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Lessmeier et al.

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(54) **METHOD FOR OPTIMIZING CRT THERAPY**

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Related U.S. Patent Documents

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A61N 1/365 (2006.01)

(52) **U.S. Cl.**
CPC ***A61N 1/36578*** (2013.01)
USPC **607/18**

(58) **Field of Classification Search**
CPC A61N 1/36578
USPC 607/18
See application file for complete search history.

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(Continued)

Primary Examiner — Eric D. Bertram

(74) *Attorney, Agent, or Firm* — William A. Jeckle

(57) **ABSTRACT**

A method to optimize CRT therapy using ventricular lead motion analysis, either radiographically or with three dimensional electromagnetic mapping, to determine whether focal dyssynchrony is present at baseline, and whether biventricular pacing improves synchronicity and fractional shortening, and if no improvement is evidenced, changing the timing offset, pacing configuration and/or repositioning the ventricular leads to optimize effectiveness of CRT therapy. Various uses of this method include: diagnostic, with temporary leads to determine presence or absence of dyssynchrony and response to pacing; and therapeutic, to guide lead placement and programming during implant of CRT, and to optimize reprogramming of CRT during follow-up.

25 Claims, 10 Drawing Sheets

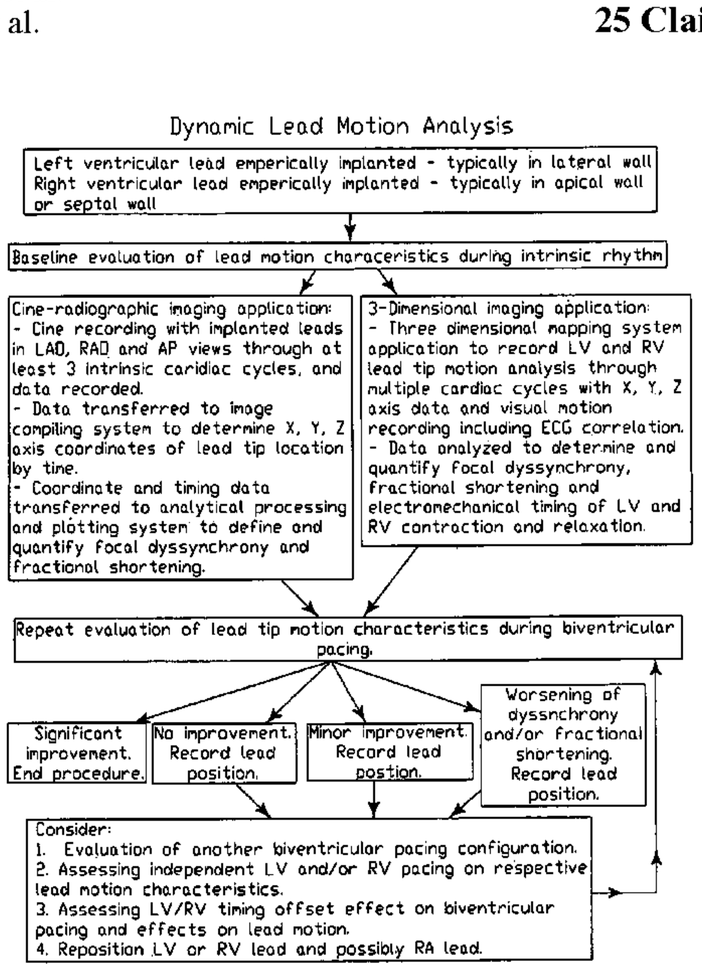

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FIG. 1

RIGHT SIDE
LEFT SIDE
53
48
H
49
30
31
32
35
38
33
34
46
46
37
54
36
RIGHT SIDE
LEFT SIDE

FIG. 2

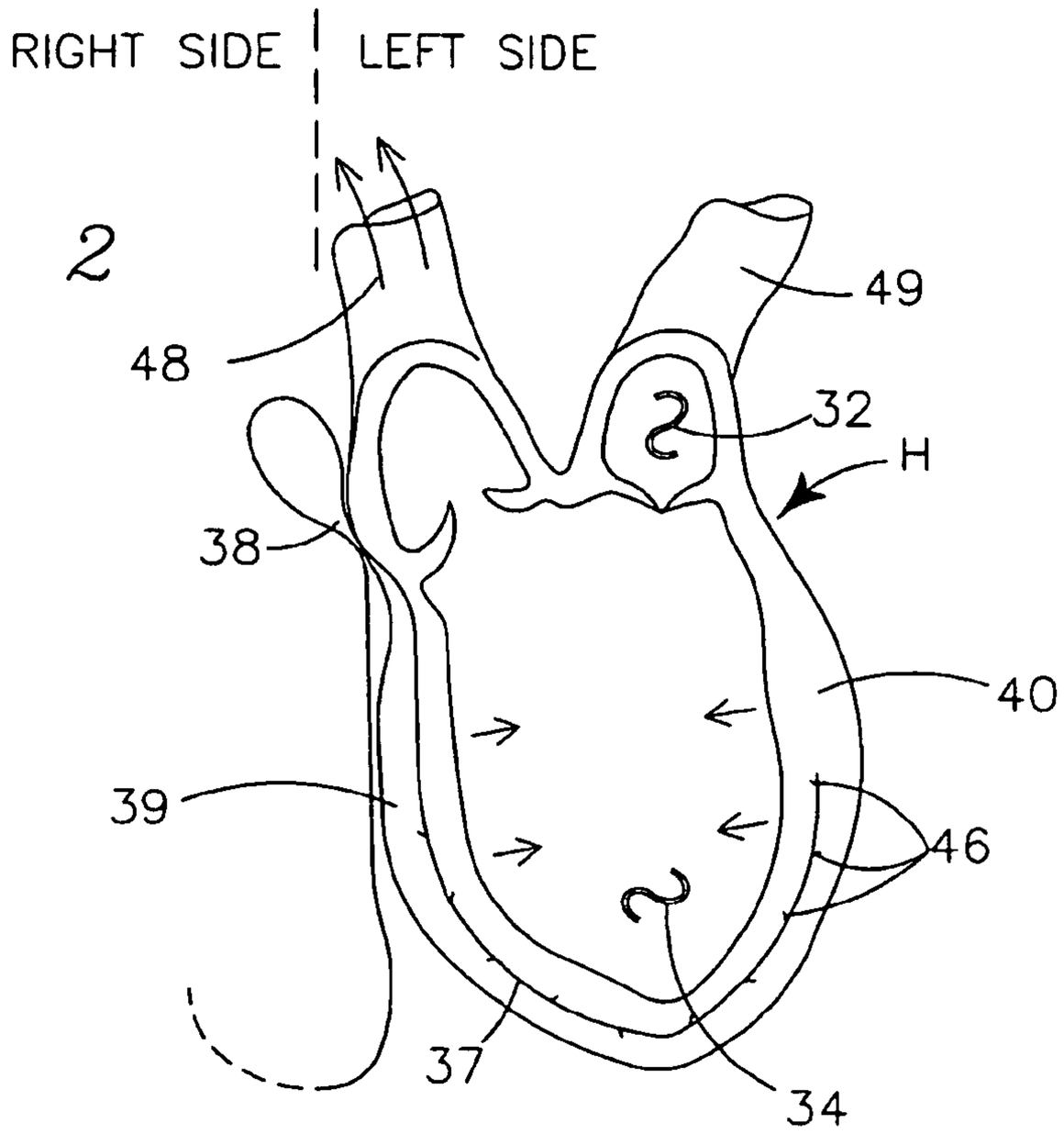

RIGHT SIDE
LEFT SIDE
48
49
32
H
38
40
39
46
37
34

FIG. 3

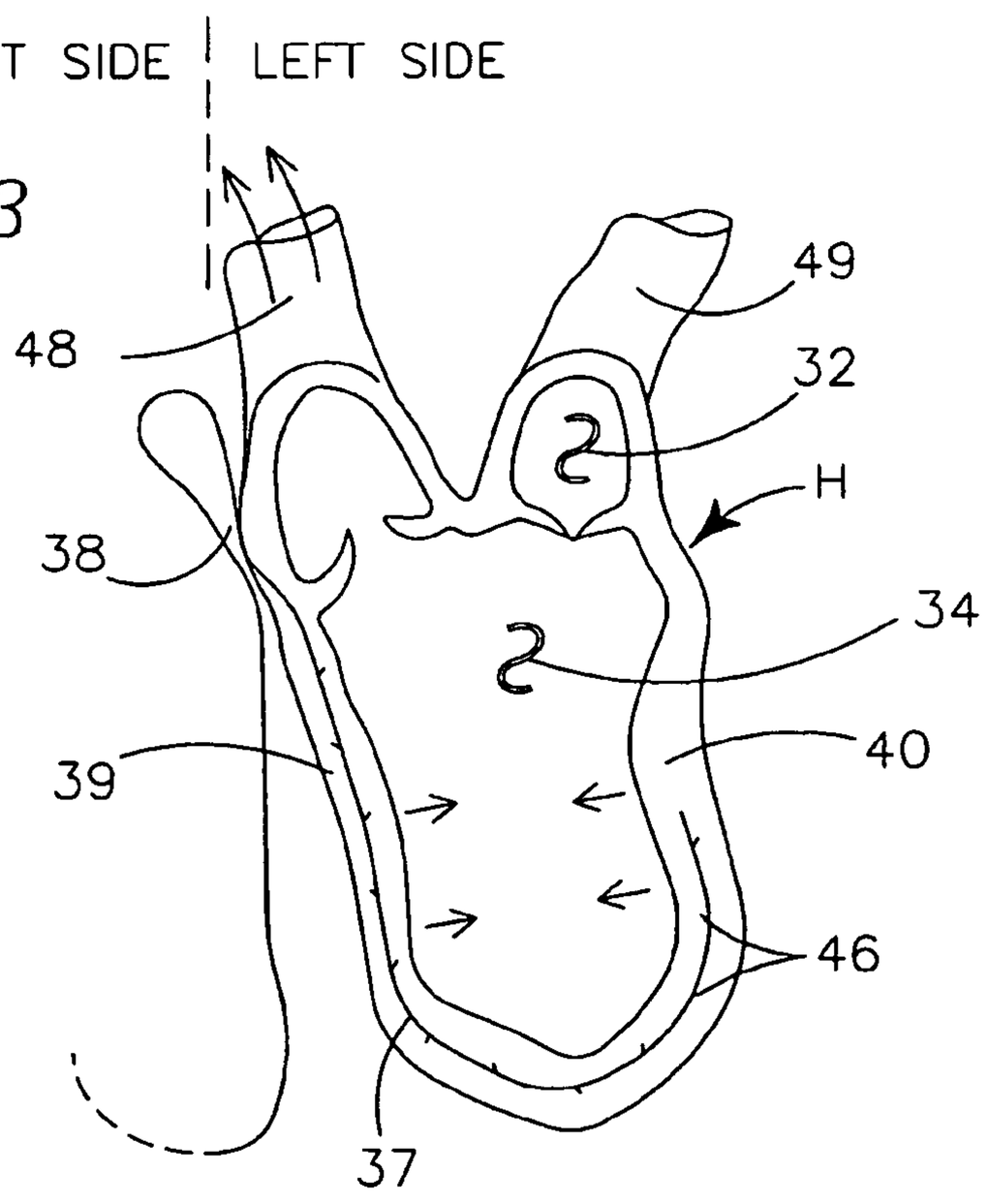

RIGHT SIDE
LEFT SIDE
48
49
32
H
38
34
40
39
46
37

FIG. 4

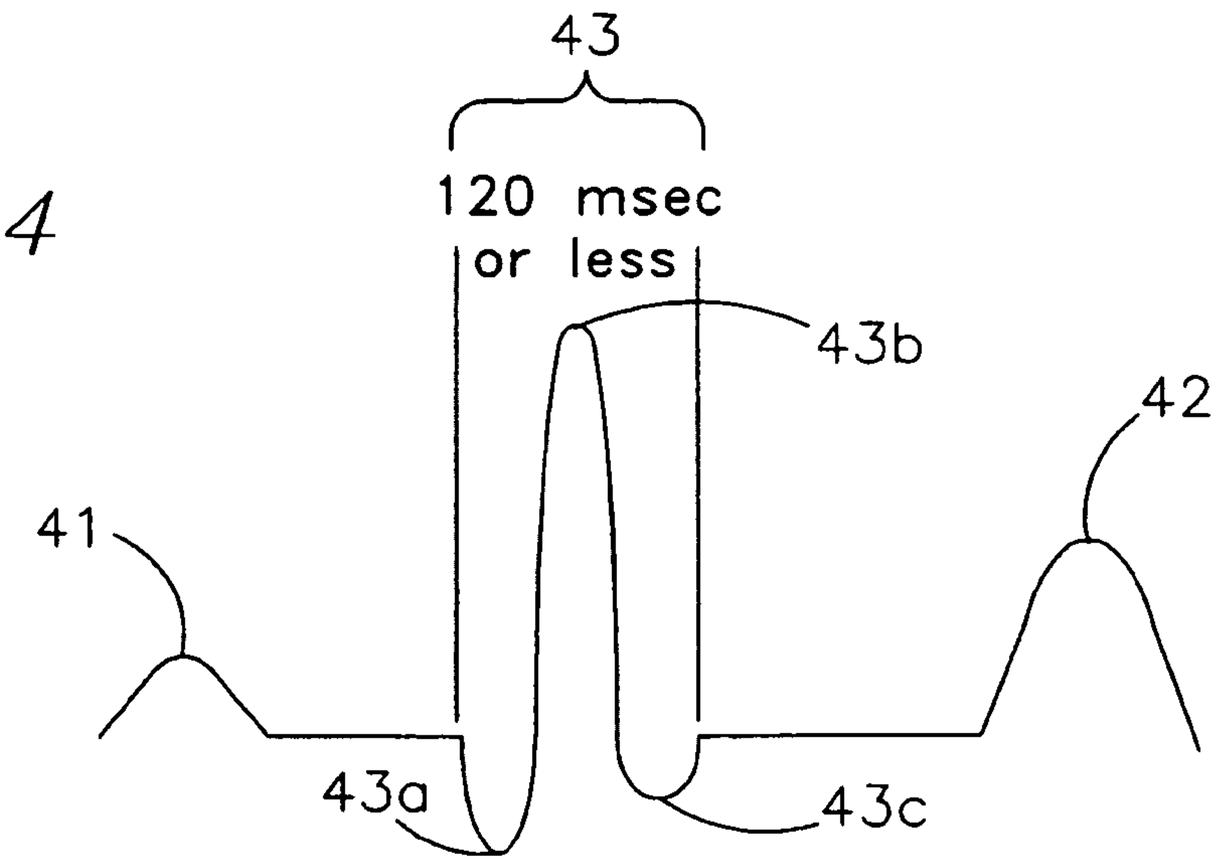


FIG. 5

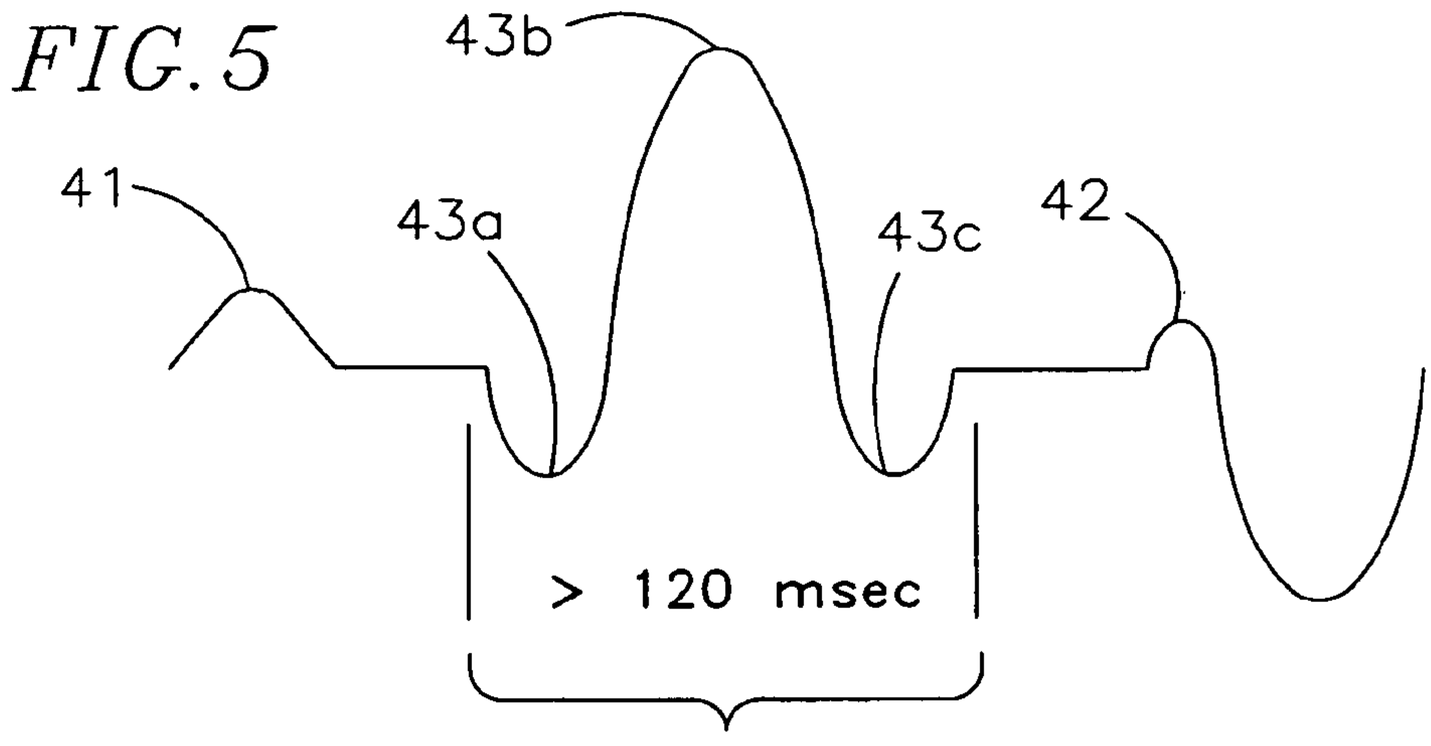


FIG. 6

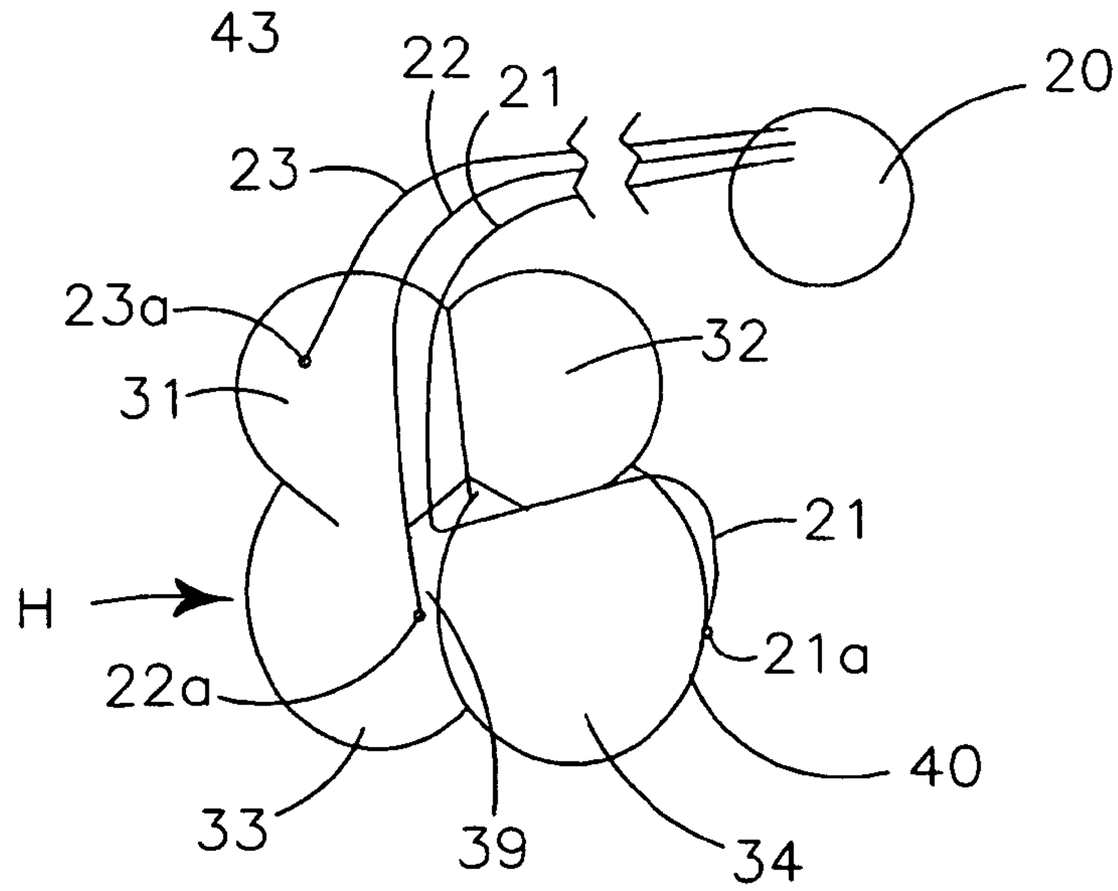


FIG. 7

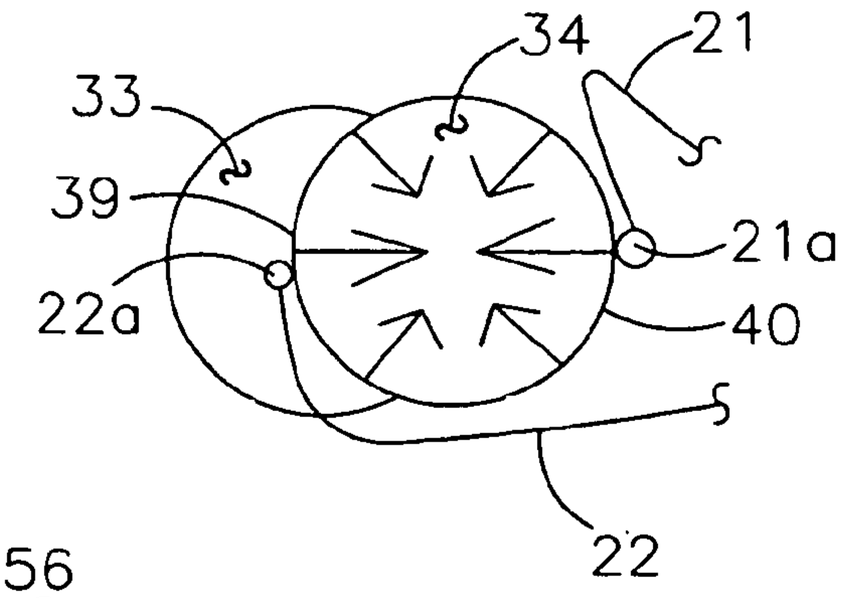
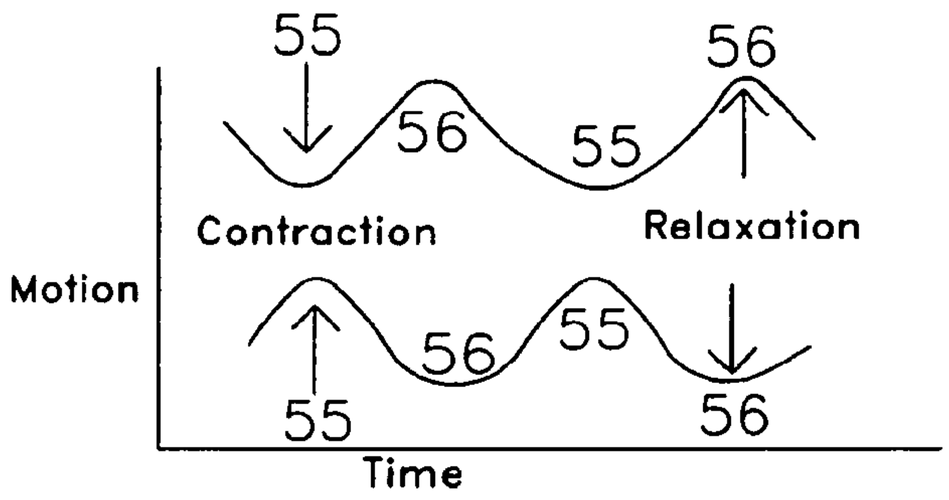


FIG. 8



Synchronous
lead tip Motion

FIG. 9

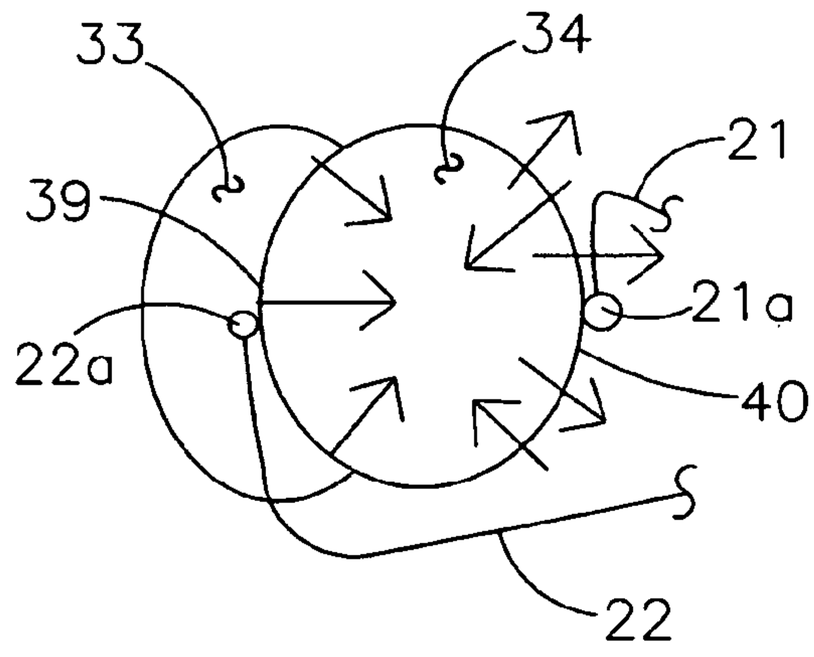
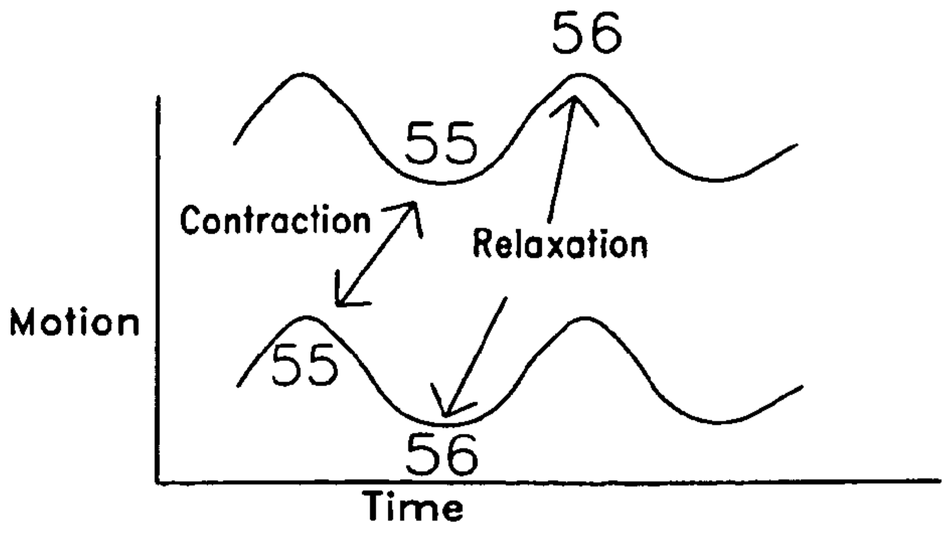
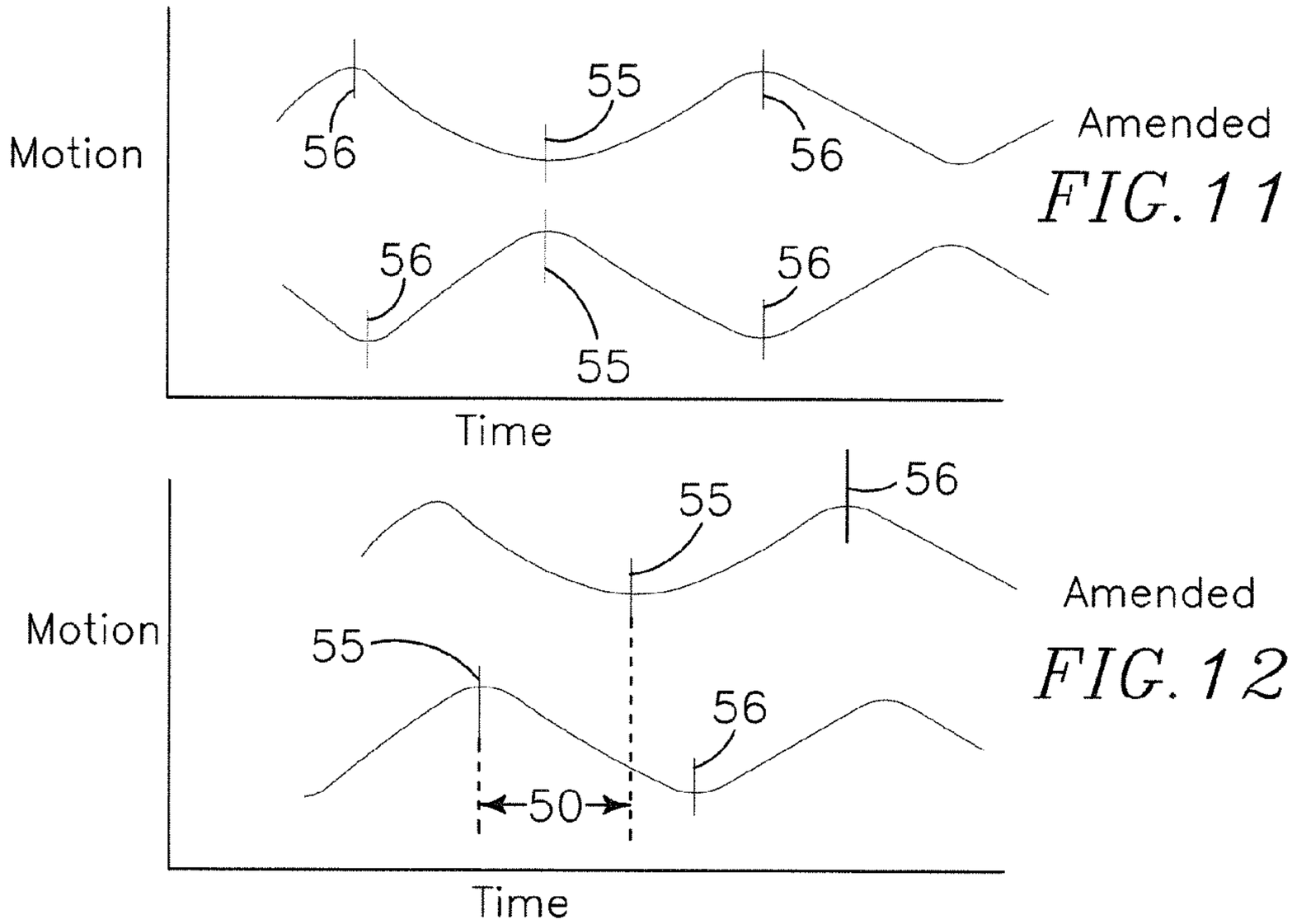


