Extracardiac Recordings of His-Purkinje Activity During Conduction Disorders and Junctional Rhythms

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SUMMARY

Previous investigations have demonstrated a surface recording technique using signal averaging to detect electrical activity during the "isoelectric" P-R segment. Various physiological and pharmacological interventions suggest that the source of these potentials is the His-Purkinje system (HPS). In order to assess the sensitivity of averaged recordings to changes in HPS activation, i.e., conduction defects in the HPS, recordings were made directly from the heart surface using a bipolar, anterior-posterior epicardial lead in 13 dogs which underwent thoracotomy. The signal was amplified, filtered and averaged using a digital computer for purposes of signal enhancement. The epicardial averaged lead (EAL) contained activity coincident with HPS depolarization and similar to those recorded by leads on the body surface of intact dogs from previous studies. The standard ECG and His bundle electrogram from an electrode catheter served as references in localizing and assessing several conduction disorders experimentally produced by traumatic and ischemic injury. Among the disorders produced were: 1) atrioventricular (A-V) nodal block which resulted in loss of recorded activity in the EAL following the P wave. 2) First and second degree intra-His bundle block produced by anterior septal artery ligation showed split His potentials in the HBE (1°) and 2:1 conduction with block in the His bundle (2°). In the blocked beats the EAL showed a reproducible portion of the activity coincident with proximal His bundle activity of the split His potentials in both cases. 3) In four cases of proximal right bundle branch block produced by anterior septal artery ligation the relatively proximal portions of HPS activity in the EAL showed marked diminution. 4) Two cases of distal His bundle or bilateral bundle branch delay were seen as prolonged H-V time and a normal QRS pattern. The early and late portions of the HPS activity in the EAL were not markedly changed while the middle portion was prolonged and fractionated. 5) Junctional rhythms produced by crushing the SA node resulted in no atrial activity occurring prior to HPS depolarization in the EAL. However, the QRS was preceded by HPS activity whose onset was coincident with the H recorded in the His bundle electrogram. The EAL showed consistent and reproducible morphology and timing of HPS activity at different heart rates during normal conduction and consistent alterations of the HPS activity during abnormal conduction.

Additional Indexing Words:
- Electrophysiology
- Ischemic injury
- Heart rate
- His bundle electrogram
- Trauma

A TECHNIQUE has been recently described to record electrical activity from the His-Purkinje system (HPS) in both the experimental animal and man using body surface electrodes. The technique employs signal averaging which will enhance a periodic signal that is buried in noise. Using increased amplifier gain, a narrow bandwidth, and greater time resolution a selected portion of the surface ECG, in this case the P-R segment, can be enhanced so that reproducible waveforms preceding the QRS are observed. This waveform usually has an onset concomitant with the His bundle activation when compared to a simultaneously recorded His bundle electrogram. The HPS waveform does not change its relationship to ventricular activation (V) nor its morphology at different atrial paced rates in dogs. Administration of procaine amide will cause an increase in HPS-V time proportional to the H-V time measured by the His bundle electrogram. In the present report further validation of the presumed HPS waveform was performed by producing conduction disorders in the canine heart during which the HPS waveforms were monitored. Since all of these disorders involved a thoracotomy, a lead system using epicardial electrodes was employed. However, comparative body surface recordings from a previous...
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study² are presented for qualitative and quantitative correlation with extracardiac recordings.

