COMPARATIVE STUDY OF FIVE PREOPERATIVE METHODS FOR THE LOCALIZATION OF ACCESSORY PATHWAYS IN THE WOLFF-PARKINSON-WHITE SYNDROME

SHIWEN YUAN, M.D.*,# TAKASHI IWA, M.D.*, TAKURO MISAKI, M.D.*
KEIICHI MUKAI, M.D.*, YASUHIRO MATSUNAGA, M.D.*, HIROYUKI BANDO, M.D.*
KENICHI NAKAJIMA, M.D.*,* AND TETSUO NUMA, M.D.***

One hundred and thirty-four cases of Wolff-Parkinson-White syndrome were studied to evaluate the relative usefulness of electrocardiography (ECG), electrophysiological studies (EPS), body surface mapping (BSM), gated blood-pool phase analysis (nuclear studies), and vectrocardiography (VCG) in the localization of the accessory pathway (ACP). In comparison with the final localization verified by intraoperative studies, 93.4% in 8-region ACP localization (97.7% in 4-region ACP localization) could be correctly localized by ECG using our criteria, 83.9% (86.8%) by EPS, 82.6% (95.8%) by BSM, 78.8% (87.7%) by nuclear studies, and 67.3% (78.0%) by VCG. It was concluded that: (a) ACP can be localized preoperatively with considerable accuracy by using our simple ECG criteria. (b) The EPS method has some limitation, especially with respect to 8-region ACP localization. (c) Our observation showed no evidence that BSM, VCG, or nuclear studies were superior to ECG in ACP localization. (d) Among the 5 methods studied, ECG and EPS appear to be the appropriate procedures for preoperative ACP identification.

KNOWLEDGE of the location of the accessory pathway (ACP) in patients with the Wolff-Parkinson-White (WPW) syndrome is of great importance in guiding the surgical intervention, in selecting patients likely to benefit from catheter ablation, and in obtaining clues to the existence of multiple ACPs. Accordingly, many methods have been used for preoperative ACP localization, such as electrocardiography (ECG), electrophysiological studies (EPS), body surface mapping (BSM), gated blood-pool

phase analysis (nuclear studies), and vectorcardiography (VCG). The present study was designed to evaluate the clinical usefulness of these 5 preoperative methods of ACP localization.

PATIENTS AND METHODS

Of 151 patients with WPW syndrome who underwent surgical intervention at Kanazawa University Hospital between December 1982 and February 1986, 134 met the following criteria for entry into this study: (a) a delta wave in the resting 12-lead ECG; (b) the presence of ACP (s) responsible for the delta wave and for reciprocating tachycardia (RT)

Key words:

WPW syndrome
Accessory pathway
Localization of accessory pathway
ECG in WPW syndrome

(Received June 20, 1990; accepted December 14, 1991)

*The Department of Surgery (I) ; **Department of Nuclear Medicine, and ***Department of Health and Physical Education, Kanazawa University, Japan

#Present address: The Department of Cardiology, Suzhou Medical College Suzhou, China

Mailing address: Takashi Iwa, M.D., Department of Surgery (I), Kanazawa University School of Medicine, 13-1 Takaramachi, Kanazawa 920, Japan

TABLE I ACP LOCALIZATION BY DELTA WAVE POLARITY

<i>Right cardiac type</i>	VI ($\pm$)
Anterior	III, aVF (+)
Lateral	III, aVF (Variate in between)
Posterior	III, aVF (-)
<i>Right septal type</i>	VI (-)
Anteroseptal	III, aVF (+)
Posteroseptal	III, aVF (-)
<i>Left cardiac type</i>	VI (+)
Anterior	III, aVF (+)
Lateral	I ($\pm$) or (-), aVL (-)
Posterior	III, aVF (-)
Posteroseptal	III, aVF (-)

and/or rapid ventricular response during atrial fibrillation as verified by EPS and intraoperative studies; (c) loss of the delta wave, no evidence of conduction via the ACP, and disappearance of tachyarrhythmia attacks after surgical division of the ACP. There were 99 male and 35 female subjects with a mean age of 41.5 years (range 0.3–74). Twenty-one patients had associated cardiac disorders: 15 had Ebstein's anomaly, 3 had rheumatic heart disease, and 3 had coronary heart disease.

