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Case Report

One substrate, two tachycardias and multiple QRS morphologies

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1. Clinical scenario

A 31-year-old woman with structurally normal heart, presented with hemodynamically stable, recurrent palpitations for several years. The presenting electrocardiogram (ECG) and intracardiac electrograms (EGM) are shown in Figs. 1 and 2, respectively.

2. What is the differential diagnosis from the ECG, and how does the intracardiac tracing help to narrow it down?

ECG analysis: It is an irregularly irregular broad complex rhythm. On initial assessment, there are no discernible P waves preceding the QRS complexes, raising suspicion for atrial fibrillation. Upon closer inspection, inverted P waves (Fig. 1: red arrow), negative in lead aVF and positive in lead aVR, can be observed following most QRS complexes. The RP intervals are variable, and there is evidence of ventriculoatrial (VA) dissociation (Fig. 1: red asterisk). Notably, the QRS complexes exhibit continuous, subtle variations in morphology. The predominant pattern is consistent with left bundle branch block (LBBB), with a QRS axis that varies between 0 and -60°.

Based on the ECG:

- Atrial tachycardia can be ruled out as not all QRS complexes are preceded by P waves.
- Atrio-ventricular reentry tachycardia (AVRT) can also be excluded due to the presence of atrioventricular (AV) dissociation.
- The remaining differential diagnoses include:
  1. Ventricular tachycardia
  2. Junctional tachycardia with broad QRS (aberrancy or conduction via bystander infranodal accessory pathway (AP), such as a nodoventricular or nodofascicular fiber)
  3. Enhanced automaticity originating from a right atriofascicular pathway
  4. Non-sustained atrioventricular nodal reentrant tachycardia (AVNRT) with broad QRS (aberrancy or a bystander accessory pathway)

Electrophysiology study findings: The catheters were placed in lateral high right atrium (HRA), His region (His), coronary sinus (CS) and right ventricle (RV). Baseline rhythm was sinus with a positive HV interval of 38 msec. As shown in Fig. 2, frequent, short runs of a wide-complex tachycardia with varying QRS morphologies, consistent with the clinical tachycardia, were observed (Note: The His-bundle signal in Fig. 2 is of poor quality, low-amplitude and far-field, therefore not easily discernible. His catheter position was adjusted to get better signals in subsequent tracings).

- The variable VA intervals seen on the ECG were confirmed, there was intermittent VA block.
- A negative HV interval during the tachycardia ruled out AVNRT.

3. What diagnostic manoeuvre can be performed at this stage?

- The transient and irregular nature of the tachycardia precluded diagnostic manoeuvres such as septal refractory premature atrial contractions or atrial overdrive pacing.

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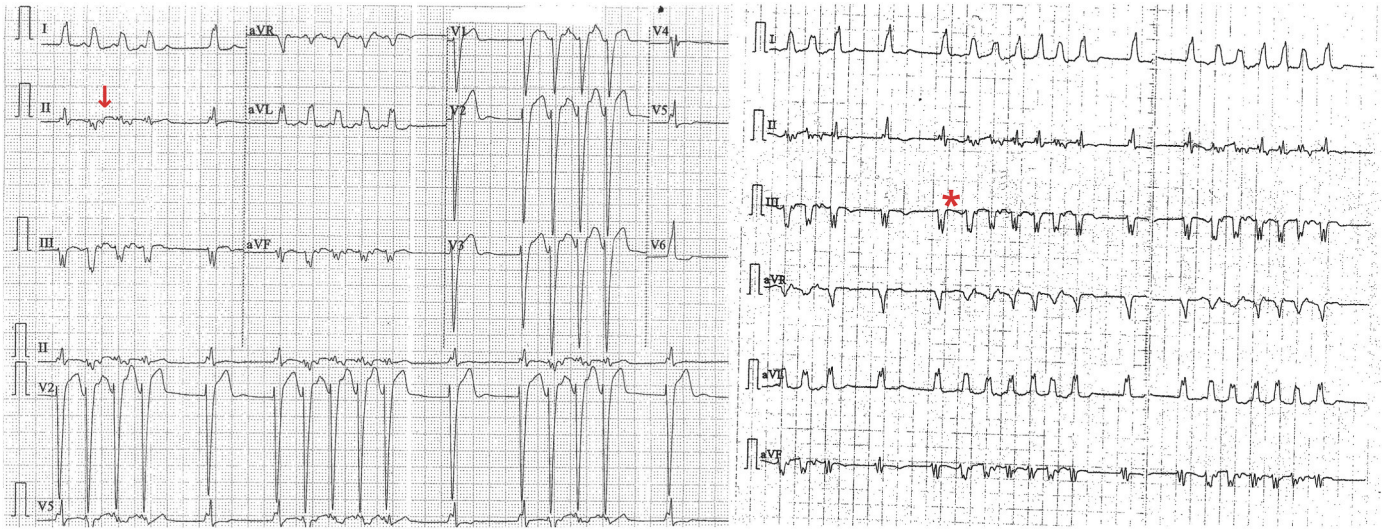

Fig. 1. The presenting ECG showed runs of wide complex tachycardia with inverted p-waves (red arrow) and intermittent atrioventricular dissociation (red asterix).