Methods

Fifteen adult mongrel dogs weighing 10–20 kg were anesthetized with intravenously administered sodium pentobarbital (30 mg/kg). The animals were intubated and placed on a mechanical respirator. ECG leads II and aV3 were continuously displayed (E for M, DR 8). Either a right or left thoracotomy was made through the 4th intercostal space. The pericardium was incised and the heart exposed. Two teflon-coated silver wires (0.005 in); with the ends bare for 0.25 inch were inserted subepicardially at the base of the ventricle, using hypodermic needles, to form a bipolar with an anterior-posterior axis. One wire was placed in the epicardium adjacent to the origin of the left posterior descending artery (−pole); the other wire was placed at the base of the pulmonary artery (+pole). They were in the same plane (anterior-posterior) when either the right or left sided approach was used. The epicardial wires were then led to the input of a battery powered preamplifier (Princeton Applied Research 113) having a gain of 1000. This signal was filtered (Krohn-Hite 3750) between 80 and 300 Hz (24 dB/oct roll-off) and further amplified at gains varying between 25 and 300 (Analog Devices 171). The conditioned epicardial lead was then averaged using one of two schemes of signal averaging. The first, described in more detail in a previous paper,² used an atrial pacing pulse (2 msec duration, 7–15 volts at rates between 120 and 270/min from a Grass S88 stimulator and SIU5 isolation unit) as a trigger to a hard wired averager (Hewlett Packard 5480). This device selects a window of desired width (usually 100 msec) of the cardiac cycle relative to the paced pulse. The addition of these successive windows eliminates the random noise and accumulates the signals which are periodic and temporally related to the fiducial point, i.e., signals associated with the cardiac cycle. It is possible to insert a time delay following the paced pulse so that a window comprising the P-R segment is averaged. It is also possible to inhibit the selection of every other window so that either the conducted or blocked beats of 2:1 conduction can be selectively studied. This scheme of averaging is limited to cases where atrial pacing is used and cannot be used when the time between the paced pulse and events of interest (HPS conduction) is inexact.

The second averaging scheme utilized a specially designed QRS detector similar in principle to one described by Brandlon and Brody³ for determining a fiducial point of the ECG. The over-all magnitude of the QRS and amplitude of the first derivative of the QRS were the criteria used for accurate QRS detection. Using the triggered sweep of an oscilloscope (Tektronix 568) the observed consistency of QRS detection was monitored and only when an accuracy of ±1 msec was achieved was an animal further studied. With the assurance of accurate QRS detection the epicardial lead, standard ECG and/or His bundle electrogram were digitized (Hewlett-Packard 5610, 10 bit analog/digital converter) at a sampling rate of 2.5 KHz per channel and fed into a digital computer (Hewlett Packard 2100A) for signal averaging via appropriate software. The program allowed the user to select a window (usually 100 msec) which occurred up to 200 msec prior to the fiducial point generated by the QRS detector.

Reproducible records were obtained at several paced heart rates. Reproducibility of the averaged waveforms was used for determining the number of cardiac cycles necessary for a valid average. This was usually between 16 and 64 depending upon the amount of noise present or physiological variability during dynamic disease states, e.g., ischemic injury due to artery ligation (see below). In cases where complete separation of atrial activity and HPS activity could not be obtained using atrial pacing alone, propranolol or practolol (1 mg/kg) was administered intravenously. After initial recordings of the epicardial averaged lead were obtained, an electrode catheter was inserted via the common carotid artery to obtain the His bundle electrogram (HBE) at the aortic root. Control records were then repeated to insure that the electrode catheter recording did not influence the interpretation of the HPS waveform recorded initially.

The conduction disorders within the HPS were produced by anterior septal artery ligation and rapid atrial pacing. Intra-His bundle block was diagnosed by continuous monitoring of the HBE which showed widening and loss of amplitude of the H potential (first degree block). With further ischemia the H fragmented with block usually occurring between the split H and H′ (second degree block).⁸ Bilateral bundle branch delay or distal His bundle block was interpreted from recordings of a normal QRS pattern with a prolonged H-V time. No evidence of a split His bundle potential was observed in the HBE.

Right bundle branch block (RBBB) was seen as a marked decrease of the amplitude of the R wave in lead II and the S wave in lead aV3 with the development of a terminal, broad, frequently slurred S wave in lead II and an R′ in lead aV6.¹⁰

Local injection of formaldehyde into the A-V nodal region through the anterior right atrial wall resulted in complete A-V dissociation.¹¹ No H potential followed the atrial activity in the HBE, but each QRS was preceded by an H potential.

A-V junctional rhythms were produced by crushing the SA nodal area. Atrial activity either slightly preceded or occurred simultaneously with or after the QRS complex. The QRS was essentially unchanged from the QRS complex of sinus rhythm.