FIG. 10



Dyssynchronous
lead tip Motion

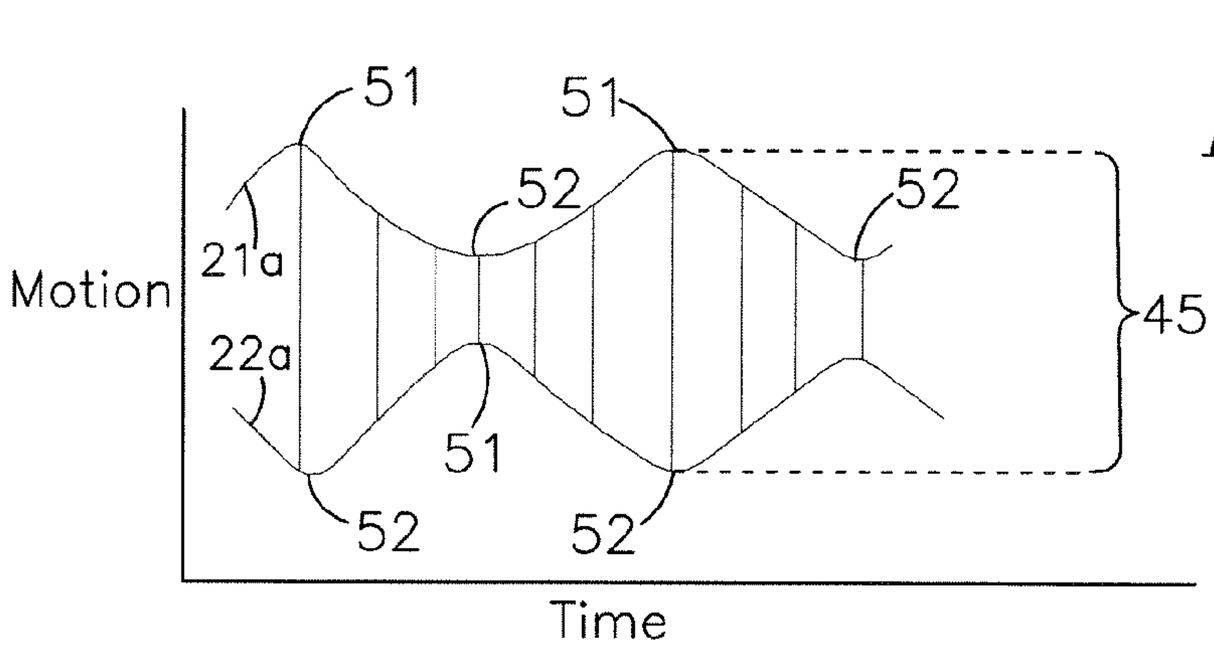


Amended

FIG. 11

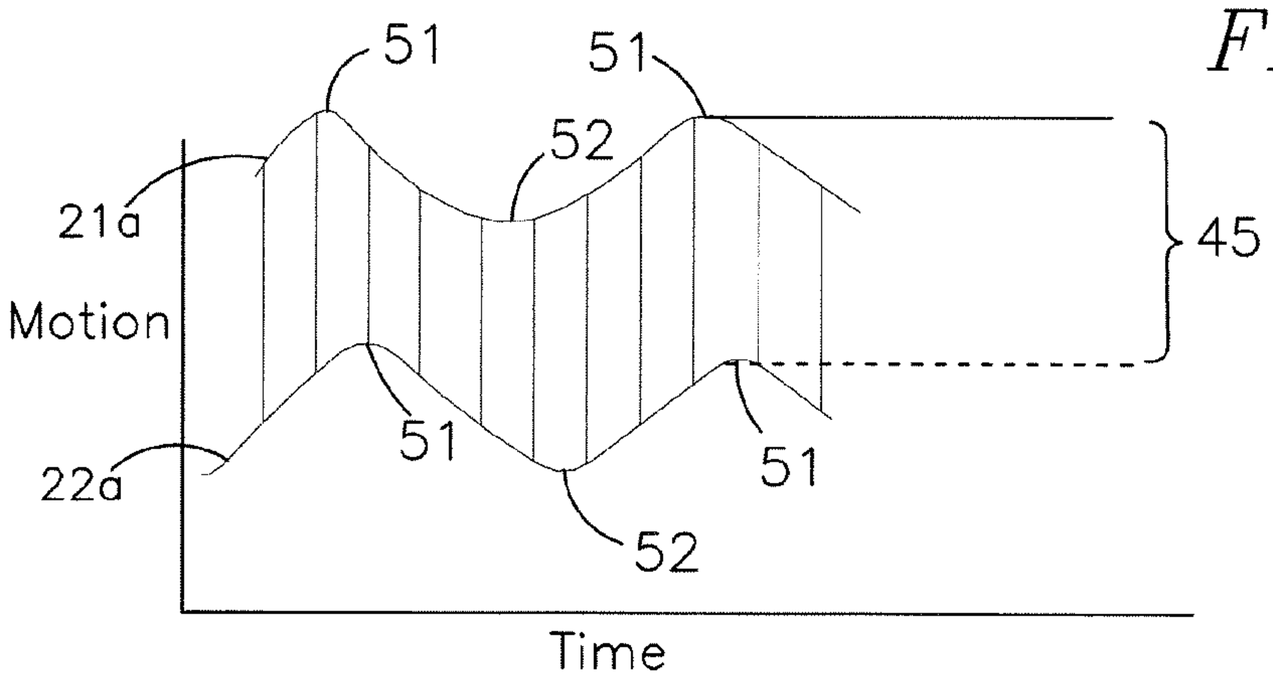
Amended

FIG. 12



Amended

FIG. 13



Amended

FIG. 14

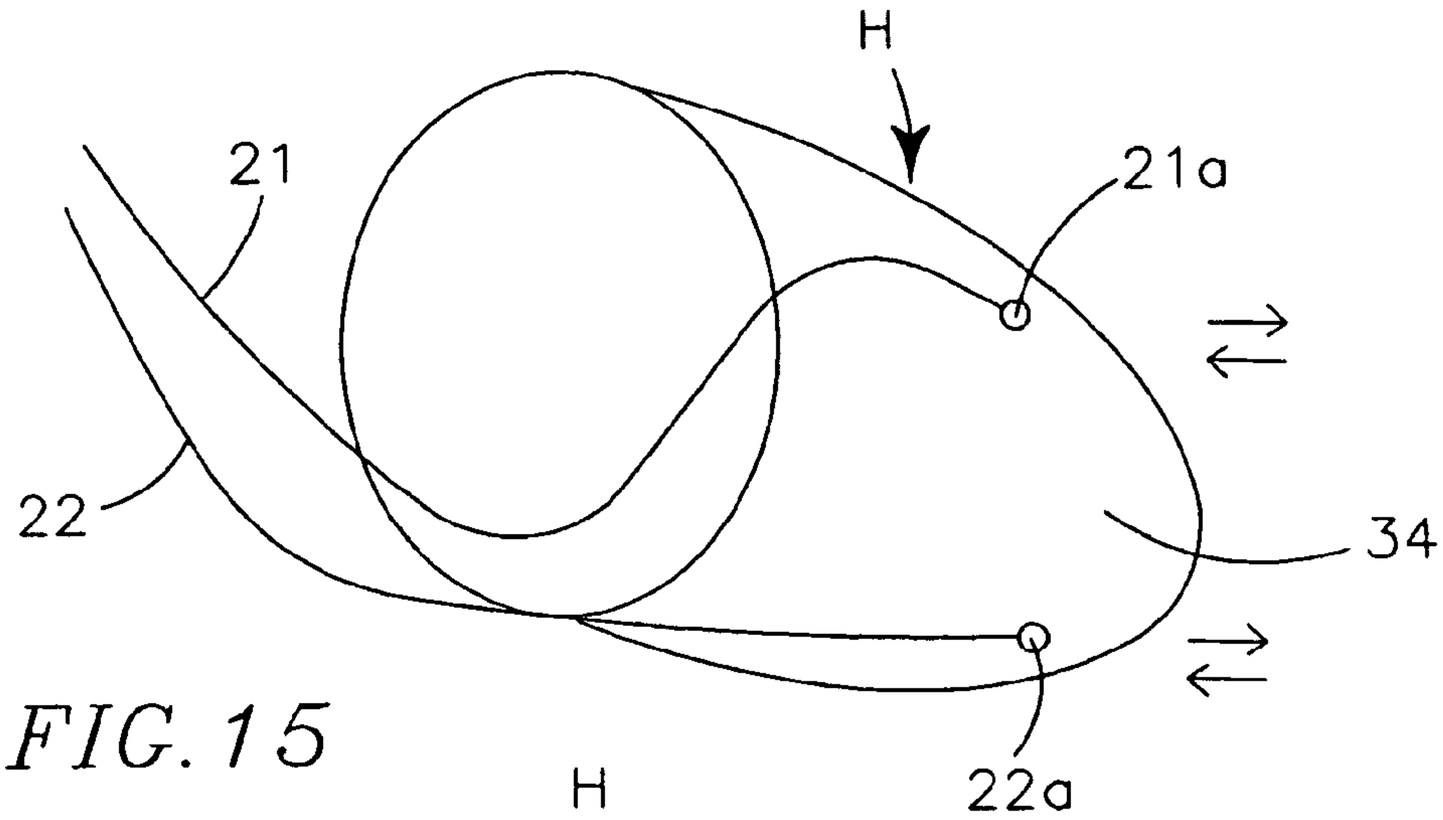


FIG. 15

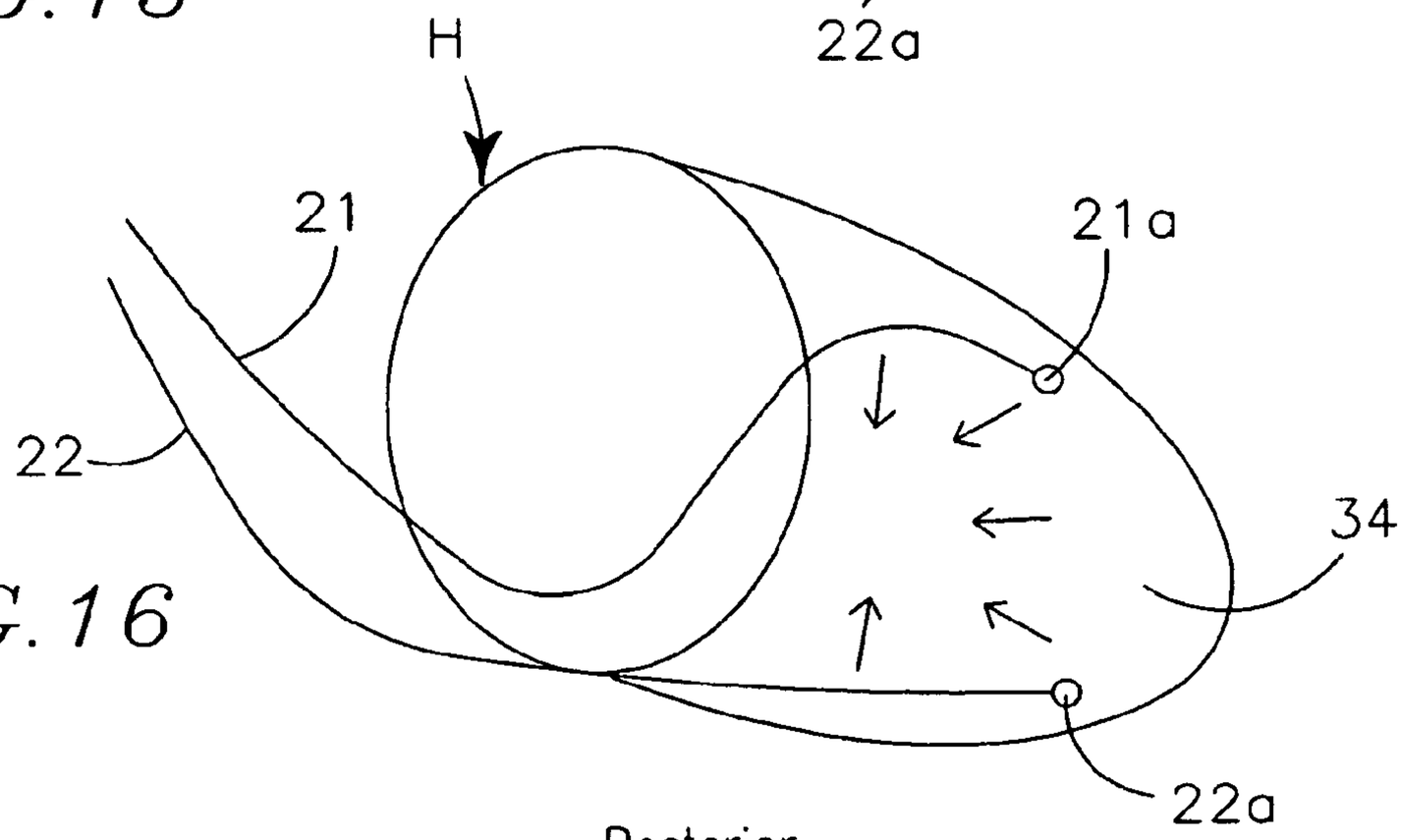


FIG. 16

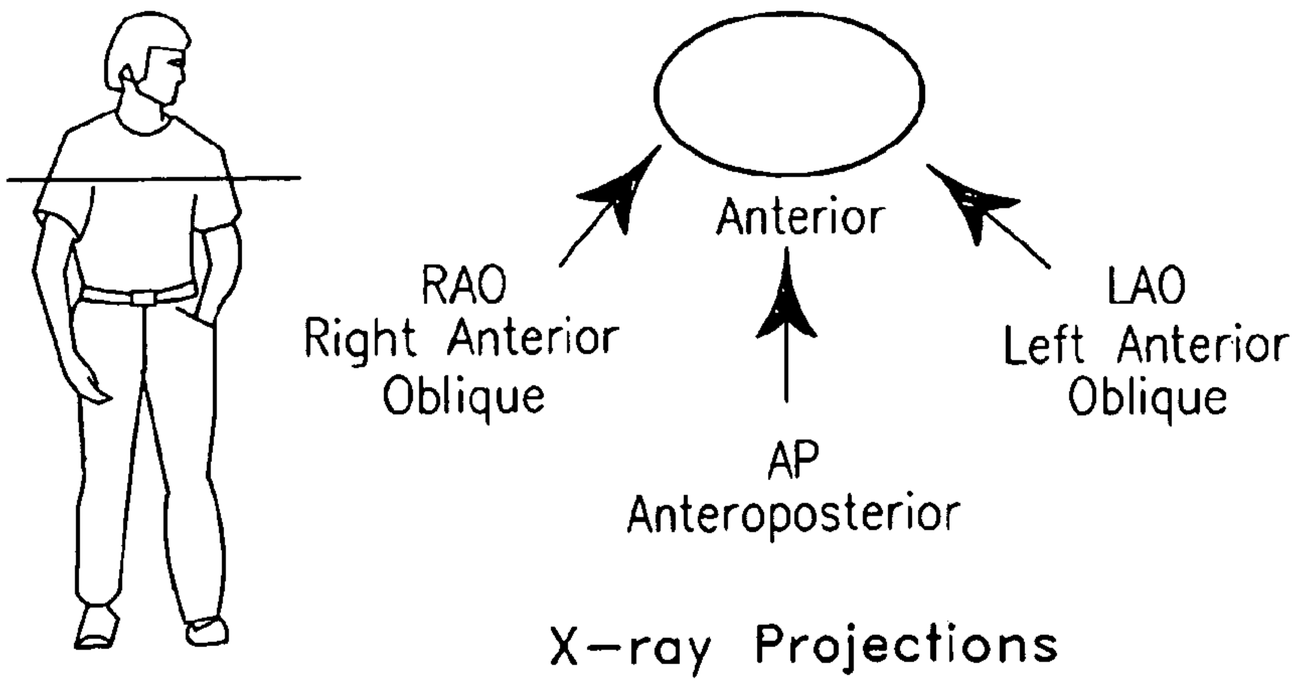


FIG. 17

FIG. 18

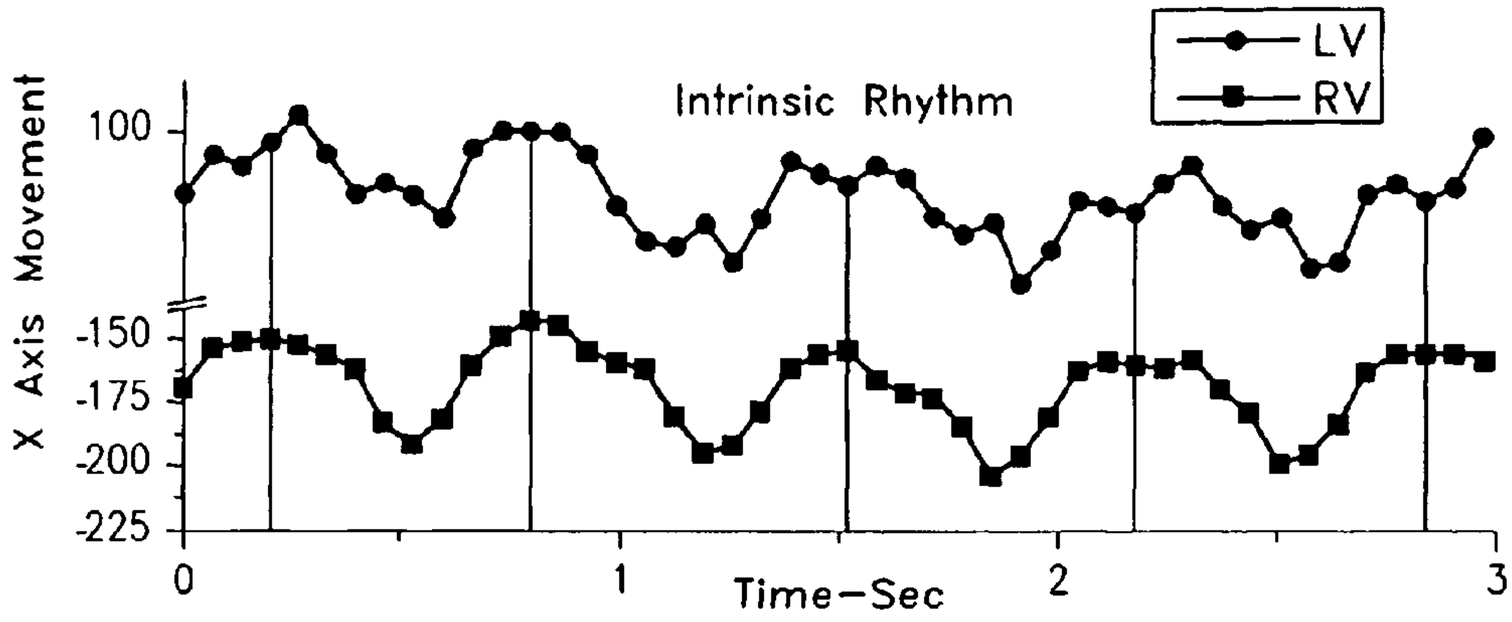
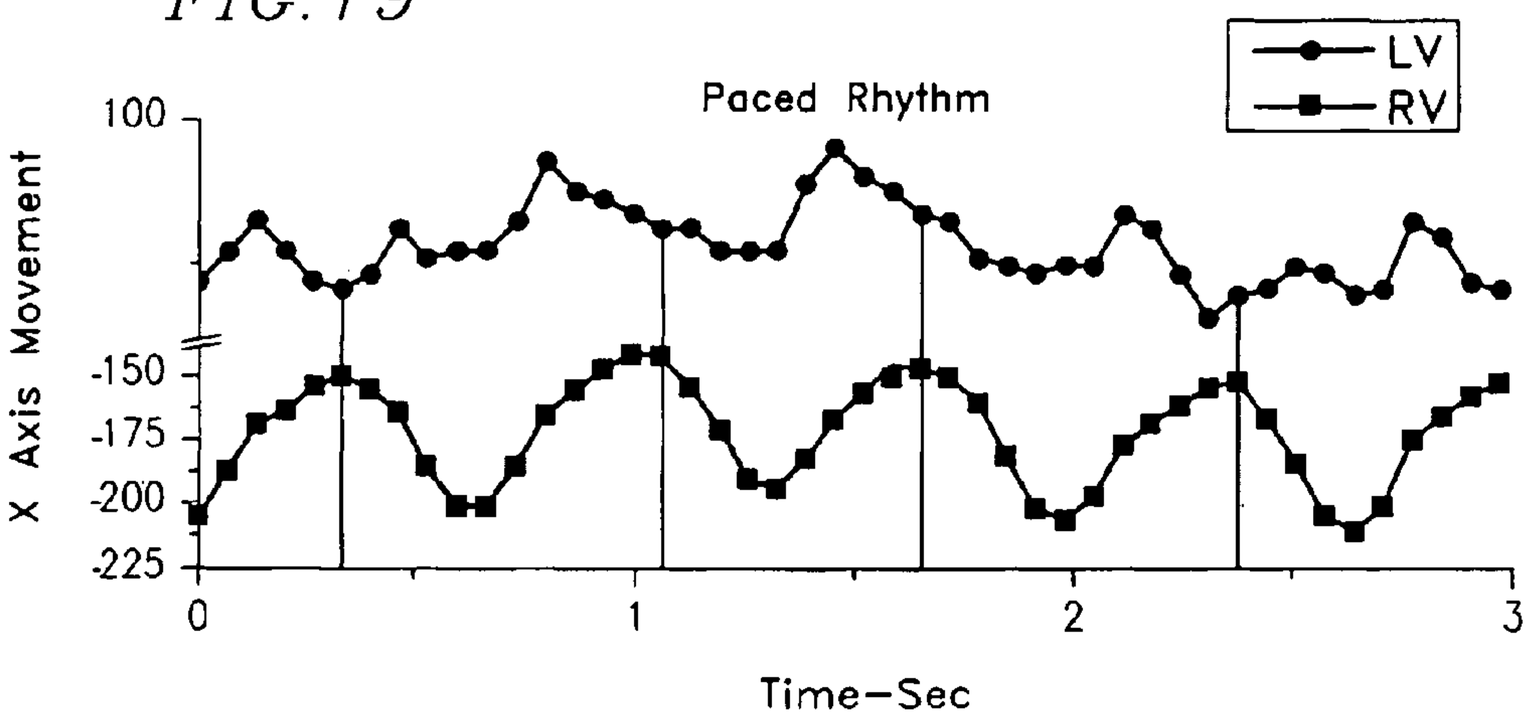

