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Usefulness of Body Surface Potential Mapping for Clinical Diagnosis and Research

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In recent years body surface potential mapping (BSPM) with large numbers of electrodes has been shown not only to provide an excellent research tool but also to be clinically useful (Liebman 1990, Liebman *et al.* 1981, 1984, 1987, 1988, 1989, 1991, 1992a,b, 1993, Widman *et al.* 1988). The BSPM group at Case Western Reserve University has developed a 180 electrode system which does not require electrode paste, with the electrodes encased in various sized vests (Kavuru *et al.* 1987, Thomas and Lee 1987, Lee *et al.* 1986). The maps which are coded in pseudocolour (Huebner *et al.* 1986) have been shown to be very easy to interpret and appear to have very high resolution. In past years much has been identified utilizing the BSPM's evidence of epicardial right ventricular breakthrough (Liebman 1990). More recently the BSPM has been shown to be extremely useful in understanding and analyzing initial ventricular activation. These two areas will be briefly discussed in this paper.

It is known that normal right ventricular activation occurs from endocardium to epicardium, after which the activation wave "breaks through" the epicardium. That this can be recognized on the BSPM was first demonstrated by Taccardi, the many years of data being summarized in a review by Taccardi *et al.* (1976). Spach *et al.* (1977) experimentally demonstrated it in epicardial studies in the chimpanzee and correlated it with findings on the BSPM while Miller and Geselowitz (1978) created a computer simulation model. In order for epicardial right ventricular breakthrough (RVBT) to occur there must be normal activation through the AV node, bundle of His, right bundle branch through the moderator band, and then activation from endocardium to epicardium (Myerburg *et al.* 1972). Taccardi has also shown that not all of the activation has to be from endocardium to epicardium in order for RVBT to be recognized in the BSPM, but evidently a large percentage must be present. The more and the closer together are the electrodes (Toyama and Tabato 1981), the more likely is epicardial RVBT going to be non-invasively evident. Furthermore, we have shown that when the same excellent data is displayed in the standard manner (in black and white) and separately in pseudocolour, RVBT will more likely be evident in the pseudocolour display.

In advanced right bundle branch block (RBBB) (Liebman *et al.* 1984), epicardial RVBT is not evident for it has been shown that activation occurs first in the left ventricle where epicardial left ventricular breakthrough is recognized, after which there is a conduction across the septum to activate the right ventricle. This lack of epicardial RVBT has allowed the clinical diagnosis of advanced RBBB instead of severe right ventricular hypertrophy (RVH) as well as partial RBBB instead of RVH with terminal right conduction delay (Liebman *et al.* 1987). As concerns the latter, Taccardi urges some caution since there could be some patients where partial RBBB is present, but there is still enough normal conduction from the endocardium to epicardium to recognize epicardial RVBT.

There are many other aspects of congenital heart disease where recognition of epicardial RVBT has been revealing. For example, in the three types of hypoplastic right ventricle (pulmonary atresia, tricuspid atresia, and tricuspid atresia plus transposition of the great arteries), the embryology appears to be distinctly different. In the first type, where the ventricle, though small, is developmentally similar to normal, with inflow, body and outflow areas, RVBT is late as in RVH with standard size ventricles. In the second two types, (tricuspid atresia, with and without transposition), the ventricles are less well developed, yet RVBT is also present, but occurs early. In patients who have had successful surgery for coarctation of the aorta, partial RBBB is often diagnosed on standard electrocardiography (ECG) many years later. However, in a study by Liebman *et al.* (1993) the BSPM demonstrated no cases of partial RBBB, but in 19 of the 24 patients RVH was definitely present with clear evidence for RVBT. The vectorcardiogram (VCG) suggested RVH in 11 of 19 and the standard ECG in only 6 of 19 patients. It is postulated that the persistent RVH is related to hyperplasia in late foetal and early

neonatal life. Hypertrophy based on a larger number of cells may apparently remain electrocardiographically evident for many years and perhaps throughout life.

Another area where epicardial RVBT has been of interest is the Wolff-Parkinson-White (WPW) syndrome (Liebman *et al.* 1991, Carlson and Liebman 1992), since activation of the ventricles is the result of fusion from both the normal conduction system and *via* the accessory connection. In a series of cases where the position of ventricular insertion was surgically proven as well as in others where an electrophysiological study had been done, the presence and the time of RVBT was utilized to estimate the amount of preexcitation. If the accessory connection is into the right ventricle, slow conduction in that ventricle occurs with no evidence for RVBT. If the accessory connection is into the left ventricle, RVBT always occurs, presumably from activation of much of the right ventricle *via* the normal conduction system. In addition, in some cases of right ventricular as well as left ventricular posteroseptal connections, there was RVBT as well. Assuming that the average normal time for RVBT is 30 ms in an adult, then if RVBT occurred at 60 ms, we postulate that preexcitation reaches the ventricle approximately 30 ms before it reaches it *via* normal conduction.

As concerns initial QRS activation and its great use in BSPM, obviously the WPW patients provide excellent examples. Most students attempting to predict accurately the position of ventricular insertion of the accessory connection utilizing standard ECG analyze the delta wave at 40 ms. In the above series (Liebman *et al.* 1991) as well as in a more recently presented series utilizing radiofrequency ablation (Carlson and Liebman 1992), the BSPM was more accurate than was the electrophysiological study in predicting the position. Furthermore, the Giraudon grid was used with its 17 distinct locations (Giraudon *et al.* 1986). Importantly the average time, when the position of the accessory connection was recognized, was 16 ms (most commonly between 15 and 18 ms). However, earlier and later times occurred as well, so that no method identifying one time to look at is appropriate.

An initial QRS directed to the left in patients with congenital aortic stenosis is known to be associated with increased severity, based upon a large series previously reported (Ankeney *et al.* 1983). In an effort to be able to postulate the mechanism for this finding, a BSPM study was performed, studying 19 patients with severe congenital aortic stenosis. In normals, at the onset of ventricular activation, there is a maximum over the sternum or slightly to the left – and usually superior. The minimum is more variable but is usually left anterior or posterior and inferior to the maximum. In the patients with severe aortic stenosis, the initial maximum was well to the left, near the anterior axillary line, with two BSPM's posterior to that line. The minima were more variable but always to the right of the peak positive potentials, as well as being more anterior. Although electrocardiographic theory is not well enough known in this regard, it is reasonable to hypothesize that potentials from the very hypertrophied free posterior wall in congenital valvular aortic stenosis are contributing to the observation.

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