Sixteen cases of intermittent WPW syndrome were included, with only the data showing antegrade conduction over the ACP being used. For patients with double manifest ACPs were included to evaluate the different sensitivities of these 5 methods in such cases. Each of their ACPs was counted as 0.5 of a case in the statistical analysis. Concealed type WPW syndrome was excluded, since this study aimed to assess the diagnostic sensitivity of these methods for manifest ACPs (only EPS is capable of detecting a concealed ACP). The technically incomplete data were also excluded. All the patients underwent the patients underwent the examinations of ECG and EPS, but only the later cases were studied by BSM, nuclear studies, and VCG. Altogether 130 ECG, 121 EPS, 72 BSM, 73 nuclear studies, and 75 VCG recordings were used in this study.

ACP localization by ECG. The resting 12-lead ECG was recorded in sinus rhythm at 25 mm/s and a gain of 10 mm/mV. If multi-

ple ECG recordings were available, the one demonstrating the most preexcitation was chosen for analysis. If two or more ECG patterns of preexcitation were found in a single patient, then all of them were analyzed to detect multiple ACPs. All the subjects underwent multiple channel simultaneous ECG recordings; 48 had 6 channel and 84 had 3 channel recording. The onset of the delta wave was defined as the time when the initial slurring was observed in one of the simultaneously recorded leads. ACP localization was mainly based on the polarity of the delta wave in leads VI, III, aVF, I, and aVL, as reported by Iwa et al (Table I).¹ The delta wave was taken as positive (+) when the initial slurring had a positive deflection, negative (-) when the initial slurring had a negative deflection; and isoelectric ($\pm$) when there was no slurring or when an initial upward or downward deflection was followed by a reversed deflection to the baseline during the first 40 msec of the QRS complex. In lead VI, however, when the initial r wave followed a downward slurring and QRS main deflection, even if the initial r wave was up to 50 msec wide, the delta wave was also judged as ($\pm$).

ACP localization by BSM. Body surface isopotential maps were constructed using a HPM-6500 BSM system with 87 mapping electrodes placed on the chest surface. ACP localization was performed using criteria determined on the basis of our previous study.² In brief, localization was mainly based on the body surface locations of the potential minimum and the spatial relationships between the minimum and maximum on the maps of the delta wave (40 msec after the onset of QRS complex).

ACP localization by nuclear studies. The nuclear studies included planar phase analysis and single photon emission computed tomography (ECT).^{3,4} For planar phase analysis, three projections (left anterior, right anterior, and left lateral oblique) were used, and functional maps of the sequence of ventricular wall motion were generated. The phase images were evaluated using a color-coded isocount display, and the initial phase was taken to correspond to the location of the ACP. For ECT, gated transaxial images were reconstructed after data acquisition. Then short-axis slices were obtained, and the

Fig.1. Diagram of a cross-section of the heart at the level of the annulus fibrosus showing the 8 and the 4 preselected anatomical regions for ACP localization. The numbers around the annulus indicate the mapping points used in this study and the number in parenthesis indicate the number of ACPs at each region in this series of 134 cases with 138 ACPs.

RAS: right anteroseptal; RAW: right anterior wall; RLW: right lateral wall; RPW: right posterior wall; RPS: right posteroseptal; LPW-LPS: left posterior wall and posteroseptal; LLW: left lateral wall; LAW: left anterior wall; RFW: right free wall; LFW: left free wall; PS: posteroseptal.

ACP location was determined by phase analysis of the ventricular basal slice. Length-based Fourier analysis of the ventricular basal slice was also performed for more precise ACP localization⁴. Subjects undergoing only planar phase analysis were excluded from this study.