LV
RV
Intrinsic Rhythm
X Axis Movement
100
-150
-175
-200
-225
0
1
2
3
Time-Sec

FIG. 19


LV
RV
Paced Rhythm
X Axis Movement
100
-150
-175
-200
-225
0
1
2
3
Time-Sec

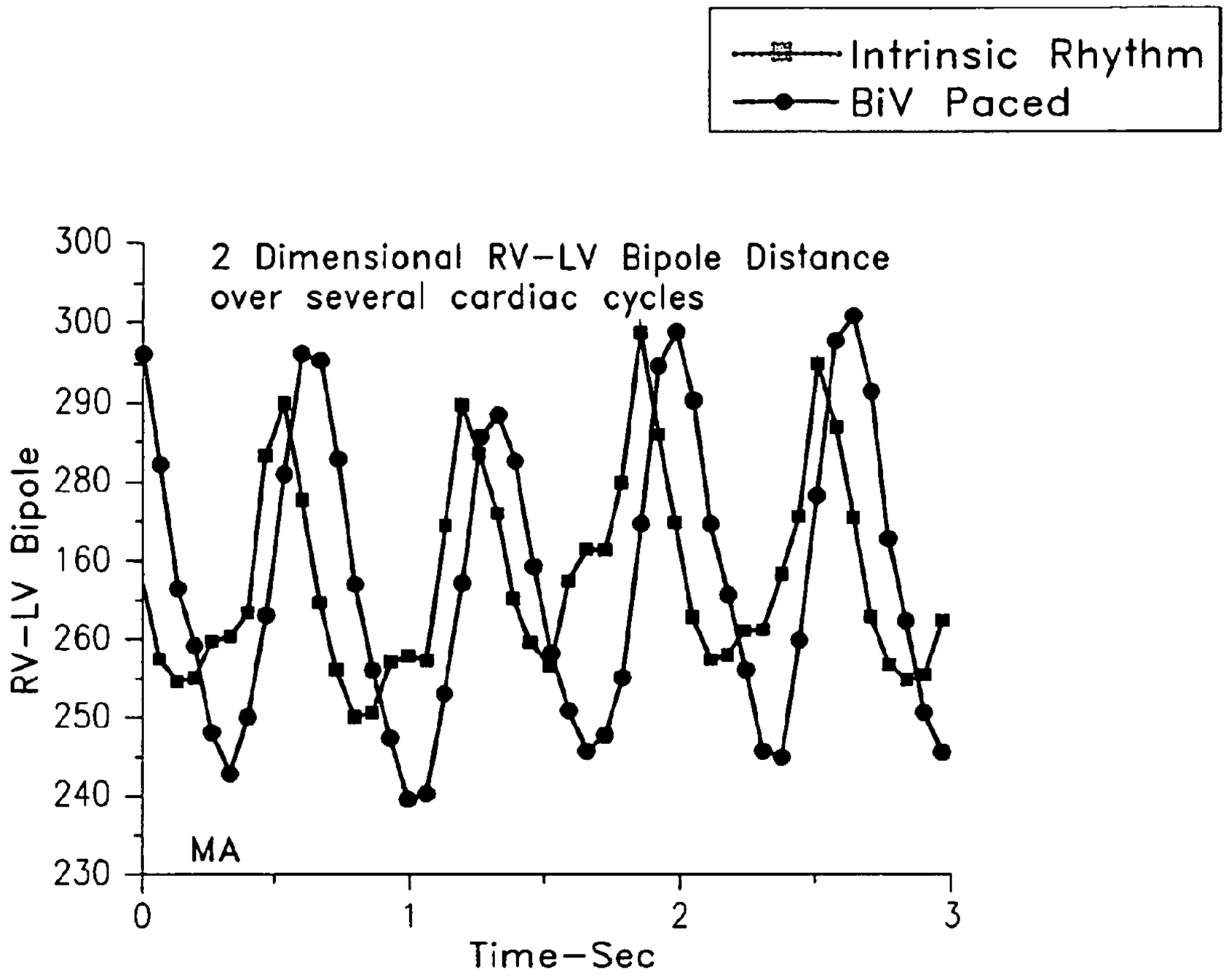
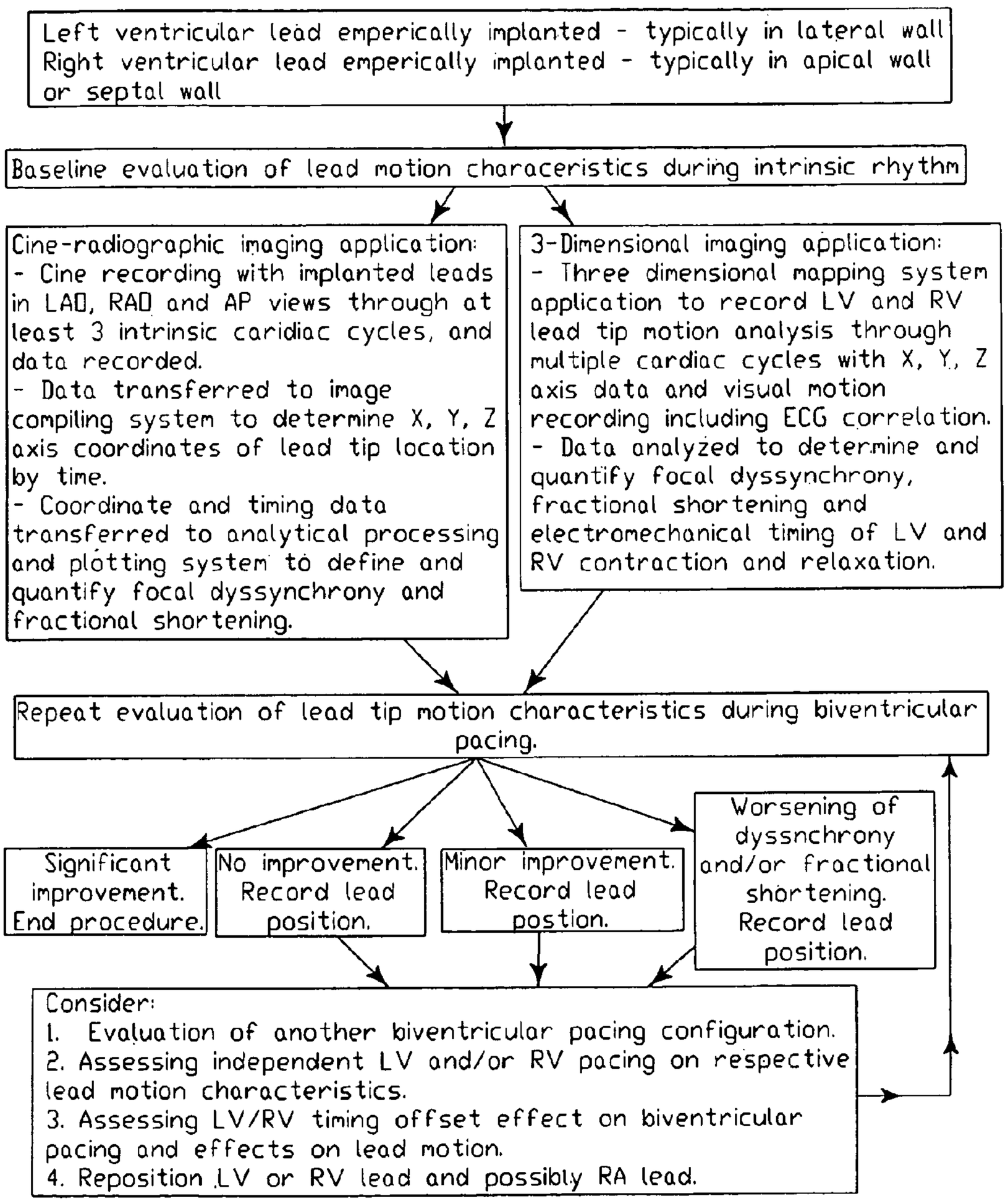

Intrinsic Rhythm
BiV Paced
2 Dimensional RV–LV Bipole Distance
over several cardiac cycles
RV–LV Bipole
300
300
290
280
160
260
250
240
230
MA
0
1
2
3
Time–Sec

