponding to an electrical depolarization of the respective cardiac muscle tissue;

determining means for verifying the presence of both electrical depolarization and physical cardiac muscle contractions in the respective chamber of the heart; and

wherein the control means includes means for inhibiting the pulse generating means from
generating a stimulation pulse in the respective chamber of the heart when the determining means verifies the presence of both electrical depolarization and physical cardiac muscle contractions.

36. The implantable cardiac stimulating device of Claim 30, further comprising an electrogram sensing means for sensing electrical cardiac signals in a second chamber of the heart corresponding to an electrical depolarization of the respective cardiac muscle tissue, wherein:

the timing means includes means for controlling a second interval;

the pulse generating means includes means for generating stimulation pulses in the second chamber of the heart in the absence of the second type of physical cardiac muscle contractions during the second interval; and

the control means includes means for inhibiting the pulse generating means from generating a stimulation pulse in the second chamber of the heart in the presence of the second type of physical cardiac muscle contraction during the second interval.

37. An implantable cardiac stimulating device for generating stimulation pulses to cardiac tissue, the device comprising:

mechanical detection means for detecting physical atrial muscle contractions and physical ventricular muscle contractions;

timing means for controlling a plurality of time intervals, the plurality of time intervals
including at least an A-V time interval, the A-V time interval being triggered by the detection of an atrial muscle contraction;

pulse generating means for generating a stimulation pulse in the ventricle at the end of the A-V interval in the absence of a detected ventricular muscle contraction during the A-V interval; and

control means, responsive to the mechanical detection means, for inhibiting the pulse generating means from generating a stimulation pulse in the ventricle in the presence of a ventricular muscle contraction during the A-V interval.

38. The implantable cardiac stimulating device of Claim 37, wherein the implantable cardiac stimulating device includes at least one implantable stimulation lead, wherein the mechanical detection means comprises:

a single mechanical sensor located within the at least one implantable stimulation lead at a location which can sense mechanical contractions from both the atrium and the ventricle.

39. The implantable cardiac stimulating device of Claim 37, wherein the implantable cardiac stimulating device includes at least one implantable stimulation lead, wherein the mechanical detection means comprises:

a first mechanical sensor located within the at least one implantable stimulation lead at a
location which can sense mechanical contractions in the atrium; and
a second mechanical sensor located within the at least one implantable stimulation lead at a location which can sense mechanical contractions in the ventricle.

40. The implantable cardiac stimulating device of Claim 37, further comprising:

electrogram sensing means for sensing electrical cardiac signals in at least one of the atrial and ventricular chamber of the heart corresponding to an electrical depolarization of the respective cardiac muscle tissue;
determining means for verifying the presence of both electrical depolarization and physical cardiac muscle contractions in the at least one of the atrial and ventricular chamber of the heart, the determining means having an output coupled to one of the timing means or the control means so that one of an A-V time interval is triggered by the detection of a verified atrial muscle contraction or a stimulation pulse is inhibited by a verified ventricular muscle contraction.

41. A method for stimulating selected cardiac tissue, the method comprising the steps of:
mechanically detecting physical cardiac muscle contractions from the selected cardiac tissue;
generating stimulation pulses to the selected cardiac tissue in the absence of
detected physical cardiac muscle contractions; and
inhibiting the generation of a stimulation pulse in the presence of detected physical cardiac muscle contractions.

42. The method, as recited in Claim 41, wherein:

the mechanically detecting step includes mechanically detecting physical atrial cardiac muscle contractions and physical ventricular muscle contractions;

the generating step includes generating stimulation pulses to atrial and ventricular cardiac tissue, the stimulation pulses being separated in time by an A-V delay; and

the inhibiting step includes inhibiting the generation of a stimulation pulse in the atrium when an atrial cardiac muscle contraction is detected and inhibiting the generation of a stimulation pulse in the ventricle when a ventricular cardiac muscle contraction is detected.

43. The method, as recited in Claim 41, wherein:

the mechanically detecting step includes mechanically detecting physical atrial cardiac muscle contractions and physical ventricular muscle contractions;

the generating step includes generating stimulation pulses to ventricular cardiac tissue, the stimulation pulses being triggered by the
detection of an atrial cardiac muscle contraction and separated in time by an A-V delay; and
the inhibiting step includes inhibiting the generation of a stimulation pulse in the ventricle when a ventricular cardiac muscle contraction is detected.
Fig. 1

Fig. 2

SUBSTITUTE SHEET (RULE 26)
Fig. 3

Protodiastole
Ejection
Isometric contraction

Isometric relaxation
Rapid inflow
Diastasis
Atrial systole

Aortic valve opens
Aortic valve closes

Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
Electrocardiogram
Phonocardiogram

Fig. 5
Substitute sheet (Rule 26)
INTERNATIONAL SEARCH REPORT

A. CLASSIFICATION OF SUBJECT MATTER
IPC(6) :A61N 1/365
US CL :607/018
According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED
Minimum documentation searched (classification system followed by classification symbols)
U.S. : 607/17-26

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched
NONE

Electronic data base consulted during the international search (name of data base and, where practical, search terms used)
NONE

C. DOCUMENTS CONSIDERED TO BE RELEVANT

<table>
<thead>
<tr>
<th>Category*</th>
<th>Citation of document, with indication, where appropriate, of the relevant passages</th>
<th>Relevant to claim No.</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>6-16, 19-22, 24-29, 33-40, 42, 43</td>
</tr>
<tr>
<td>Y</td>
<td>US, A, 4,899,752, (COHEN), 13 February 1990. See entire document.</td>
<td>7-10, 14, 15, 25, 26</td>
</tr>
<tr>
<td>Y, P</td>
<td>US, A, 5,334,222, (SALO ET AL.), 02 August 1994. See column 6, lines 18-23</td>
<td>12, 13, 21, 22</td>
</tr>
</tbody>
</table>

[X] Further documents are listed in the continuation of Box C. [ ] See patent family annex.

* Special categories of cited documents:
*A* document defining the general state of the art which is not considered to be part of particular relevance
*E* earlier document published on or after the international filing date
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*O* document referring to an oral disclosure, use, exhibition or other means
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*G* document member of the same patent family

Date of the actual completion of the international search
27 MAY 1995

Date of mailing of the international search report
19 JUN 1995

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<th>Category</th>
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