ACP localization by VCG. VCG was performed using the standard Frank lead system. The initial QRS vector enlarged with a calibration of 0.125 mV/10 mm, and the P and T loops were excluded to facilitate the delta vector analysis. ACP localization by VCG was mainly based on the orientation of the vector 20 msec after the onset of the QRS as well as the maximal vector of the QRS. For a left-sided ACP, the characteristics of both the delta vector and the QRS maximal vector were obtained from the left sagittal plane, while for a right-sided ACP the features of the delta vector were obtained from the horizontal plane and those

of the QRS maximum from the frontal plane⁵.

ACP localization by EPS. Each patient underwent conventional EPS according to the previously published method^{1,6}. In brief, multi-electrode catheters were positioned at the high right atrium, the coronary sinus, the atrioventricular junction, and the right ventricular apex. Intracardiac electrograms were recorded together with 3 surface leads of the ECG. During ventricular pacing and/or reciprocating tachycardia, mapping using an electrode catheter placed in the coronary sinus was performed to locate a left-sided ACP, and multi-point mapping along the tricuspid annulus using an electrode catheter in the right atrium was performed to locate a right-sided ACP.

The final ACP localization was based on the following intraoperative studies⁷: (a) epicardial mapping using a 6-bipolar electrode catheter, (b) monitoring of ECG pattern

TABLE II CORRECTLY LOCALIZED CASES (%) IN 8-REGION ACP LOCALIZATION

Available data	ECG 130	EPS 121	BSM 72	Nuclear 73	VCG 75
Exactly correct	94.5 (72.7)	54 (44.6)	37 (51.4)	29.5 (29.5)	27.5 (6.7)
Relatively correct	26.5 (20.4)	47.5 (39.3)	22.5 (31.3)	28 (38.3)	23 (30.7)
Total	121 (93.1)	101.5 (83.9)	59.5 (82.6)	57.5 (78.8)	50.5 (67.3)

ECG vs. # & # $p < 0.05$, ## $p < 0.01$; VCG vs. # $p < 0.05$, #* $p < 0.01$
 The differences between all the other results was not significant

TABLE III CORRECTLY LOCALIZED CASES (%) IN 4-REGION ACP LOCALIZATION

Available data	ECG 130	EPS 121	BSM 72	Nuclear 73	VCG 75
Exactly correct	116.5 (89.6)	82 (67.8)	59 (81.9)	48 (65.8)	48.5 (64.7)
Relatively correct	10.5 (8.0)	23 (18.7)	10 (13.5)	16 (21.3)	10 (13.0)
Total	127 (97.7)	105 (86.8)	69 (95.8)	64 (87.7)	58.5 (78.0)

ECG vs. * $p < 0.005$, ** $p < 0.05$, *** $p < 0.001$; VCG vs * $p < 0.005$
 The differences between all the other results was not significant

normalization during division of a right-sided ACP with the heart beating, and (c) electrophysiological testing after interruption of the ACP. According to the results of these intraoperative studies, the ACP locations were divided into both 8 regions and 4 regions. The 8 regions were as following: (a) right anterior, (b) right lateral, (c) right posterior, (d) right anteroseptal, (e) right posteroseptal, (f) left anterior, (g) left lateral, and (h) left posterior and posteroseptal. The 4 regions were the following: (a) left free wall, (b) right free wall, (c) posteroseptal, and (d) right anteroseptal (Fig. 1).

Each of the above mentioned 5 methods was used by one of the authors to complete 8-region and 4-region ACP localization blinded to the result of the other methods and the intraoperative studies. For 4-region localization, we used the same criteria and data as for 8-region localization, but only

distinguished the ACP location between the 4 regions. The diagnostic sensitivity of each method was evaluated by comparison with the final intraoperative localization using the following two criteria: (a) exactly correct, which meant that the preoperative localization was identical to that verified by intraoperative studies; and (b) relatively correct, which meant that the ACP was preoperatively localized at a neighboring region only one mapping point away (about 1 to 2 cm) from the final location. The diagnostic sensitivity was taken as the sum of exactly and relatively correct rate, and this was the main criterion by which the usefulness of these 5 methods was judge. For statistical analysis, the chi-square test was used.