FIG. 20

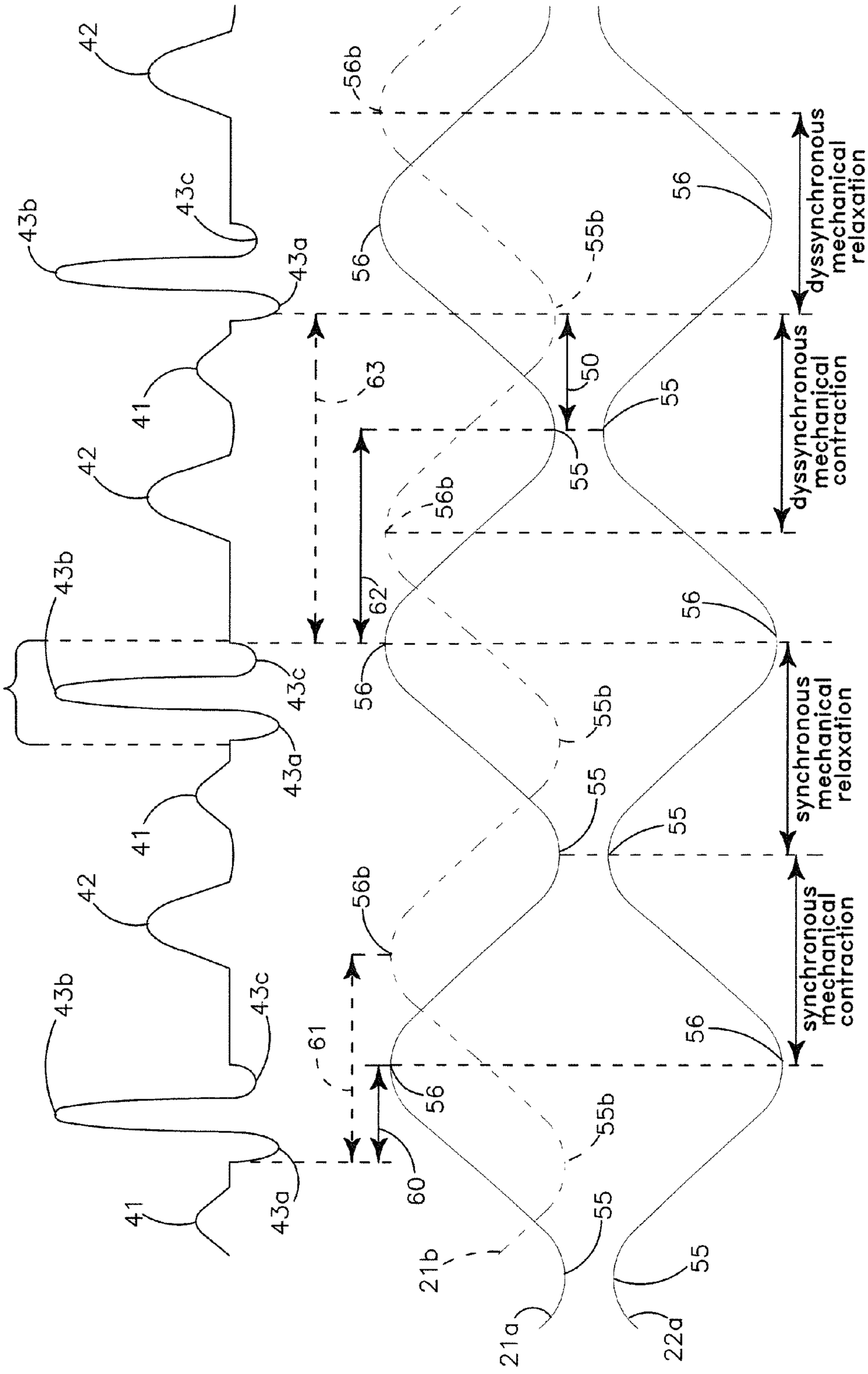
FIG. 21

Dynamic Lead Motion Analysis



New

FIG. 22


42
43b
43c
43a
41
56b
56
55b
55
dyssynchronous mechanical relaxation
dyssynchronous mechanical contraction
synchronous mechanical relaxation
synchronous mechanical contraction
63
62
61
60
50
21b
21a
22a

METHOD FOR OPTIMIZING CRT THERAPY

Matter enclosed in heavy brackets [ ] appears in the original patent but forms no part of this reissue specification; matter printed in italics indicates the additions made by reissue.

RELATED APPLICATIONS

This application claims the benefit of U.S. Provisional Patent Application No. 60/998,939 filed on Oct. 15, 2007.

BACKGROUND OF THE INVENTION

1. Field of the Invention

This invention relates to surgery, and more particularly to a method for determining [the synchronicity of] *ventricular heart wall movement throughout the cardiac cycle including* ventricular contractions*, systole and diastole* and optimizing the synchronicity of ventricular *heart wall movement including ventricular* contractions during CRT therapy and during follow-up.

2. Background and Description of Prior Art

The human heart is a pump with four chambers that beat in an organized sequence. Anatomically the heart is divided into left and right sides, and upper and lower chambers. The two upper chambers are the atria and the two lower chambers are the ventricles.

In a cardiac cycle blood enters the heart from the body's venous system though the [vena cava] *venae cavae* filling the right atrium. When the heart beats, the right atrium contracts forcing blood therein through the tricuspid valve into the right ventricle. Thereafter, contraction of the right ventricle forces the blood therein through the pulmonary artery to the lungs. Oxygenated blood returns to the heart, from the lungs, through the pulmonary [vein] *veins* and enters the left atrium. Contraction of the left atrium, which normally occurs synchronously with contraction of the right atrium, forces the blood therein through the mitral valve into the left ventricle. Contraction of the left ventricle, which normally occurs synchronously with contraction of the right ventricle, forces the blood therein outward through the aorta into the vascular system.

Systole is that portion of the cardiac cycle when the ventricular muscle cells contract causing the ventricles to force blood out of the ventricles to the lungs and body. Diastole occurs sequentially to systole and is that portion of the cardiac cycle when the ventricular muscle cells relax and the ventricles re-fill with blood from the atria.

Chamber synchrony is maintained by a complex conduction system which propagates electrical impulses to the heart muscle cells. The electrical impulses initiate the atrial and ventricular contractions.

The Sino-Atrial Node (SA node) is the pacemaker for the heart and is located in an upper portion of the right atrium. Electrical impulses spread from the SA node and cause adjacent atrial cells to depolarize in a spreading wave-front, causing the right and left atria to contract and pump blood into the respective ventricles. Depolarization and contraction of the left and right atria correlates with a "P wave" on an electrocardiogram (ECG). The electrical impulse continues propagating downwardly to the Atrio-Ventricular node (AV node) which is a small mass of highly specialized cardiac muscle fibers located in a lower portion of the right atrium. The AV node is the electrical connection from the right atrium to the right and left ventricles.

The AV node distally becomes the HIS bundle which bifurcates into a right bundle branch (RBB) and a left bundle branch (LBB). The bundle branches distally divide further into a network of Purkinje fibers which are specialized cells that conduct electrical impulses faster than other cells. Electrical impulses passing through the AV node continue through the bundle branches, and into the Purkinje fiber network encompassing the right and left ventricles. Because of the density of the Purkinje fiber network and the speed with which Purkinje fibers conduct electrical impulses, in a normal healthy heart, all the ventricular muscle cells contract synchronously during systole *and relax synchronously during diastole*. Depolarization and contraction of the ventricles correlates with the "QRS" complex on an ECG. The synchronized contraction of the atria and ventricles enhances the heart's pumping power. Thus, the heart includes both a complex electrical network of specialized conduction tissues and a complex mechanical network of chambers and valves.

A variety of disorders prevent the heart from operating normally, and these disorders may be systolic or diastolic and may cause dyssynchrony as well as abnormal contractility *and/or abnormal relaxation. Dyssynchrony is a timing abnormality*. Some of these disorders are caused by degeneration of the left ventricular conduction system which may block conduction of the electrical impulses and/or may delay propagation of the electrical impulses to the heart muscle cells. For example, left or right bundle branch block (LBBB/RBBB) is a heart failure condition that occurs when the conduction of the electrical impulses to the left or right ventricle is blocked or slowed. Bundle branch block can cause [dysschronous] *dyssynchronous* ventricular contractions which may result in heart failure. Intra-ventricular conduction delay (IVCD) is a heart failure condition that occurs when the propagation of the electrical impulses to the ventricles is "slowed down" by regional injury to myocardial tissue or by damaged Purkinje fibers that conduct the impulses slower than healthy Purkinje fibers.

When the left ventricular conduction system is damaged or "disconnected" the left ventricle muscle cells may still be excited eccentrically through muscle tissue conduction of the electrical impulses. Unfortunately, muscle tissue conduction is slower than Purkinje fiber network conduction and is also sequential. As a result, contraction of the affected portions of the left ventricle occurs in stages, rather than synchronously. For example, if a lateral wall of the left ventricle is affected by the conduction disorder, the muscle cells of the lateral wall will contract later than the muscle cells of the septal wall which is activated through normal Purkinje fiber conduction. Such dyssynchronous contraction degrades the contractility (pumping power) of the left ventricle and decreases the efficiency of the heart, which can result in, or exacerbate, heart failure.

Because the left ventricle pumps oxygenated blood to the body, a person's health is dependent upon the efficiency of the left ventricle. There are two primary methods of assessing the efficiency and pumping ability of the left ventricle; measuring Ejection Fraction, and measuring Shortening Fraction. Damage to the heart's electrical conduction system or damage to the heart's chambers and valves causes a decrease in Ejection Fraction and a decrease in Shortening Fraction.

Ejection Fraction measures the difference in the volume of blood within the left ventricle at the diastolic state, and at the systolic state, and compares the two volumes as a percentage. A normal Ejection Fraction range is 63-77% for males and 55-75% for females. Ejection Fraction percentage is determined with the following formula:

$$\frac{\left(\begin{array}{c}\text{Left Ventricle Diastolic Volume} - \\ \text{Left Ventricle Systolic Volume}\end{array}\right) \times 100}{\text{Left Ventricle Diastolic Volume}}$$

Shortening Fraction percentage measures the change in the diameter of the left ventricle between the systolic state and the diastolic state and is determined with the following formula:

$$\frac{\left(\begin{array}{c}\text{Left Ventricle End} - \text{Diastolic Diameter} - \\ \text{Left Ventricle End} - \text{Systolic Diameter}\end{array}\right) \times 100}{\text{Left Ventricle End} - \text{Diastolic Diameter}}$$

A Shortening Fraction greater than 30% is considered normal. A decrease in shortening fraction usually precedes a decrease in ejection fraction. *Timing of systole and diastole also provides measures of cardiac performance.*

Cardiac Resynchronization Therapy (CRT), also called biventricular pacing, has been shown to improve the symptoms of ventricular **[**dysschrony**]** *dyssynchrony* and abnormal contractility and improve heart failure symptoms. CRT uses biventricular pacing to synchronize left ventricular contraction by sending electrical impulses to the heart through surgically implanted electrical leads. *The timing intervals of these electrical impulses is known as Pacing Intervals. It is well recognized in the field that multiple programmable parameters, and differing pacing configurations may also be employed/applied to optimize therapy.*CRT is currently indicated for patients with left ventricular systolic dysfunction, an ejection fraction of less than 35%, a prolonged QRS complex of >120 msec and severe heart failure (New York Heart Association classification III and IV) despite maximal medical therapy.

Unfortunately, only about 65% to 70% of patients *who meet the above criteria* respond positively to CRT and the lack of positive response may be due to sub-optimal lead placement *and/or suboptimal pacing configuration*. Sub-optimal lead placement *and/or suboptimal pacing configuration* may occur because there is presently no dynamic testing of the lead positions *and/or suboptimal pacing configuration* to determine physiologic response to CRT. Further, testing of the lead positions is not performed to provide baseline measures of *ventricular heart wall movement,* ventricular **[**dysschrony**]** *dyssynchrony*, contractility or fractional shortening. As a result it is difficult to assess whether there is baseline dyssynchrony and whether there is improvement in *ventricular heart wall movement,* ventricular synchronicity, contractility and fractional shortening with current CRT implant techniques using empirically positioned leads *and/or empirically programmed pacing configurations*.

What is needed is a method to optimize the benefits of CRT therapy, to ensure optimal lead placement by dynamic assessment of lead locations during intrinsic or baseline rhythm and during biventricular paced rhythm and to provide objective measures to determine procedure effectiveness.

Our method for optimizing CRT therapy resolves various of the aforementioned drawbacks. Our method provides a tool for practitioners to objectively determine whether biventricular pacing provides physiologic benefits to the patient by allowing dynamic assessment of the motion of the ventricular leads, and therefore the motion of the ventricle walls, and provides measures of dyssynchrony, contractility and fractional shortening *and ventricular heart wall movement throughout the cardiac cycle*. The provided measures allow

assessment during intrinsic heart rhythm, to establish baseline **[**focal**]** dyssynchrony, *ventricular heart wall motion* and fractional shortening *throughout the cardiac cycle*, as well as **[**biventricular**]** paced heart rhythm to determine **[**focal**]** physiologic response to CRT therapy (i.e. changes in **[**focal**]** dyssynchrony, *ventricular heart wall motion* and fractional shortening). Our method can be utilized prognostically as a test for **[**focal dysschrony**]** *dyssynchrony* and response to pacing at temporary lead locations and with differing pacing configurations, and for optimizing CRT therapy at implant. Our method allows optimization of lead position *and/or pacing configuration* to improve patient outcomes based on physiologic assessment during the CRT procedure and during follow-up. *Our method also allows assessment of systole and diastole which may guide therapy for heart failure.*

Our invention does not reside in any one of the identified features individually but rather in the synergistic combination of all of its features, which give rise to the functions necessarily flowing therefrom as hereinafter specified and claimed.

SUMMARY

A method to optimize CRT therapy having the steps of implanting ventricular leads in a patient's heart; one application of the method involves radiologically scanning the heart over the duration of plural cardiac cycles during intrinsic heart rhythm and during biventricular paced rhythm in multiple views; determining X, Y and Z axis coordinate positional data of the ventricular leads relative to time; exporting the positional data to an analysis program for plotting and comparison of the movement of the implanted ventricular leads; determining the movement of the ventricular walls based upon the motion of the ventricular leads; comparing the intrinsic ventricular wall movement data to the paced ventricular wall movement data to assess baseline dyssynchrony and fractional shortening to determine whether pacing has improved synchronicity and fractional shortening, and if no improvement is evidenced changing the timing offset and/or repositioning the ventricular leads to another position in the heart to optimize the effectiveness of the CRT therapy. Another application of the method involves utilizing three dimensional mapping systems (such as St Jude NAVX) to delineate lead tip motion similarly allowing evaluation of baseline and paced synchronicity and fractional shortening *without detrimental effects of x-ray*.