Results

Comparative records obtained using four surface averaged leads (SAL) and four epicardial averaged leads (EAL) from eight different dogs are shown in panels A and C of figure 1. Control records were always obtained prior to inserting the His bundle recording catheter so that quantitation of the duration of the HPS waveform (HPS-V time) would not be biased by the more direct catheter measurement of the H-V time. Also, the possibility of cross-talk interference between the HBE and EAL was eliminated. In two cases this sequential procedure allowed a correlation of HPS-V times in which the initial SAL and EAL records indicated an abnormally long H-V interval, panels B and D. In each of these panels the subsequently recorded HBE confirmed the abnormally long H-V time. Each record shown in this figure was obtained during fast atrial pacing, allowing maximal time separation between atrial and ventricular activity. The EAL and adjacent SAL recordings are from different dogs, but this series of traces indicates the general similarities of each HPS waveform. In any individual dog, comparison of several successively ob-
tained averages will indicate where the reproducibility of the HPS waveform begins. In general the onset of the HPS waveform is determined during fast pacing rates and is characterized by large changes in the baseline of the P-R segment occurring prior to ventricular activation as measured on the standard ECG. The HPS waveform comprises the entire interval from this onset to initiation of the QRS.

Another method for validation of the averaged recordings and determination of HPS onset is the reproducibility of the HPS waveform at several paced rates. The need for higher paced rates and the concomitant increase of A-HPS time is apparent in figure 2. Panel A shows a reproducible HPS waveform that is contaminated by atrial activity. Increasing the pacing rate shows that indeed the HPS waveform is reproducible. At high rates a large temporal shift of atrial activity occurs due to A-V nodal delay and a clear point of onset of the HPS waveform is apparent. In several animals, pacing alone could not achieve this degree of separation of atrial and HPS activity. In these cases 1 mg/kg of propranolol or practolol was administered to increase A-V nodal conduction time.

Conduction Disorders

With the production of complete A-V nodal block by local injection of formaldehyde, a slow idioventricular rhythm followed (fig. 3B). During atrial pac-
ing, the pacer impulse was used as a fiducial point for a signal averaging window encompassing 100 msec after the end of atrial activation. Figure 3A shows the signal after averaging 32 atrial cycles, without contamination by dissociated ventricular activity. Note that following the end of the A wave, low amplitude activity (2 μV) was recorded and no reproducibility between the two successively obtained EAL recordings can be appreciated. Previous recording of HPS activity using EAL showed reproducible waveforms on the order of 20–50 μV (see figs. 1 and 2).

Anterior septal artery ligation and rapid atrial pacing has been shown to be effective in producing several forms of proximal HPS conduction disorders. Often seen with this preparation are first or second degree block within the His bundle. First degree block occurs when the His bundle deflection widens and eventually splits into H and H' deflections, but 1:1 conduction is maintained. Figure 4A is a control record displaying the ECG, HBE and EAL traces. Panels B and C, taken during ischemic injury, show the His deflection split into H and H' deflections at rates of 120 and 150 beats/min, respectively. Panel D, taken at a rate of 180 shows a further fragmentation of the H deflection. The EAL recordings in each of these panels indicate that electrical activity is recorded throughout this period of prolonged His bundle conduction. The approximate 15 μV level of this activity is less than the normal amplitude of the HPS waveform but is significantly greater in magnitude than the 2 μV levels shown during A-V nodal block in figure 3.

In another animal with anterior septal artery ligation 2:1 intra-His bundle block occurred. This can be verified by the His bundle electrogram since each blocked beat consists of atrial activity and His bundle activity (fig. 5E). By front panel control of the Hewlett-Packard 5480 signal averager, it is possible to accept every other beat for averaging. In this case of 2:1 block either the blocked beat or the conducted beat can be averaged. This applies only when the atrial pacing pulse is used as the fiducial point for the averaging window. Figure 5A shows a control record prior to ischemia which includes the ECG, HBE and EAL recordings. When second degree intra-His bundle block occurred only the blocked beat was averaged and is shown in panels B-D. In each of these panels, as the rate is increased and the A-H time of the blocked beat increases, a reproducible wave in the EAL occurs simultaneously with His bundle activation in the HBE. Note the constant time relationship between the H deflection in the HBE and the prominent peak deflection in the EAL labeled H. While this EAL peak shows a fixed relationship to the HBE, other portions of the EAL do not, indicating they are atrial in origin. The magnitude of this EAL deflection is about 15 μV which is similar to the fractionated activity shown in figure 4.

