CLINICAL SIGNIFICANCE OF BIFID T WAVES

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An attempt was made to analyze bifid T waves which appeared in different clinical conditions. Bifid T waves occurred in 16% of 600 normal children, 92% of 37 cases of childhood ventricular septal defect (VSD), 6 of 10 cases of tetralogy of Fallot (children) and 33% of 193 patients with cerebrovascular accidents (including 3 children). Sixteen cases of bifid T waves which appeared after amiodarone treatment were also analyzed. It was thought that in normal children, bifid T waves might be due to right ventricular preponderance. In VSD, the bifid T waves assumed a peculiar “dome and dart” appearance. In cerebrovascular accidents, autonomic imbalance might be at fault. In cases of treatment with adriamycin, myocardial toxicity is the most probable cause. The conclusion was made that bifid T waves can appear in different clinical settings, which must be considered individually.

The physiological counterpart of T waves in the surface electrocardiogram is ventricular repolarization. T waves may be upright, inverted, diphasic or flat, but bifid T waves have seldom been mentioned. In a previous paper, we reported ECG changes in 50 tumour patients treated with adriamycin, a cardiotoxic drug. Aside from nonspecific S-T segment changes, bifid, cloven, dimple T and spinous T waves were also found. These T waves changes were notably similar to those encountered in alcoholism and hypomagnesemia. We have also noted the appearance of bifid T waves together with prolongation of Q-T interval after amiodarone in the treatment of various arrhythmias. An attempt is therefore made to probe into the various clinical situations in which bifid T waves are likely to appear.

MATERIALS AND METHODS

The ECGs of six hundred “normal children”, aged 2–12 with no apparent heart diseases or conditions affecting the heart, 37 cases of children with ventricular septal defect (VSD), 10 cases of childhood tetralogy of Fallot, 193 cases of cerebrovascular accidents (all adults except for 3 children) and 16 patients with bifid T waves after taking amiodarone were analysed. Among the 600 normal children, 105 (16%) presented bifid T waves, mostly in V2–4 of the precordial leads. Eighty-five subjects showed a peculiar “dome and dart” form of T wave (Fig. 1-1 and Fig. 2-1). The 4–9 age group appeared to show this pattern of the T wave more often (58 cases); while the 6- and 7-year groups exhibited 13 each. The “dome and dart” T in normal children often presented a taller “dome”. Aside from the “dome and dart” T waves, bifid T waves in normal children at times showed a taller second peak, while in a slightly smaller percentage, the 2 peaks were equal. In 5 cases, bifid T waves appeared in V1 only; in 2, they appeared in leads V3–5. In another 3 cases, bifid T waves appeared inconsistently in serial ECGs. In 37 cases of VSD, 34 presented bifid T waves (92%) in leads V1 and V3. In 28 cases (75%) T waves assumed a “dome and dart” pattern, with the “dome” lower than the “dart”

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TABLE 1  INCIDENCE OF BIFID T WAVE IN VARIOUS CLINICAL STATES

<table>
<thead>
<tr>
<th>Clinical setting</th>
<th>Bifid T wave (incidence)</th>
<th>No. of “dome and dart”</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal children</td>
<td>16%</td>
<td>85</td>
<td>600</td>
</tr>
<tr>
<td>Childhood VSD</td>
<td>92%</td>
<td>28</td>
<td>37</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>60%</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular accidents</td>
<td>33%</td>
<td>2</td>
<td>193</td>
</tr>
</tbody>
</table>

(Figs. 1-2 and 2-2). This particular form of “dome and dart” pattern appeared in 9 of the 14 clinically diagnosed VSDs; the remaining 5 showed “non-specific” bifidity only. Eight of the 9 cases of VSD proven by right heart catheterization presented “dome and dart” T; In another 14 cases of operated VSD, typical “dome and dart” appeared in all in whom the defect was larger than 10 mm in diameter (11 cases); in 3 cases in whom the diameter was less than 8 mm, no bifid T was seen. In 7 cases, the “dome and dart” T disappeared postoperatively (Fig. 3); two showed no change and in another 2, the typical “dome and dart” changed to a “dimple” pattern. Of the 10 cases of tetralogy of Fallot, 8 underwent complete correction and 2 were not operated. In 6 of the 8 operated cases, 5 presented non-specific bifidity in V3; and in 1, V5 showed bifid T. In 193 cases of cerebrovascular accidents, the diagnoses of subarachnoid hemorrhage and cerebral hemorrhage were definite in 45 cases each. The diagnosis of cerebral thrombosis was certain in 40 cases, and in another 2, cerebral embolism was definite. Sixty-three cases (33%) showed non-specific bifid T waves. Of the 45 cases of subarachnoid hemorrhage, 18 exhibited T wave bifidity (40%); two of the 18 cases showed a giant T wave with a dip, giving the impression of a huge “dome and dart” (Fig. 4, upper strip), while the majority showed only “dimple” T waves (Fig. 4 lower strip). Other ECG changes included prolongation of Q-T intervals; giant T waves (upright or inverted) and depressed S-T segments; sinus bradycardia or tachycardia; atrial premature beats; atrial fibrillation or flutter, first and second degree A-V block; right bundle branch block; left anterior hemiblock and left ventricular enlargement. However, they were irrelevant to the appearance of T wave bifidity. Cases with huge “dome and dart” T showed 100% mortality; bifidity reverted in 7–10 days in all surviving cases. The incidence of bifid T wave in various clinical conditions is listed as follows (Table 1).

Note: The 16 patients presenting T wave bifidity under amiodarone treatment were selected cases whose incidence was not included.