In providing such a method it is:

a principal object to provide a method to optimize CRT therapy by evaluating and analyzing motion of the surgically implanted ventricular leads

a further object to provide a method to assess baseline ventricular synchronicity.

a further object to provide a method to dynamically assess paced ventricular synchronicity.

a further object to provide a measure of **[**focal**]** dyssynchrony.

a further object to provide a measure of **[**focal**]** contractility.

a further object to provide a method for optimizing ventricular lead placement to improve CRT therapy outcome based on physiologic assessment.

a further object to determine whether a patient will benefit from CRT therapy by testing lead locations during the CRT implantation.

a further object to provide a pre-procedure diagnostic test to predict CRT response.

a further object to avoid ineffective placement of biventricular leads.

a further object to provide a method to determine movement of ventricular walls in three dimensions *throughout the cardiac cycle*.

a further object to provide a method to graphically show movement of ventricular walls *throughout the cardiac cycle*.

a further object to improve ventricular synchrony, improve heart failure symptoms, reverse remodeling, improve ejection fraction, and decrease risk of dying.

a further object to provide a method to measure focal shortening fraction.

a further object to provide a method to measure **[**focal**]** dyssynchrony.

a further object to provide a prognostic test for **[**focal**]** dyssynchrony and for response to pacing at temporary lead tip locations *and/or differing pacing configurations*.

a still further object to provide a method for prognostic evaluation of dyssynchrony and response to biventricular pacing, and for optimizing CRT therapy by dynamically evaluating lead locations that is of new and novel design that maximizes physiologic benefits, reduces cost, improves the patient's health, prevents ineffective CRT implant placement and one that is otherwise well suited to the uses and purposes for which it is intended.

Other and further objects of our invention will appear from the following specification and accompanying drawings which form a part hereof. In carrying out the objects of our invention it is to be understood that the method, apparatus, steps and procedures are susceptible to change in design and arrangement with only one preferred and practical embodiment of the best known mode being illustrated in the accompanying drawings and specified as is required.

BRIEF DESCRIPTIONS OF DRAWINGS

In the accompanying drawings which form a part hereof and wherein like numbers refer to similar parts throughout:

FIG. **1** is a diagrammatic representation of a human heart showing the heart chambers and portions of the electrical impulse conduction system.

FIG. **2** is a diagrammatic representation of the left ventricle beginning contraction.

FIG. **3** is a diagrammatic representation similar to that of FIG. **2** showing the left ventricle in its contracted state.

FIG. **4** is a diagrammatic ECG recording of a normal cardiac cycle showing a P-wave, a T-wave and a QRS complex having a duration of <120 msec.

FIG. **5** is a diagrammatic ECU recording of an abnormal cardiac cycle showing a prolonged QRS complex of >120 msec.

FIG. **6** is a diagrammatic representation of a human heart showing a biventricular pacing mechanism having leads communicating with the right atrium, right ventricle and left ventricle.

FIG. **7** is a diagrammatic cross-section of the right and left ventricles showing positions of the ventricular lead tips as shown by a left anterior oblique (LAO) x-ray view with arrows representing synchronous ventricular wall movement.

FIG. **8** is a graphic representation correlating with FIG. **7** of movement of the right (bottom line) and left (top line) ventricular lead tips, relative to each other, during synchronous ventricular contraction.

FIG. **9** is a diagrammatic cross-section of the right and left ventricles showing positions of the ventricular lead tips as shown by a left anterior oblique (LAO) X-ray view with arrows representing dyssynchronous ventricular wall movement.

FIG. **10** is a graphic representation correlating with FIG. **9** of movement of the right (bottom line) and left (top line) ventricular lead tips, relative to each other, during dyssynchronous ventricular contraction.

FIG. **11** is a graphic representation of synchronous left ventricular contraction showing focal synchrony; left ventricular lead motion is depicted on the top with the right ventricular lead motion depicted on the bottom.

FIG. **12** is a graphic representation of dyssynchronous left ventricular contraction showing focal dyssynchrony with left ventricular lead motion depicted on the top and right ventricular lead motion depicted on the bottom.

FIG. **13** is a graphic representation of synchronous left ventricular contraction showing normal contractility with left ventricular lead motion depicted on the top and right ventricular lead motion depicted on the bottom.

FIG. **14** is a graphic representation of dyssynchronous left ventricular contraction showing reduced contractility with left ventricular lead motion depicted on the top and right ventricular lead motion depicted on the bottom.

FIG. **15** is a diagrammatic representation of the left ventricle roughly correlating with a right anterior oblique (RAO) X-ray view with arrows showing longitudinal Z-axis motion of the ventricular lead tips during synchronous left ventricular contraction.

FIG. **16** is a diagrammatic representation of the left ventricle roughly correlating with a right anterior oblique (RAO) X-ray view with arrows showing longitudinal Z-axis motion of the ventricular lead tips during dyssynchronous left ventricular contraction.

FIG. **17** is a diagrammatic representation of the three X-ray projections used for cine loop recordings.

FIG. **18** is a graphic representation of dyssynchronous lead tip movement during intrinsic heart rhythm.

FIG. **19** is a graphic representation of paced lead tip movement showing improved synchrony.

FIG. **20** is a graphic representation showing two dimensional ventricular lead tip movement along the X-axis and Y-axis during intrinsic heart rhythm and during paced heart rhythm showing improved contractility and improved shortening fraction during pacing.

FIG. **21** is a flow chart setting forth the steps of the disclosed method for optimizing CRT therapy.

FIG. 22 is a graphic representation of synchronous heart wall movement (FIGS. 7 and 8) and dyssynchronous heart wall movement (FIGS. 9 and 10) showing the correlation with a simultaneous ECG recording (FIG. 4) of plural cardiac cycles by time.

DESCRIPTION OF PREFERRED EMBODIMENT

The present invention provides a method for optimizing cardiac resynchronization therapy (CRT) by assessing movement of tips of ventricular leads implanted in a human heart. The disclosed method is expressly described in terms of the left ventricle; however application to the other heart chambers, including the right ventricle, may be readily appreciated by those skilled in the art without departing from the present inventive method. *The invention provides a method to quantify electromechanical intervals of ventricular systole and diastole to assess dyssynchrony and may also be used to characterize other forms of systolic and diastolic heart failure and may be used as a means to guide therapy.*

Our method generally provides a ventricular pacemaker **20** having a left ventricular lead **21**, a right ventricular lead **22** and an atrial lead **23**; a radiological three dimensional imaging system **24**; an image compiling system **25**; an analytical

processing system **27** and a visual display **26**. The left ventricular lead **21** has a lead tip **21**a at its terminal end, the right ventricular lead **22** has a lead tip **22**a at its terminal end and the atrial lead **23** has a lead tip **23**a at its terminal end.

CRT is currently indicated for patients with left ventricular dyssynchrony, an ejection fraction<35%, a prolonged QRS complex **43** having a duration>120 msec and severe heart failure, New York Heart Association (NYHA) classification III or IV, despite maximal medical therapy.

The normal cardiac conduction system is diagramed at FIG. **1**. The sinoatrial node **30** (SA node) proximate the right atrium **31** is the pacemaker for heart H. Electrical impulses are generated in and propagate from the SA node **30** to synchronously activate cardiac muscle cells comprising right atrium **31** and left atrium **32**. The electrical impulse then propagates downwardly to atrioventricular node **35** (AV node) which is the electrical connection from the right atrium **31** to right ventricle **33** and left ventricle **34**.

The AV node **35** distally becomes *the* HIS bundle **38** which bifurcates into left bundle branch **37** and right bundle branch **36** which conduct the electrical impulse to Purkinje fibers **46** of the right ventricle **33** and the left ventricle **34** so that electrical activation of the right ventricle **33** and left ventricle **34** occurs synchronously resulting in uniform ventricular contraction. (FIGS. **7**, **8**).

Cardiac muscle cells (not shown) need to be electrically excited to undergo mechanical contraction. During the excitation, known as depolarization, electrical signals are generated that can be recorded with an electrocardiogram (ECG) (not shown). Features of an ECG recording (FIGS. **4**, **5**) correspond to the origin of the electrical activity. Depolarization in the atria **31**, **32** generates a P wave **41**. Depolarization in the ventricles **33**, **34** generates a wave form known as a QRS complex **43** which consists of a Q-wave **43**a, an R-wave **43**b and an S-wave **43**c. A normal QRS complex **43** has a duration of less than 120 msec. (FIG. **4**). A QRS complex **43** having a duration greater than 120 msec (FIG. **5**) is abnormal and is one **[**criteria**]** *criterion* for CRT.

Damage to the conduction tissues below the AV node **35**, such as at the level of the bundle branches **36**, **37** or lower, can result in dyssynchronous activation of the ventricles **33**, **34** which may lead to ventricular dysschrony. Ventricular dysschrony is defined as non-uniform contraction of the ventricles **33**, **34** due to delayed activation. (FIGS. **9**,**10**). Damage to the conduction tissues may also cause a prolonged QRS complex **43**. Current guidelines use a prolonged QRS complex **43** as a surrogate to identify ventricular dyssynchrony. Unfortunately, a prolonged QRS duration and ventricular dyssynchrony only show a rough correlation to one another.

As shown in FIGS. **2** and **3**, the left ventricle **34** starts to contract after an electrical impulse (not shown) propagating down from the left bundle branch excites muscle cells (not shown) of septal wall **39** and lateral wall **40**. As the muscle cells contract they become shorter and thicker causing the septal and lateral walls **39** and **40** respectively, to contract inwardly towards each other to pinup blood out of the left ventricle **34** to the body (not shown) through the aorta **48**.

As shown in FIG. **6**, CRT uses an atrial lead **23** having a lead tip **23**a positioned in the right atrium **31**, a right ventricular lead **22** having a lead tip **22**a positioned on right ventricular apex or septal wall **39** and a left ventricular lead **21** having a lead tip **21**a implanted on left ventricular lateral wall **40**, left ventricular anterolateral wall (not shown) or left ventricular posterolateral wall (not shown) to provide pacing on both sides of the left ventricle **34** to resynchronize left ventricle **34** activation.

FIGS. **7** and **9** represent a cross-section view of the right ventricle **33** and left ventricle **34** similar to a left anterior oblique (LAO) X-ray view (FIG. **17**) and show the relative positions of the right ventricular lead tip **22**a and the left ventricular lead tip **21**a. Arrows represent direction of ventricular wall movement during synchronous systole/contraction (FIG. **7**) and **[**dysschronous**]** *dyssynchronous* systole/contraction (FIG. **9**). *Simultaneous electrocardiographic display provides an ability to determine electromechanical measurements of systole and diastole during intrinsic rhythm and in response to pacing.*

FIGS. **8** and **10** are graphic representations of the motion of the left ventricular lead tip **21**a and the motion of the right ventricular lead tip **22**a *through plural complete cardiac cycles* in the short axis shown in FIGS. **7** and **9**. FIGS. **7** and **8** show normal synchronous ventricular contraction *and relaxation* while FIGS. **9** and **10** show dyssynchronous ventricular contraction *and relaxation* evidenced by a timing difference of the left ventricular lead tip **21**a and right ventricular lead tip **22**a at maximum contraction **55** and relaxation **56**. As shown, maximal contraction **55** of the left ventricular lead tip **21**a occurs at a trough in the graphic representation of the lead tip movement and is 180 degrees out of phase as compared to the maximal contraction **55** of the right ventricular lead tip **22**a which occurs at a crest in the graphic representation of the lead tip movement.

FIGS. **11** and **12** are lead tip **21**a, **22**a motion schematics. Synchronous ventricular contraction (FIG. **11**) is evidenced by simultaneous (vertically aligned) right ventricular lead tip **22**a and left ventricular lead tip **21**a maximal contraction **55**. Dyssynchronous ventricular contraction (FIG. **12**) is evidenced by a timing delay **50** between maximal contraction **55** of the lead tips **21**a, **22**a.

The difference in time **50** **[**to**]** *between* maximal contraction **55** of the right ventricular lead tip **22**a and left ventricular lead to **21**a is a focal measure of dyssynchrony. FIG. **12** illustrates this measurement at **50**. The difference in time **50** from onset of electrical activation (start of the QRS complex **43**) to maximal contraction **55** of the right or left ventricular lead (not shown), also provides a measure of electromechanical dyssynchrony. *FIG. 22 shows the timing correlation of the electrical activation of the heart H as it relates to the mechanical activation of the heart H. Other electromechanical measures including rate of contraction and/or relaxation and/or duration of contraction and/or relaxation (relative to overall cardiac cycle length) as well as timing of contraction and/or relaxation, may have potential use to guide therapy of systolic or diastolic heart failure.*

FIGS. **13** and **14** illustrate quantification of focal contractility by measuring the distance between the right ventricular lead tip **22**a and the left ventricular lead tip **21**a at time stamped points in a cardiac cycle. Average vertical distance **45** (FIGS. **13**, **14**) between the lead tips **21**a, **22**a at the same time stamp is the measure of focal contractility. FIG. **13** shows normal contractility represented by **[**vertically aligned**]** *synchronous movement of the left ventricular lead tip 21a and the right ventricular lead tip 22a wherein* troughs **52** **[**and**]** *representing contraction motion of the left ventricular lead tip 21a are vertically aligned with* crests **51** **[**that**]** *representing contraction motion of the right ventricular lead tip 22a, and same time stamp relaxations* are widely separated *45* vertically **[**while**]**. FIG. **14** shows reduced contractility represented by *absence of vertical alignment of the troughs 52 of the left ventricular lead 21a motion with the crests 51 of the right ventricular lead 22a motion and* lessened vertical separation *45* between the lead tips **21**a, **22**a at the same time

stamp caused by ventricular [dysschrony] *dyssynchrony* **50**. Using contractility measures, fractional shortening may also be determined.

Analysis of lead tip **21**a, **22**a motion in the left ventricular short axis (approximated in the LAO projection) provides data on concentric contraction and radial motion. (FIGS. **7**, **9**). Left ventricular lead tip **21**a and right ventricular lead tip **22**a motion in the RAO view provides data to determine longitudinal (Z-axis) motion. Synchronous Z-axis contraction and synchronous Z-axis relaxation of the ventricles **33**, **34** is evidenced by parallel lines of motion for the right ventricular lead tip **22**a and left ventricular lead tip **21**a (FIG. **15**) while dyssynchronous Z-axis contraction and dyssynchronous Z-axis relaxation is evidenced by non-parallel lines of motion for the right ventricular lead tip **22**a and left ventricular lead tip **21**a. (FIG. **16**).

With radiologic analysis a cine loop recording (not shown) is made in left anterior oblique (LAO), right anterior oblique (RAO) and anterior posterior (AP) projections (FIG. **17**) during plural complete cardiac cycles to document motion of the left ventricular lead tip **21**a and the right ventricular lead tip **22**a during intrinsic heart rhythm as well as during paced heart rhythm.

The cine loop recording data is exported, preferably in an AVI format, to the image compiling system **25** which is preferably a physics motion analysis program **25** such as Tracker™ software from Open Source Physics, Inc. wherein the X-axis, Y-axis and Z-axis coordinates for the left ventricular lead tip **21**a and the right ventricular lead tip **22**a are determined for each recorded cine frame and identified by time stamps throughout the plural cardiac cycles. Cine is no less than 15-30 frames per second (fps) to ensure accurate time stamps.

Table 1 sets forth a sample of the data collected by the physics motion analysis program **25** showing left ventricular lead tip **21**a positions. For each position the cine frame time is noted as is the X-axis coordinate and the Y-axis coordinate.