RESULTS

The distribution of ACP localization in

these 134 cases (with 138 ACPs) is shown in Fig. 1, and the results of 8-region and 4-region ACP localization using these 5 preoperative methods are detailed in Tables II and III. In total, 93.1% of the ACPs could be correctly localized by ECG, 83.9% by EPS, 82.6% by BSM, 78.7% by nuclear studies, and 67.3% by VCG in 8-region localization. The corresponding results for 4-region localization were 97.7% by ECG, 86.8% by EPS, 95.8% by BSM, 87.7% by nuclear studies, and 78.0% by VCG.

The difference of the diagnostic sensitivity between 8-region and 4-region localization by BSM was significant (82.6% vs 95.8% $p < 0.05$), while differences for the other 4 methods were not statistically significant. The difference of the diagnostic sensitivity between ECG and each of the other 4 methods was significant for both 8-region and 4-region localization, except for 4-region localization by BSM. The differences between EPS or BSM and VCG in 8-region localization and the difference between BSM and VCG in 4-region localization were also significant, while the differences between all the other methods were not statistically significant (Tables II and III).

Among the patients with double ACPs, 8 of the 8 ACPs were correctly localized by ECG, 6 by EPS and BSM, 3 by nuclear studies, and 3 by VCG in both 8 and 4-region ACP localization. All of the 4 patients manifested two alternating types of preexcitation pattern. Both of the ACPs were detected by ECG in these 4 cases, while EPS and BSM only did so in 3 cases, nuclear studies did so in 2 cases, and VCG was completely unable to do so.

The manifested preexcitation were by ECG in all 16 cases of intermittent WPW syndrome. In comparison, this was achieved by EPS in 12 cases, by BSM in 7 cases, by nuclear studies in 4 cases, and by VCG in 2 cases. Of the 10 cases incorrectly localized by ECG, 7 were correctly localized by EPS, 3 by BSM and nuclear studies, and none by VCG.

There was no evidence that either the location of the ACP or the associated cardiac influenced the diagnostic sensitivities of these 5 methods.

DISCUSSION

ACP Localization by ECG

Several sets of criteria for locating the ACP using the 12-lead ECG have been reported in recent years, of which Gallagher's criteria⁶ are the best known. However, as has been pointed out by Milstein⁸ and Lindsay⁹, Gallagher's criteria are difficult to use in practice, because the ECG features of his 10 different ACP locations overlap to a considerable degree. In addition, these criteria require maximal preexcitation (a QRS greater than 140 msec) which usually is not present without atrial pacing.

Milstein et al established an algorithmic method, and reported that observers blinded to the results of mapping could correctly identify about 90% of ACPs. This algorithm took the polarity of the delta wave, the QRS pattern, and the QRS axis into consideration to achieve 4-region ACP localization. Lindsay et al proposed criteria for 5-region ACP localization based on the frontal QRS axis, the leads in which the negative delta wave appears, and the point of R wave transition ($R/S > 1$) in the precordial leads. They reported that 60 of 66 ACPs could be identified. Reddy et al¹⁰ and a committee of the World Health Organization (WHO)¹¹ have also published their criteria formed as a composite of the previous criteria. Lemery et al¹² tested the criteria of Gallagher and the WHO in their series of 47 operated cases. The results was unfavorable, as only 32% of their cases showed an ACP location in agreement with that determined by these two sets of criteria. It is thus obvious that all the above-mentioned criteria need to be further verified by a larger surgically proved series.

Our ECG criteria for ACP localization are based on approximately 400 surgically treated WPW cases. They are simple to follow, as only 5 of the 12 lead resting ECG leads are used (Table I), and show good agreement between the predicted ACP location and that determined by intraoperative studies. We found that 93.1%, in the 8 regions and 97.7% in the 4 regions of the ACPs could be correctly localized in this study, while our previous study showed that even inexperienced doctors or medical students could correctly localize about 90% of the ACPs by using these criteria⁷. Our results

are in contrast to the negative conclusions reached by Lemery et al.² Although they have limitations, the results of the present study show that our ECG criteria provide a simple and practical method for preoperative ACP evaluation.