DISCUSSION
Bifid T waves are commonly seen in the

Fig.1. (from AWA S, Am Heart J 80: 619, 1970. With permission) Various patterns of bifid T waves. 1. in normal children. 2. “dome and dart” T wave in VSD in children. 3. pointed T wave usually seen in ASD. 4. atypical “dome and dart” in tetralogy of Fallot.
precordial V2–V4 leads of children, presumably due to asynchronous repolarization of the two ventricles consequent to right ventricular preponderance in childhood. Awa analyzed 249 cases of VSD, patent ductus arteriosus (PDA), atrial septal defect (ASD) and tetralogy of Fallot, with 520 normal children in the same age range as control. It was found that 85% of children

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with VSD and PDA presented bifid T waves, less in tetralogy of Fallot (Fig. 1) and lesser still in normal children. The bifid T waves in VSD assumed a peculiar “dome and dart” appearance in most cases, with the “dart” taller than the “dome”. The dip between the dome and dart often reached the isoelectric line. Awa speculated that the second peak represented repolarization of the upper part of the right ventricle. In addition, volume overload of the right ventricle could have placed the right ventricle nearer to the chest wall to make repolarization of the right ventricle more evident.

We reviewed the ECGs of 600 “normal” children, aged 2–12, with no clinical evidence of heart disease or conditions affecting the heart; 16% exhibited bifid T waves, limited to lead V3 in most of the cases. Bifid T waves appeared in leads V3 and V5 (V4 was skipped) in only 2 cases, while in another 5, they were noticed in V1 only. 14.1% of normal children presented “dome and dart” T waves. In 37 children with VSD, 34 presented bifid T waves. However, only 28 of the 34 showed the typical “dome and dart” appearance (Fig. 2, upper strip). In the 23 cases in which the diagnosis of VSD was confirmed either by right heart catheterization or operation, 19 presented “dome and dart” T waves in the precordial V2–4 leads.

As to drug-induced bifid T waves, three

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Fig. 4. Leads V1, V3 and V5. Upper strip: record from a 13-year-old child with subarachnoid hemorrhage, showing huge “dome and dart” T waves in V3. Lower strip: record from a 63-year-old female also with subarachnoid hemorrhage, showing non-specific bifid T waves in V3.

Fig. 5. ECG taken from a patient under amiodarone treatment, showing broad T waves with dimples, more evident in leads V3 and V5.
possibilities exist. Drugs affecting the central nervous system, such as the psychotropics and neuroleptics, can induce changes in myocardial repolarization probably through disturbances in metabolism, myocardial injury or autonomic imbalance. Secondly, drugs with amiodarone as the representative can directly affect myocardial repolarization. Lastly, cardiotoxic drugs like adriamycin probably exert a direct toxic effect through ionic or enzymic changes.

Matabo observed that 2 weeks after the administration of large doses of apripamines, chlorothiapines, ethylaminodol or phenothiazine, ECG first presented flat T waves, and bifid T waves then appeared in leads II and V2–4 shortly after in about 50% of the cases. As co-enzyme Q could revert the bifid T waves to normal8 it was thought that this category of drugs could affect the redox system of myocardial cell, inducing degenerative changes in the myofibrils, accumulation of glycogen and increase of myocardial catecholamines.

Alvarez also noted that bifid T waves could appear 24 hours after phenothiazines6 It was thought that prevention of intracellular potassium efflux by phenothiazine was the underlying mechanism at fault, affecting repolarization of the ventricles. At this stage, however, the above is purely speculative.

Amiodarone induced bifid T waves (Fig. 5) are clearly a reflection of disturbances of repolarization.

We have noted spinous, bifid and dimple T waves after adriamycin treatment of various kinds of tumors1 changes similar to those seen in alcoholic cardiomyopathy.2 Magnesium deficiency or the superoxide radical might be the cause.5

Millard and Abildskov noted the appearance of bifid T waves in subarachnoid hemorrhage, carotid artery obstruction, cerebral trauma and intracranial tumor.8 Yamour pointed out that there is a frontal-hypothalamic-tegmental tract; the tegumentum contains abundant autonomic nuclei. This tract is further connected to the cervical sympathetic stellate ganglion which, when stimulated, can cause Q-T prolongation and T wave alternans.9,10 In the present series of 193 cases of cerebrovascular accidents due to various causes, bifid T waves appeared in 33%, mostly in cases of subarachnoid hemorrhage. It is known that giant T waves and Q-T prolongation can appear in subarachnoid hemorrhage, so it is natural to conjecture that bifid T waves in cerebrovascular accidents might be due to stellate ganglion stimulation through the frontal-hypothalamic-tegmental tract. The appearance of bifid T waves after severe endurance exercises may also be due to the same autonomic imbalance.

As to the mechanism of bifid T waves, Kamiyama proved in dog experiments that T wave bifidity was due to asynchronous repolarization of the left and right ventricles; the first peak was produced by repolarization of the left ventricle, while the second peak was due to right ventricular repolarization. Left ventricular muscle strips (including a portion of right ventricular epicardial muscle strip and anterior interventricular groove) were used in his experiments, action potentials synchronous with ECGs. Delay of repolarization at the interventricular groove was thought to be the cause of the dip between the 2 peaks.11 Watanabe confirmed this hypothesis by experimentation.12

In conclusion, the result of our clinical analysis of the various conditions under which bifid T wave appears indicates that bifid T waves can appear in different clinical settings. Right ventricular preponderance in normal children, volume overload in VSD, or autonomic disturbance in cerebrovascular accidents could be the cause. In other circumstances, myocardial metabolitic, ionic or enzymic changes affecting ventricular repolarization might be at fault.

REFERENCES

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