TABLE 1

t	x	y
0	116.426	22.788
0.066	115.926	17.283
0.132	112.422	20.285

The X axis coordinate data, the Y axis coordinate data and the time data for each lead tip **21**a, **22**a, **23**a in each view is then exported to analytical processing system **27** having a computer operating *system, such as* Origin™ software manufactured by Origin Lab Corp. of Northhampton, Mass., USA. Paired analyses comparing the intrinsic heart rhythm data and the paced heart rhythm data, is performed for each radiographic view. (LAO, RAO and AP). The motion of the left ventricular lead tip **21**a and the motion of the right ventricular lead tip **22**a motion is then visually presented, such as by graphing, showing the time difference **50** to maximum contraction **55** between the right ventricular lead tip **22**a and left ventricular lead tip **21**a which provides a focal measure of [dysschrony] *dyssynchrony* (FIGS. **11**, **12**) and the percentage of shortening from maximum diastole to maximal systole between the left ventricular lead tip **21**a and the right ventricular lead tip **22**a providing a measure of contractility and shortening fraction. *These measures can also be correlated with simultaneous electrocardiographic (ECG) recording to provide electromechanical intervals of ventricular systole and diastole to assess dyssynchrony and may also be used to consider other forms of systolic and diastolic heart failure and may be used as a means to guide therapy.* Baseline [dysschrony] *dyssynchrony*, baseline contractility [and], baseline shortening fraction *and ventricular heart wall motion in systole and diastole* are then compared with paced [dysschrony] *dyssynchrony*, *paced ventricular heart wall motion,* paced contractility [and], paced shortening fraction *and ventricular heart wall motion in systole and diastole* at the current lead **21**, **22** positions to determine the effectiveness of CRT. (FIGS. **18**-[**20**] *22*). Using the measures it is possible to assess whether there is focal improvement in [dysschrony] *dyssynchrony*, contractility [and], shortening fraction *and ventricular heart wall motion in systole and diastole* with pacing at the current lead locations and pacing configuration. Other pacing configurations such as isolated right ventricular or left ventricular pacing, or pacing with RV-LV offset could also be similarly assessed.

This method is also applicable using a three dimensional mapping system such as St Jude Medical NAVX to document lead tip motion without x-ray use. In such an application, *the* 3D mapping [patches are placed] *system is set up* for standard use and the left and right ventricular leads **21**, **22** are connected to the [NAVX monitor] *monitoring system* allowing 3-Dimensional recording of the motion of the monitored lead tips **21**a, **22**a during multiple cardiac cycles during intrinsic and paced rhythm. This technique allows correlation with ECG and allows measurement of electromechanical intervals (time from onset of QRS complex to peak contraction, *through plural complete cardiac cycles*) of either lead **21**, **22** and limits respiratory interference. (*FIG. 22*).

FIG. **18** shows dysschronous lead tip **21**a, **22**a movement during intrinsic heart rhythm at a plurality of time stamps. FIG. **19** shows motion of the lead tips **21**a, **22**a during paced rhythm at a plurality of time stamps showing improvement and more synchronous ventricular contraction **55**.

In the absence of an ECG recording, systole is defined as earliest maximal contraction **55** of either ventricular lead tip **21**a, **22**a or in the case of severe akinesis, by the maximal two dimensional shortening between the two ventricular lead tips **21**a, **22**a. Similarly, diastole is defined as earliest maximal relaxation *56* of either ventricular lead tip **21**a, **22**a or in the case of severe akinesis, by the maximal two dimensional lengthening between the ventricular lead tips **21**a, **22**a. When ECG recording is available, electromechanical intervals can be determined such as the onset of QRS to peak contraction *or relaxation, duration of contraction and relaxation intervals* of the left or the right ventricular lead tips **21**a, **22**a respectively *during intrinsic and paced rhythms*.

Left ventricle lead tip **21**a motion and right ventricle lead tip **22**a motion are assessed in the LAO view during intrinsic heart rhythm. The position of both lead tips **21**a, **22**a is identified at each time stamped cine frame using the image compiling system **25**. The lead tip **21**a, **22**a positions are documented at time intervals in two-dimensions (the X-axis correlates roughly with the short axis of the left ventricle **34** in the LAO view; the Y-axis, although also in the short axis of the left ventricle **34**, correlates more directly with respiratory cardiac motion). The lead tip **21**a, **22**a motion data is then transferred to the analytical processing system **27**.

In the X-axis, the motion of the left ventricular lead tip **21**a and motion of the right ventricular lead tip **22**a is plotted showing systole and diastole, lead excursion and the relation of right ventricle **33** to left ventricle **34** upon contraction **55**. The time differential **50** from maximal right ventricle **33** contraction **55** to maximal left ventricle **34** contraction **55** is used to quantify local dyssynchrony **50**. (FIG. **11**, **12**). A zero timing difference (FIG. **11**) is consistent with synchronous

ventricular contraction **55**. A positive timing difference indicates right ventricle **33** maximal contraction **55** precedes left ventricle **34** maximal contraction **55** while a negative timing difference indicates left ventricle **34** maximal contraction **55** precedes right ventricle **33** maximal contraction **55**. (FIG. **12**). Multiple measurements are taken and averaged for consistency.

In two-dimensional analysis (FIGS. **13**, **14** and **20**) fractional shortening is determined for each contractile cycle. Fractional shortening is a measure of heart contractility and is measured using two-dimensional LAO view data to calculate the maximal distances between the lead tips **21**a, **22**a at the start and end of each cardiac cycle using the following formula: Shortening Fraction (%)=(maximal distance from right ventricle lead tip **22**a to left ventricle lead tip **21**a−minimal distance from right ventricle lead tip **22**a to left ventricle lead tip **21**a)×100/(maximal distance from right ventricle lead tip **22**a to left ventricle lead tip **21**a).

The distance measurements are repeated and assessed during biventricular pacing, during right ventricular pacing and during left ventricular pacing, as well as with left ventricular/right ventricular pacing offsets and differing left ventricular pacing configurations. The pacing measurements are then compared with the distance measurements taken during intrinsic heart rhythm.

If there is no significant improvement in dyssynchrony or significant improvement in shortening fraction, consideration is given to altering the pacing offset, changing the pacing configuration, or changing left or right ventricular lead tip **21**a, **22**a position.

Three-dimensional lead tip **21**a, **22**a motion analysis may be performed by using simultaneous bi-plane imaging in left anterior oblique (LAO) and right anterior oblique (RAO) views. In the three-dimensional application of the method, the LAO view is adjusted to represent the true short axis of the left ventricle **34** and represents radial shortening. (FIG. **7**). The RAO view is obtained at a 90° angle. With simultaneous cine in these two views, the left ventricular lead tip **21**a motion data is plotted to determine short axis movement (X and Y axis) and right ventricular lead tip **22**a motion data is plotted to obtain longitudinal motion (Z axis). (FIGS. **15**, **16**). Using X, Y and Z axis coordinates, three-dimensional left ventricular lead tip **21**a motion and three dimensional right ventricular lead tip **22**a motion is determined. Using the three-dimensional technique and analysis thereof, individual lead tip **21**a, **22**a motion, **[**dysschrony**]** *dyssynchrony* and fractional shortening can also be determined and graphed.

Three-dimensional lead tip *21a, 22a,* motion analysis may be obtained using a series of topical patches (not shown) applied to the patient's chest (not shown) using a **[**global**]** *three dimensional* positioning approach to document the ventricular lead tip **21**a, **22**a positions by time. Commercially available **[**motion analysis systems**]** *three dimensional imaging systems*, such as a NAVX system, by St. Jude Medical Inc. may be used to perform the three dimensional lead tip motion analysis *and avoid X-ray exposure*. The data is collected using the right ventricular lead tip **22**a as a reference and the left ventricular lead tip **21**a as input during intrinsic ventricular rhythm and paced ventricular rhythm. Lead tip **21**a, **22**a motion is documented *throughout the cardiac cycle including systole and diastole* during intrinsic heart rhythm and during the paced biventricular rhythm, paced right ventricular rhythm and paced left ventricular rhythm. Other left ventricular pacing configurations and left ventricular/right ventricular pacing offsets may also be documented and assessed. Simultaneous ECG input allows electromechanical measurements of timing from QRS onset to peak mechanical contraction **55** of left or right ventricles **34**, **33** respectively during intrinsic and paced rhythms *throughout plural complete cardiac cycles*.

Having described our method for optimizing CRT, its operation may be understood.

FIG. 22 is a compilation of FIGS. 4, 8, 10, 11 and 12 showing an ECG recording of three successive cardiac cycles showing the P-wave 41, the QRS complex 43 and the T-wave 42 by time. Left ventricular heart wall movement is graphically shown by left ventricular lead 21a movement, and right ventricular heart wall movement is graphically shown by right ventricular lead 22a movement. Maximal ventricular heart wall contraction is shown at 55 (which also marks the beginning of mechanical relaxation) and maximal ventricular heart wall relaxation is shown at 56 (which also marks the beginning of mechanical contraction). 60 is a measure of electromechanical delay between onset of the QRS complex 43 (beginning of electrical depolarization of the heart H) to the beginning of mechanical contraction when there is a timing abnormality leading to dyssynchrony which is shown by dashed line 21b having a maximal delayed contraction at 55b and maximal delayed relaxation at 56b. 62 is a measure of the electromechanical delay between end of the QRS complex 43 (beginning of electrical repolarization of the heart H) to the beginning of mechanical relaxation 55. 63 is a measure of the electromechanical delay between end of the QRS complex 43 (beginning of electrical re-polarization of the heart H), to the beginning of mechanical relaxation 55b when there is a timing abnormality as illustrated by dashed line 21b.

The electromechanical delays 60, 61, 62 and 63 shown in FIG. 22 are only two examples of timing delays that may be identified and examined using our described and claimed method. Other timing delays and periods such as, but not limited to, rate and duration of contraction and/or relaxation and the corresponding phase relationships are likewise contemplated herein and may also be used for therapy, treatment and management of timing disorders of the heart H.

A patient is identified as having perceived ventricular systolic **[**dyschrony**]** *dyssynchrony*. The patient may be identified by diagnostic use of our method using temporary pacing catheters in the right ventricle **33** and coronary sinus (for left ventricular pacing) similar to a diagnostic electrophysiologic study to assess for baseline dyssynchrony and to predict potential response to CRT.

Alternatively, in a patient identified as a candidate for CRT under the current guidelines, our method may be used to optimize lead tip **21**a, **22**a, **23**a positions and improve CRT response during follow-up.

The first step of the method is the implantation of the leads **21**, **22**, **23** into the patient's heart H. Initially, the lead tip **21**a, **22**a, **23**a implantation positions are determined empirically using prior studies that have identified the locations typically generating the greatest physiologic benefit from pacing.

The leads **21**, **22**, **23** are positioned using known catheters and known procedures. As shown in FIG. **1**, the atrial lead **23** is positioned in the right atrium with the atrial lead tip **23**a affixed to the right atrium **31**. The right ventricular lead **22** is positioned in the right ventricle with the right ventricular lead tip **22**a attached to the right ventricular apex or septum **47**. The left ventricular lead **21** is generally placed in a lateral wall **40** position of the left ventricle **34** via the coronary sinus (allowing for anatomic constraints) or **[**epicardially**]** *on the*

left ventricular epicardium. The pacemaker or defibrillator **20** is connected to the leads **21**, **22**, **23** opposite the lead tips **21**a, **22**a, **23**a.

A radiographic imaging system **24** is used to make cine loop image recordings (not shown) of the heart H in the LAO, RAO and AP views (FIG. **17**) through at least three complete cardiac cycles during intrinsic heart rhythm. The cine is at a minimum of 15-30 frames per second and time stamps are recorded on each cine frame. The positions of the left ventricular lead tip **21**a and the right ventricular lead tip **22**a are tracked throughout the cardiac cycles.

The intrinsic rhythm cine loop recordings are converted into an AVI format and transferred to the image compiling system **25**, such as a Tracker™ system from Open Sources Physics, Inc. The X-axis, Y-axis and Z-axis coordinates for the left ventricular lead tip **21**a and the right ventricular lead tip **22**a are determined by the image compiling system **25** and the appropriate time stamps are accorded to each set of coordinates. The compiled data of intrinsic heart rhythm is transferred to the analytical software program **27** to provide a baseline measure of dysschrony and contractility.

The pacemaker **20** is activated and electrical pacing impulses generated within the pacemaker **20** are sent through the leads **21**, **22**, **23** to the lead tips **21**a, **22**a, and **23**a for paced activation of the heart H. Biventricular pacing, right ventricular pacing and left ventricular pacing may be performed and various left ventricular pacing configurations or left ventricular/right ventricular timing offsets may also be assessed and utilized.

The radiographic imaging system **24** is again used to make cine loop image recordings (not shown) of the heart H in the LAO, RAO and AP views through at least three complete cardiac cycles during the paced heart rhythm configurations. The cine is at a minimum of 15-30 frames per second (fps) and time stamps are recorded on each cine frame. The position of the left ventricular lead tip **21**a and the right ventricular lead tip **22**a are tracked throughout the cardiac cycles.

The paced rhythm cine loop recordings are converted into an AVI format and transferred to the image compiling system **25**. The X-axis, Y-axis and Z-axis coordinates for the left ventricular lead tip **21**a and the right ventricular lead tip **22**a are determined by the image compiling system **25** and the appropriate time stamps are accorded to each set of coordinates. The compiled results of paced heart rhythm are transferred to the analytical software program **27** to provide a measure of paced **[**dysschrony**]** *dyssynchrony* and contractility *and ventricular heart wall motion throughout systole and diastole*.

The analytical software program **27** plots the data from the intrinsic heart rhythm and plots the data from the paced heart rhythm on graphs and generates a visual display **26** showing the motion of the lead tips **21**a, **22**a by time. The visual display **26** may be printed or electronically displayed graphs and will show the measures of **[**dysschrony and**]** *dyssynchrony,* contractility *and ventricular heart wall motion throughout systole and diastole* for both intrinsic heart rhythm and the paced heart rhythm.

The visual display is interpreted by the physician performing the procedure to determine if there has been improvement in **[**dysschrony**]** *dyssynchrony* and an improvement in contractility *or ventricular heart wall motion* as a result of the pacing.

If assessment of the results shows no significant improvement in contractility, **[**or significant improvement in dysschrony**]** *dyssynchrony or ventricular heart wall motion*, the physician may re-assess pacing with an alternative left ventricular pacing configuration, such as using left ventricular/right ventricular pacing offsets, or move the left ventricular lead tip **21**a to another position on the heart H such as to a more atypical position on the lateral wall **40**, and/or the physician may change the position of the right ventricular lead tip **22**a. The physician may also change offset of the pacemaker **20** to change the timing of the electrical impulses directed to the ventricular lead tips **21**a, **22**a.

The procedure for making a cine loop recording of the paced heart rhythm is repeated for the new lead tip **21**a, **22**a positions in the LAO, RAO and AP views and the data is exported for compiling, analysis and comparison against the intrinsic heart rhythm data. If no significant improvement is shown as a result of the new lead tip **21**a, **22**a position, the procedure may be repeated until improvement is achieved or patient condition requires the procedure be discontinued.

If assessment of the results shows only minimal improvement in contractility or minimal improvement in dysschrony, the physician will record the positions of the lead tips **21**a, **22**a in the heart H and then may change the positions of the lead tips **21**a, **22**a to improve the effects of pacing. The procedure for making a cine loop recording of the paced heart rhythm is repeated for the new lead tip **21**a, **22**a positions in the LAO, RAO and AP views and the data is exported for compiling, analysis and comparison against the intrinsic heart rhythm data. If no significant improvement is shown as a result of the new placement, the procedure may be repeated again or the lead tips **21**a, **22**a may be repositioned to the earlier position that showed some improvement with pacing.

If assessment of the results shows significant improvement in contractility and significant improvement in **[**dysschrony**]** *dyssynchrony and ventricular heart wall motion*, the physician will end the procedure.

Use of our method with a three dimensional mapping system such as NAVX (St Jude Medical Inc) allows three dimensional lead tip motion assessment in similar fashion without the detrimental effects of X-ray exposure and also provides ECG correlation as previously described.