ECG can also be of benefit in revealing intermittent ACPs and detecting multiple ACPs, since it could be easily repeated to record intermittent manifestation and alternate preexcitation patterns. The manifested preexcitation patterns were recorded by ECG in all 16 cases of intermittent WPW syndrome in this series, but not by the other 4 methods. In the 4 cases of double ACP manifesting two types of preexcitation pattern, ECG correctly localized both of the double ACPs by the recording of antegrade conduction via the different ACPs. Many other limitations of ACP localization by ECG have been reported by previous investigators.⁷⁻⁹ Our experience using Iwa's criteria showed that to determine the polarity of the delta wave in cases showing minimal preexcitation was often difficult. Sometimes, a completely incorrect localization was produced by a misinterpretation of the delta wave in only one lead.

Evaluation of The Other Preoperative Methods

One of the main purposes of EPS in WPW patients is to detect the ACP location. Endocardial mapping within the coronary sinus and along the tricuspid annulus during ventricular pacing or induced RT may give accurate ACP localization. EPS also has the advantage of detecting concealed ACPs and ACPs in cases with minimal preexcitation by demonstrating retrograde and/or antegrade conduction via the ACP. Also, intermittent preexcitation may become persistent during atrial pacing. The present study showed that EPS correctly localized 7 of the 10 ACPs which were incorrectly localized by ECG, which indicates that its use has advantages over ECG and the other methods we tested in some cases, even though it was inferior to ECG in total sensitivity.

There are also limitations with regard to ACP localization by EPS. In cases with a right-sided ACP, mapping along the tricuspid annulus is difficult because the catheter cannot be properly placed. Gallagher et al.³

developed a special catheter to facilitate appropriate placement in the right heart, but it is not yet widely used. We used an ordinary USCI electrode catheter for mapping in the right atrium, and this may be partly responsible for the lower diagnostic sensitivity of EPS in our series, especially in cases with right-sided ACPs (77.0% for right-sided cases vs 83.9% for the whole EPS group in 8-region localization). Inability to pass the catheter into the coronary sinus in left-sided cases is another problem with EPS. There were 9 such cases among the 79 patients with left-sided ACP in this series, and in 5 of these the ACP was incorrectly localized although we used a catheter in the main pulmonary artery instead. This could be the second reason for the relatively lower sensitivity of EPS. Concealed WPW syndrome was excluded from this study, and this may have also influenced the diagnostic sensitivity of EPS, for only EPS can detect concealed ACP.

The sensitivity of BSM was lower than ECG (82.6% vs 93.1%, $p < 0.05$) in 8-region localization, while that was no statistical difference from ECG (95.8% vs 97.7%, $p > 0.4$) in 4-region localization. Moreover, the difference of the sensitivity between 8-region and 4-region localization by BSM was statistically significant (82.6% vs 95.8%, $p < 0.05$). This indicates that though BSM has its limitations in the fine localization of the ACP (8-region), its sensitivity is higher in 4-region ACP localization. The same trend of improvement in sensitivity of the other 4 methods could be seen by comparing the results of 8-region and 4-region localization, though the differences were not statistically significant. Despite the many favorable reports on the use of BSM in WPW syndrome, our findings suggest that BSM is no better than ECG. Moreover, the complicated manipulations of the procedure itself also limit its clinical application in ACP localization.

The main nuclear method used in the present study was ECT. It has been reported to be of high sensitivity in ACP localization.^{3,4} In the present study, the sensitivity of nuclear studies in 8-region and 4-region ACP localization was only 78.8% and 87.7%, lower than that of ECG, EPS and BSM. This was considered to be mainly due to the limitations of the method itself. Low spatial

resolution can make it difficult to pinpoint 8-region ACP locations and frequent echo and/or premature beats during data acquisition can lead to the fusion of difficult patterns. Inability to detect very low amplitudes of wall motion and minimal preexcitation made it difficult or even impossible to localize the ACP in such cases. Also, both septal preexcitation and normal excitation may have their earliest phase in the septal region and are then difficult to distinguish by nuclear studies^{3,4}