Another conduction disturbance after anterior septal artery ligation was characterized by a normal QRS in the ECG and an increased H-V time in the HBE. The control record of figure 6A has an H-V time of 37 msec and HPS-V time of 34 msec. After ischemic injury the H-V time increased to 46 msec and the HPS-V time increased to 44 msec. The early and late portions of the HPS waveform show a clear similarity before and after ligation. The dissimilarity of the HPS waveform in each EAL occurs mainly in the middle portion, corresponding to the time during which the distal His bundle and main bundle branches would be activated.

Right bundle branch block (RBBB) patterns in the ECG leads were often observed after anterior septal artery ligation. Figure 7 shows the ECG, HBE and EAL in two such cases of RBBB. The upper EAL in each panel is the control while the lower EAL was ob-
tained after ischemic injury. There is a diminution in amplitude of almost the entire HPS waveform, while atrial activity in the EAL did not change in magnitude. This negates a possible change in recording gain.

A-V Junctional Rhythms

It is possible to obtain any of three relationships between atrial and ventricular activity during A-V junctional rhythms after crushing the SA node. In the first case portions of atrial activity would superimpose upon HPS activity and prevent possible averaging by overloading the high gain amplifier system. When atrial activity occurs simultaneously with or after ventricular activity, this contamination of the HPS activity would not occur. Figure 8 shows data from two experiments where junctional rhythms were produced. In panels A and B, respectively, the control and junctional rhythm are shown. The A wave follows the His deflection and immediately precedes ventricular activation in the HBE of figure 8B. A part of the EAL in this case is contaminated with atrial activity and the HPS waveform does not appear to be reproducible. In another experiment (panels C and D) during the junctional rhythm atrial activity occurs within the QRS complex. The EAL in panel D shows prenventricular activity occurring simultaneously with the onset of His bundle activation (HBE). The distal portions of the HPS waveform are reproducible during supraventricular and junctional rhythms; however there are differences in the proximal portions of the HPS waveforms. These findings are discussed below.

Discussion

Signal averaging has been previously employed to record prenventricular activity in body surface leads. The signals measured approximately 1–7 μV and required high amplification and averaging between 128 and 1024 cardiac cycles. In the present study an epicardial lead was utilized for several reasons. Placing electrodes closer to the HPS would allow recording of greater potentials generated by these structures. Also, surface electrodes are subject to many more bioelectric sources, thus increasing the amount of “noise” present, e.g., electrical activity of chest wall muscles. By enhancing the desired potential and reducing the sources of noise the signal-to-noise ratio is increased. This lessens the number of averaged cycles necessary to obtain reproducible data to as few as 16 and generally no higher than 64. By using the epicardial leads interventions leading to conduction disorders could be made and compared to a control state as opposed to utilizing surface recordings before and after thoracotomy. The thoracotomy itself would change the volume conductor properties of the chest and comparison of surface recordings before and after could be invalid.

Figure 1 shows the essential similarities between the HPS waveform of the EAL after thoracotomy and that of the SAL in a different series of dogs without thoracotomy. The lead axes for both types of recordings were the same (antero-posterior axis). Thus, except for the differences in the magnitude a simple linear transformation can be made between the
Second degree intra-His bundle block as a result of anterior septal artery ligation. Panel E shows leads II, aV_{1}, and HBE indicating 2:1 conduction with block occurring distal to the recorded H deflection. Control records of the ECG, HBE and EAL are shown in panel A before anterior septal artery ligation. Averaging only activity following the blocked P wave is shown in panels B, C and D at rates of 120, 160 and 180 beats/min, respectively. Note the peak in the EAL, labeled H, which is temporally linked with the H deflection of the HBE at each heart rate. The time lines of panel E are 1 sec apart. (Each trace was obtained after 16 cardiac cycles.)