This method may also be utilized during routine follow-up of patients with CRT, utilizing external patches and CRT analysis to provide lead tip motion analysis during office reprogramming to maximize CRT therapy. *This method, by either X-Ray or three dimensional mapping system, may be utilized diagnostically or therapeutically to define electromechanical measures of contraction and ventricular heart wall motion, including responses to pacing, to uniquely assess systolic and diastolic heart failure and guide medical and CRT therapy.*

The foregoing description of our invention is necessarily of a detailed nature so that a specific embodiment of its best mode may be set forth as is require, but it is to be understood that various modifications of details, and rearrangement, substitution and multiplication of steps and apparatus may be resorted to without departing from its spirit, essence or scope.

Having thusly described our invention, what we desire to protect by Letters Patent, and

What we claim is:

1. A method for determining and optimizing left ventricular synchrony during cardiac resynchronization therapy comprising in combination:

identifying a patient as having perceived ventricular systolic dyssynchrony who may benefit from cardiac resynchronization therapy;

implanting left ventricular and right ventricular leads in the patient's heart, each lead having a lead tip at a first end portion;

positioning each lead tip initially at a location prior published studies have shown generate the greatest physiologic benefit from pacing;

connecting a pacemaker to the plural leads opposite the lead tips;

using a radiographic imaging system to make cine loop image recordings of the patient's heart in left anterior oblique, right anterior oblique and anterior-posterior views through at least three complete cardiac cycles during intrinsic heart rhythm and assigning time stamps to each cine frame to ascertain the position of each lead tip throughout the intrinsic cardiac cycles;

transferring the intrinsic rhythm cine loop image recordings to an image compiling system for compiling into intrinsic heart rhythm data and for determining an X-axis, a Y-axis and a Z-axis coordinate for each lead tip for each time stamped cine frame;

transferring the X-axis, the Y-axis and the Z-axis coordinate for each lead tip for each intrinsic rhythm time stamped cine frame to an analytical software program to determine a baseline measure of dyssynchrony and contractility at the current lead tip locations in the patient's heart;

activating the pacemaker to send electrical pacing impulses through the ventricular leads to the lead tips for paced activation of the patient's heart;

using the radiographic imaging system to make cine loop image recordings of the heart in left anterior oblique, right anterior oblique and anterior-posterior views through at least three complete cardiac cycles during the paced heart rhythm and assigning time stamps to each cine frame to ascertain the position of each lead tip throughout the paced cardiac cycles;

transferring the paced rhythm cine loop image recordings to the image compiling system for compiling into paced heart rhythm data and for determining the X-axis, Y-axis and Z-axis coordinate for each lead tip for each paced time stamped cine frame;

transferring the X-axis, the Y-axis and the Z-axis coordinate for each lead tip for each paced rhythm time stamped cine frame to the analytical software program to determine a measure of paced ventricular dyssynchrony and paced ventricular contractility at the current lead tip locations in the patient's heart;

plotting the intrinsic heart rhythm coordinate data and plotting the paced heart rhythm coordinate data and generating a visual display showing the motion of the lead tips by time so that the intrinsic heart rhythm coordinate data may be compared against the paced heart rhythm coordinate data;

interpreting the intrinsic heart rhythm coordinate data and the paced heart rhythm coordinate data to determine if the paced activation of the patient's heart decreases the ventricular dyssynchrony relative to the intrinsic ventricular dyssynchrony and increases ventricular contractility relative to the intrinsic contractility; and

ending the cardiac resynchronization therapy if the interpretation of the paced heart rhythm data compared against the intrinsic heart rhythm data shows increased contractility and increased synchrony with paced activation of the patient's heart at the current lead tip locations.

2. The method for determining and optimizing left ventricular synchrony of claim **1** wherein a right ventricular lead tip is located on the patient's heart's right ventricular septum.

3. The method for determining and optimizing left ventricular synchrony of claim **1** wherein a right ventricular lead tip is located on the patient's heart's right ventricular apex.

4. The method for determining and optimizing left ventricular synchrony of claim **1** wherein: a left ventricular lead tip is located on a left ventricular lateral wall.

5. The method for determining and optimizing left ventricular synchrony of claim **1** wherein: a left ventricular lead tip is located on a left ventricular anterolateral wall.

6. The method for determining and optimizing left ventricular synchrony of claim **1** wherein: a left ventricular lead tip is located on a left ventricular posterolateral branch of the coronary sinus.

7. The method for determining and optimizing left ventricular synchrony of claim **1** further comprising:

if interpretation of the results shows no improvement in contractility and no improvement in synchrony, documenting the position of the lead tips in the heart;

changing the position of at least one lead tip; and

repeating the steps of claim **1** for activating the pacemaker, imaging, compiling, identifying coordinate positions and comparing the paced heart rhythm data against the intrinsic heart rhythm data.

8. The method for determining and optimizing left ventricular synchrony of claim **1** further comprising:

if interpretation of the results shows minimal improvement in contractility and minimal improvement in synchrony, documenting the position of the lead tips in the heart;

changing the position of at least one lead tip; and

repeating the steps of claim **1** for activating the pacemaker, imaging, compiling, identifying coordinate positions and comparing the paced heart rhythm data against the intrinsic heart rhythm data.

9. The method for determining and optimizing left ventricular synchrony of claim **1** further comprising:

if interpretation of the results shows minimal improvement in contractility and minimal improvement in synchrony, documenting the position of the lead tips in the heart;

changing the pacing configuration; and

repeating the steps of claim **1** for activating the pacemaker, imaging, compiling, identifying coordinate positions and comparing the paced heart rhythm data against the intrinsic heart rhythm data.

10. The method for determining and optimizing left ventricular synchrony of claim **1** further comprising:

if interpretation of the results shows minimal improvement in contractility and minimal improvement in synchrony, documenting the position of the lead tips in the heart;

changing the ventricular pacing offsets; and

repeating the steps of claim **1** for activating the pacemaker, imaging, compiling, identifying coordinate positions and comparing the paced heart rhythm data against the intrinsic heart rhythm data.

11. The method for determining and optimizing left ventricular synchrony of claim **1** further comprising:

if interpretation of the results shows minimal improvement in contractility and minimal improvement in synchrony, documenting the position of the lead tips in the heart;

changing the timing of the electrical impulses; and

repeating the steps of claim **1** for imaging, compiling, identifying coordinate positions and comparing the paced heart rhythm data against the intrinsic heart rhythm data.

12. The method for determining and optimizing left ventricular synchrony of claim **1** **[**further comprising**]** *wherein*:

[using**]** a three dimensional **[**mapping**]** system *is used* to generate **[**a**]** three dimensional lead **[**tip**]** motion assess-

ment [without] *instead of X-ray to avoid* the detrimental effects of X-ray exposure [to provide ability] to determine electromechanical measurements related to dyssynchrony.

13. The method for determining and optimizing left ventricular synchrony of claim [**1** wherein] *12 further comprising*:

[the method is utilized] during routine follow-up care of patients having previously undergone cardiac resynchronization therapy[;] using plural topical heart monitor patches to provide three dimensional analysis [and the present method] to provide lead tip motion analysis during office reprogramming of the pacemaker to maximize long term benefits of cardiac resynchronization therapy.

14. The method for determining and optimizing left ventricular synchrony of claim [**1**] *12* wherein:

three-dimensional lead tip motion analysis is performed [using simultaneous bi-plane imaging in left anterior oblique and right anterior oblique imaging views;

the left anterior oblique view is adjusted to represent short axis of the left ventricle to show radial shortening;

the right anterior oblique view is obtained at a 90° angle;

simultaneous cine is performed in the two views;

the left ventricular lead tip motion data is plotted to determine short axis movement (X and Y axis) and right ventricular lead tip motion data is plotted to obtain longitudinal motion (Z axis);

using the X, Y and Z axis coordinates, three-dimensional left ventricular lead tip motion and three dimensional right ventricular lead tip motion is determined] *without the detrimental effects of x-ray to determine left ventricular lead tip motion and right ventricular lead tip motion* to graph and analyze dyssynchrony [and], fractional shortening, *and heart wall movement*.

15. The method for determining and optimizing left ventricular synchrony of claim [**1**] *12* wherein:

three-dimensional lead tip motion analysis is obtained using plural topical patches applied to the patient's chest using a global positioning approach to document the ventricular lead tip positions by time;

data is collected using [the right ventricular] *one* lead tip as a reference and [the left ventricular] *a second* lead tip as input during intrinsic ventricular rhythm and various configurations of paced ventricular rhythm;

lead tip motion is documented during intrinsic heart rhythm and during the paced biventricular rhythm including assessing right and left ventricular pacing offsets, paced right ventricular rhythm and paced left ventricular rhythm, at differing [right and left ventricular] lead locations *and differing pacing configurations*; and

simultaneous ECG [input] provides electromechanical measurements of timing from QRS onset to peak mechanical contraction of left and right ventricles during intrinsic and paced rhythms.

16. The method for determining and optimizing left ventricular synchrony of claim **1** wherein:

the patient is identified as a candidate for cardiac resynchronization therapy by diagnostic use of the method using temporary pacing catheters in the right ventricle and coronary sinus to assess for baseline dyssynchrony and to predict potential response to cardiac resynchronization therapy.

17. The method for determining and optimizing left ventricular synchrony of claim **1** wherein:

the cine is not less than 15 frames per second and time stamps are recorded on each cine frame.

18. A method for determining and optimizing ventricular heart wall motion in a patient having ventricular heart wall movement dysfunction comprising in combination:

identifying a patient as having perceived ventricular heart wall movement dysfunction who may benefit from therapy;

placing pacing leads having electrodes at a lead tip end portion in the patient's heart;

positioning each lead initially at a location published studies have shown to generate physiologic benefit from pacing;

using radiographic imaging to make recordings of the heart in multiple views through plural complete cardiac cycles including systole and diastole during intrinsic heart rhythm and assigning a time stamp to ascertain the position of a lead throughout the cardiac cycles including systole and diastole;

determining an X-axis, a Y-axis, and a Z-axis coordinate for the lead position for the time stamp;

compiling the intrinsic heart rhythm data;

analyzing the compiled intrinsic heart rhythm data to determine a baseline measure of dyssynchrony contractility and/or ventricular heart wall motion through the plural complete cardiac cycles including systole and diastole;

applying an electrical signal to the leads to pace the heart;

using radiographic imaging to make recordings of the heart in multiple views through plural complete cardiac cycles including systole and diastole during paced heart rhythm and assigning a time stamp to ascertain the position of each lead throughout the plural complete cardiac cycles including systole and diastole;

determining an X-axis, a Y-axis, and a Z-axis coordinate for each lead position for each time stamp;

compiling the paced heart rhythm data;

analyzing the compiled paced heart rhythm data to determine a measure of dyssynchrony and contractility through plural complete cardiac cycles including systole and diastole;

comparing the intrinsic heart rhythm data and the paced heart rhythm data and generating a visual display showing the motion of the leads throughout the plural complete cardiac cycles by time;

interpreting the intrinsic heart rhythm data and the paced heart rhythm data to determine if there is an improvement in dyssynchrony, contractility or ventricular heart wall movement throughout the plural complete cardiac cycles resulting from pacing;

if interpretation of the paced heart rhythm data shows no improvement or minimal improvement in synchrony, contractility, or or ventricular heart wall movement, documenting the position of the lead tips in the heart;

changing the pacing configurations, and/or changing a lead tip position and repeating the imaging, compiling, identifying coordinate positions, and comparing the paced heart rhythm data and intrinsic heart rhythm data; and

ending the procedure if assessment of the paced heart rhythm data shows improvement in synchrony, contractility, or ventricular heart wall movement.

19. The method for determining and optimizing ventricular heart wall motion of claim 18 further comprising:

simultaneous electrocardiographic (ECG) monitoring to determine electromechanical and mechanical measurements of ventricular heart wall movement, electrical depolarization and re-polarization throughout the plural complete cardiac cycles, rates of systolic and dias-

tolic ventricular wall motion, duration of systole and diastole, during intrinsic and paced rhythm, for diagnostic assessment of systolic and diastolic heart failure, and to guide therapy.

20. A method for determining and optimizing ventricular heart wall motion in a patient having ventricular heart wall movement dysfunction comprising in combination:

identifying a patient as having perceived ventricular heart wall movement dysfunction who may benefit from therapy;

placing pacing leads having electrodes at a lead tip end portion in the patient's heart;

positioning each lead initially at a location published studies have shown to generate physiologic benefit from pacing;

using three dimensional imaging to make recordings of the lead electrodes movement through plural complete cardiac cycles including systole and diastole during intrinsic heart rhythm and assigning a time stamp to ascertain the position of each electrode throughout the cardiac cycles;

determining an X-axis, a Y-axis, and a Z-axis coordinate for each electrode position for the time stamps;

compiling the intrinsic heart rhythm data;

analyzing the compiled intrinsic heart rhythm data to determine a baseline measure of dyssynchrony, contractility and ventricular heart wall motion throughout the plural complete cardiac cycles including systole and diastole;

applying an electrical stimulus to the leads to pace the heart;

using three dimensional imaging to make recordings of the lead electrodes movement through plural complete cardiac cycles including systole and diastole during paced heart rhythm and assigning a time stamp to ascertain the position of each electrode position throughout the plural complete cardiac cycles;

determining an X-axis, a Y-axis, and a Z-axis coordinate for each electrode position for the time stamps;

compiling the paced heart rhythm data;

analyzing the compiled paced heart rhythm data to determine a measure of dyssynchrony, contractility and ventricular heart wall motion throughout the plural complete cardiac cycles including systole and diastole;

comparing the intrinsic heart rhythm data and the paced heart rhythm data and generating a visual display showing the motion of the lead throughout the plural complete cardiac cycles by time;

interpreting the intrinsic heart rhythm data and the paced heart rhythm data to determine if there is improvement in dyssynchrony, contractility and ventricular heart wall motion throughout the plural complete cardiac resulting from the pacing; and

if interpretation of the intrinsic heart rhythm data and the paced heart rhythm data shows no/minimal improvement in synchrony, contractility, or ventricular heart wall movement, documenting the position of the leads in the heart;

changing the lead position and/or changing a pacing configuration and/or changing ventricular pacing offsets; and

repeating the steps of claim 20 for imaging, compiling, identifying coordinate positions, and comparing the intrinsic heart rhythm data and the paced heart rhythm data; and

ending the procedure if assessment of the results shows improvement in synchrony, contractility, or ventricular heart wall movement.

21. The method for determining and optimizing ventricular heart wall motion of claim 20 wherein: one lead is positioned in the patient's right ventricle to optimize ventricular synchrony, ventricular contractility, and/or ventricular heart wall motion.

22. The method for determining and optimizing ventricular heart wall motion of claim 20 wherein: one lead is positioned in the patient's coronary venous system to optimize ventricular synchrony, ventricular contractility, and/or ventricular heart wall motion.

23. The method for determining and optimizing ventricular heart wall motion of claim 20 wherein: one lead is positioned on the patient's left ventricular epicardium to optimize ventricular synchrony, ventricular contractility, and/or ventricular heart wall motion.

24. The method for determining and optimizing ventricular heart wall motion of claim 20 further comprising:

simultaneous ECG to determine electromechanical and mechanical measurements of ventricular heart wall movement related to dyssynchrony, contractility, and electrical depolarization and repolarization ventricular heart wall motion throughout the plural complete cardiac cycles, as a diagnostic tool during lead implant and during cardiac resynchronization therapy follow up.

25. The method for determining and optimizing ventricular heart wall motion of claim 20 further comprising:

simultaneous electrocardiographic (ECG) monitoring to determine electromechanical and mechanical measurements of contraction and ventricular heart wall motion including electrical depolarization and re-polarization throughout the plural complete cardiac cycles, rates of systolic and diastolic ventricular heart wall motion and relative duration of systole and diastole during intrinsic and paced rhythm for diagnostic assessment of systolic and diastolic heart failure to guide therapy.

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