There have only a few reports on ACP localization using VCG^{14,15} and these showed that only a rough localization limited to left, right, and septal regions of the heart could be achieved. An attempt has also been made to formulate criteria to locate the ACP in 8 different regions by VCG⁵. Though the results showed that the orientations of both the delta and QRS maximal vectors vary regularly with different locations of the ACP, the overlap between neighboring regions limited its clinical application and reduced its sensitivity. In cases with the delta wave overlapping the latter part of the P wave and in cases with minimal preexcitation, the localization is even more difficult. In the present study, ACP localization using our reported VCG criteria⁵ showed the lowest sensitivity both in 8-region and 4-region localization. Furthermore, the procedure and its clinical application are complicated than the use of conventional ECG. For these reason, VCG is no longer used for ACP localization in our hospital.

REFERENCES

1. IWA T, KAWASUJI M, MISAKI T, et al: Localization and interruption of accessory conduction pathway in Wolff-Parkinson-White syndrome. *J Thorac Cardiovasc Surg* **80**: 271, 1980
2. IWA T, MAGARA T: Correlation between localization of accessory conduction pathway and body surface maps in Wolff-Parkinson-White syndrome. *Jpn Circ J* **45**: 1192, 1981
3. NAKAJIMA K, BUNKO H, TADA A, et al: Nuclear tomographic phase analysis: Localization of accessory conduction pathway in patients with Wolff-Parkinson-White syndrome. *Am Heart J* **109**: 809, 1985
4. NAKAJIMA K, MISAKI T: Localization of accessory conduction pathway by gated emission computed tomography and Fourier analysis. In *Cardiac Arrhythmias: Current topics*, ed by IWA T, WATANABE Y, MISAKI T, Excerpta Medica, Tokyo, 1987, p505
5. NUMA T, IKEDA T, KITaura T, et al: Location of accessory pathways and vectrocardiograms. *Ann Sci Kanazawa Univ* **23**, 37, 1986
6. GALLAGHER JJ, PRITCHETT ELC, SEALY WC, et al: The preexcitation syndromes. *Prog Cardiovasc Dis* **20**: 285, 1978
7. IWA T, MUKAI T, et al: Surgical management of the Wolff-Parkinson-White syndrome. In *Cardiac Arrhythmias: Recent progress in Investigation and Management*, ed by IWA T, FONTAINE G, Elsevier, Amsterdam, 1988, p239
8. MILSTEIN S, SHARMA AD, GUIRAUDON GM, et al: An algorithm for the electrocardiographic localization of accessory pathways in the Wolff-Parkinson-White syndrome. *PACE* **10**: 555, 1987
9. LINDSAY BD, CROSSEN KJ, CAIN ME: Concordance of distinguishing electrocardiographic features during sinus rhythm with the location of accessory pathway in the Wolff-Parkinson-White syndrome. *Am J Cardiol* **59**: 1093, 1987
10. REDDY GV, SCHAMOROTH L: The localization of bypass tracts in the Wolff-Parkinson-White syndrom the surface electrogram. *Am Heart J* **113**: 984, 1987
11. WILLEMS JL, ROBLES DE MEDINA EO, BERNARD R, et al: Criteria for intraventricular conduction disturbances and preexcitation. *J Am Coll Cardiol* **5**: 1261, 1985
12. LEMERY R, HAMMILL SC, WOOD DL, et al: Value of the resting 12 lead electrocardiogram and vectrocardiogram for locating the accessory pathway in patients with the Wolff-Parkinson-White syndrome. *Br Heart J* **58**: 324, 1987
13. GALLAGHER JJ, PRITCHETT ECL, BENDITT DG, et al: New catheter techniques for analysis of the sequence of retrograde atrial activation in man. *Eur J Cardiol* **6**: 1, 1977
14. TONKIN AM, WAGNER GS, GALLAGHER JJ, et al: Initial forces of ventricular depolarization in Wolff-parkinson-White syndrome. analysis based upon localization of accessory pathway by epicardial mapping. *Circulation* **52**: 1030, 1975
15. TALWAR KK, BLOMSTROM P, EDVARDSSON N, et al: Spatial vectrocardiography in the Wolff-Parkinson-White syndrome: Correlation with epicardial mapping findings. *PACE* **7**: 979, 1984