Alteration of the EAL recording as the result of right bundle branch block (RBBB) induced by ischemia. Each panel of this figure shows the ECG, HBE and EAL prior to RBBB. After anterior septal artery ligation RBBB was observed in the ECG. Alteration of the HPS waveform is shown in the lower EAL of each panel. Note that the magnitude of atrial activity in the EAL is maintained before and after injury, but there is an over-all reduction of the HPS magnitude.
levels within the volume conductor. Two qualitative tests were used to validate each recorded HPS waveform. First, before any single HPS waveform can be considered valid it must be compared to another taken successively so that reproducibility can be assured. This rules out contamination by asynchronous periodic noise such as 60 Hz interference or that due to respiration. The second validating test is the simultaneous averaging of a signal of known morphology. For example all of the control records had an ECG and/or HBE simultaneously averaged. Both of these signals are recorded with less amplification, are less noisy, and can be readily identified compared to EAL recordings. If after the averaging procedure the H deflection of the HBE or the Q wave of the ECG appeared slurred or otherwise deformed in relation to a single His bundle or QRS deflection, then the time window position was not constant in the record obtained. These two tests provide reliable guidelines for accepting any particular averaged record as a valid one.

Another important point of validation involves the amplitude of the recorded signals. In a previous study we showed that a 500 Hz sine wave of 1 volt magnitude delivered at the end of an electrode catheter in the aortic root of the dog could be detected on the surface of the chest. Depending upon the lead axis, the magnitude of this test signal varied between 1-20 mV. This simulated a dipole source in the area of the His bundle with a large enough voltage so that measurement could be made using standard equipment. This is essentially an empirical method for determining the attenuation of the thorax. In this case the attenuation factor would be between 1000 and 50. Applying this attenuation factor to the actual His bundle source potential (approximately 0.5 mV as recorded with the His bundle catheter in the aortic root) then the surface lead would record a 0.5-10 µV potential from the His bundle. The HPS waveform recorded on the body surface of the dog by using signal averaging measured 1-7 µV. It follows that the closer one records to the source of the signal the higher the amplitude of the signal recorded. Accordingly, the anterior-posterior leads on the heart surface not only showed the same morphology as a similarly recorded surface lead (fig. 1) but the amplitudes were considerably greater: EAL = 25-50 µV and SAL = 1-7 µV. Furthermore, baseline noise level using the EAL was found to be in the order of 2 µV (see fig. 3). This value can be used to assess reproducible waveforms recorded from the HPS under normal or pathologic conditions. Thus, in figure 5C and D the EAL shows signals whose amplitudes are 5-15 µV. The larger amplitude signal has a constant time relationship to the recorded portion of the split His potential (H) in the blocked beats. However, several smaller amplitude deflections follow this deflection. These may be due to some form of decremental and variable conduction in the ischemic His bundle and these potentials were not reproducible on a beat-to-beat basis. During such dynamic processes the uses of signal averaging may be limited.

Conduction velocity may be an important factor in determining the amplitude and morphology of the HPS waveform. If conduction slows in a structure then a functional disorder may appear. For example, the ECG criteria for RBBB may be the result of slow conduction rather than complete cessation of conduction in the right bundle. Conduction slowing in the His bundle or equal delay in both bundle branches may result in increased H-V time and no change in the ECG. In the case of split His potentials (fig. 4) various longitudinal or lateral portions of the His bundle may be exhibiting conduction slowing. The EAL
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depicts this as a fractionated, low magnitude waveform which may be difficult to interpret since its amplitude approaches the system noise level and its occurrence may be variable from beat to beat, thus causing slurring and attenuation, i.e., loss of reproducibility. In addition, like the A-V node, these damaged areas of the HPS will generate small signals with a frequency content lower than the 80 Hz cutoff (fig. 4D).

With the production of RBBB it was observed that there was an overall decrease in the magnitude of the HPS signal (fig. 7). It cannot be entirely appreciated why this overall change occurred since the right bundle probably represents a smaller tissue mass than the left bundle, whose normal activation should appear at this time. The particular lead axis used (see Methods) may favor the recording of right bundle activation over the rest of the HPS thus accounting for the large change in the HPS waveform due to altered right bundle branch conduction. As stated earlier, the maintained level of atrial activity would rule out any change in the recording system gain or possible shift in lead axis. On the other hand, the waveforms making up the HPS potential probably represent resultant vectors of HPS activation and with the application of filter frequencies of 80–300 Hz, alteration of conduction may not correspond to changes expected with conventional ECG signals filtered between 0.1 and 200 Hz. Further studies using different lead axes and filter settings under similar experimental conditions will be required.

Early in our studies we were forced to consider that another possible source of the HPS waveform is atrial activity. Changing the sequence of A-V conduction would negate this possibility. The ability to use the QRS as a fiducial point, i.e., use of the QRS detection and pre-trigger averaging, allowed the assessment of A-V junctional rhythms. Figure 8D was obtained during a junctional rhythm in which atrial activation was buried in the QRS yet the HPS waveform persisted. The exact morphology of the HPS waveform is different from the control. Sherf and James have postulated that during junctional rhythms the HPS may be eccentrically activated (severe cases of which cause aberration) and this may account for the slightly different appearance of the early and middle portions of the HPS waveform. Note that during the nodal rhythm the late portions of the HPS waveforms are quite similar to the control. Since the QRS during nodal rhythms was not aberrant in this case, some normalization of the HPS activation may have occurred via lateral interconnections in the proximal His-Purkinje system. In the case of the junctional rhythm in figure 8A and 8B a similar change in the early portion of the HPS waveform is seen but the late portion is contaminated by large atrial activity thus precluding an accurate interpretation of this aspect of the recording.

Amplifier gain is usually set so that electrode catheter recordings will only detect potentials generated by nearby tissues. Thus the His bundle electrogram is a relatively local recording dependent upon the position of the electrode catheter and electronic conditioning of the signal. When the electrodes are in apposition to the His bundle, activity of the distal bundle branches is not recorded at the gains employed. In contrast, the HPS waveform is recorded by “distant” electrodes which are not significantly closer to the His bundle than to more distal portions of the His-Purkinje system. Therefore there is no particular selection by proximity of certain components of the His-Purkinje system.

With electrodes placed at a distance from the His-Purkinje system on the boundaries of the volume conductor, recordings should have information from the entire His-Purkinje system. The relative size and shape of the HPS waveform is dependent upon several factors. Filtering is necessary for attenuating low frequency atrial activity such as atrial repolarization, but alters the configuration of the HPS waveform much the way a differentiator would. Since the bundle of His is smaller in size than either bundle branch, activity in the early portion of the HPS waveform would be expected to be smaller. As the larger bundle branches and peripheral Purkinje fibers are activated, appropriate variation in HPS waveform amplitude can be expected. With the activation of the even larger ventricular mass the recordings become of such great relative magnitude that saturation of the digitizing devices occurs. Lead placement is important for maximizing activity from different structures, but since the His bundle and its bundle branches and ramifications are not at right angles to each other, no lead will entirely show a His spike without evidence of the bundle branches and peripheral Purkinje network activation. These reasons indicate that no facile waveform relationship can be made between the His bundle electrogram and averaged leads. The HBE does provide a timing indicator when observing the average leads, but relative magnitudes and comparisons of peaks and onsets is naive. A large deflection from an electrode catheter does not necessarily correspond to a large deflection measured with a distant electrode system.

Using the anterior-posterior lead axis both on the surface of the body and from the epicardium of the canine heart very similar waveforms (3-4 waves of consistent amplitude) have been recorded during activation of the His-Purkinje system. The SAL and the EAL have been shown to be consistent means of
detecting His-Purkinje activation in normal states. The use of the EAL in abnormal states, i.e., conduction disorders, has further demonstrated the usefulness and sensitivity of the signal averaging technique. Thus the derived HPS waveform can be segmentally divided into early, middle and late portions. This segmental analysis fits well with the anatomic segmentation of the HPS into the His bundle, bundle branches and Purkinje network but must be used with caution. Even though lesions occurring in the His bundle and bundle branches selectively alter the early and middle portions of the HPS waveform, more work must be done before adopting this interpretation. The similarity between the EAL recordings and SAL recordings suggests that future work involving the SAL can utilize the insights obtained with the invasive method. It remains to be determined if the signal averaged leads will be more sensitive than the ECG in diagnosing small but significant conduction disorders in the HPS. Also we hope to determine whether this new method of recording will provide more information about conduction than intracardiac leads in conjunction with the ECG, i.e., His bundle-electrocardiography.

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Extracardiac recordings of His-Purkinje activity during conduction disorders and junctional rhythms.
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Circulation. 1975;51:802-810
doi: 10.1161/01.CIR.51.5.802
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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