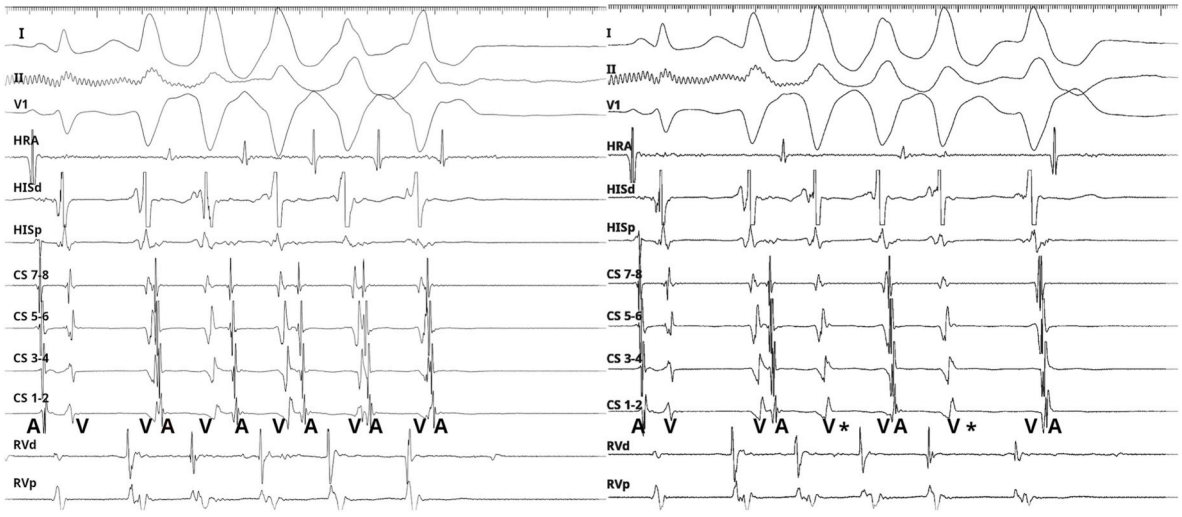

Fig. 2. During electrophysiology study, runs of wide complex tachycardia similar to the clinical ECG were observed. The local electrograms (EGMs) showed that VA intervals were not fixed, continuously changing, and there was intermittent VA block. HRA: High right atrium; HISd: His-bundle distal EGM; HISp: His-bundle proximal EGM; CS 7,8: proximal coronary sinus EGMs; CS 1,2: distal coronary sinus EGMs; RVd: Right ventricle distal EGM; RVp: Right ventricle proximal EGM.

- In Fig. 3, lateral right atrial pacing manoeuvre (during sinus rhythm) is shown. Right atrial pacing resulted in manifest pre-excitation. The pre-excited QRS complexes exhibited negative delta waves in lead V1 with a late transition by V5, and positive delta waves in leads II and aVF. The maximally pre-excited QRS morphology was similar to that of the clinical tachycardia.
- Decremental conduction property was elicited as well, characterized by a progressive increase in the stimulus-to-delta interval with atrial extra-stimulus (Fig. 3: with atrial extra-stimulus 400/290 msec, stim-to-delta prolongs).
- The negative HV interval and retrograde His activation (distal earlier than proximal) can also be appreciated during atrial pacing.
- The early right ventricular signal, nearly simultaneous with QRS onset, suggests the accessory pathway inserts near the RV catheter position (i.e., close to the RV apex).
- This manoeuvre thus confirms the presence of a right free-wall atrioventricular accessory pathway with decremental properties, i.e., atriofascicular accessory pathway.

Subsequently, following a ventricular extra-stimulus, the patient developed a sustained, regular, broad-complex tachycardia (Fig. 4).

4. What diagnostic manoeuvre should be performed during this sustained broad complex tachycardia?

- Fig. 4 shows a regular broad complex tachycardia with negative HV with QRS morphology very similar to the earlier ill-sustained irregular one.
- In contrast to the earlier arrhythmia, here regular RR intervals with 1:1 fixed VA association can be appreciated.
- Atrial overdrive pacing accelerated the tachycardia without changing the QRS morphology, thus ruling out ventricular tachycardia. The tachycardia continued post atrial pacing cessation.
- The diagnostic manouvre of choice here is delivering septal refractory premature atrial complex (PAC) during the tachycardia. If the PAC enters the tachycardia circuit and resets it, i.e., either advances or delays the next QRS as well as the subsequent atrial signal, it proves participation of the accessory pathway in an antidromic tachycardia circuit.

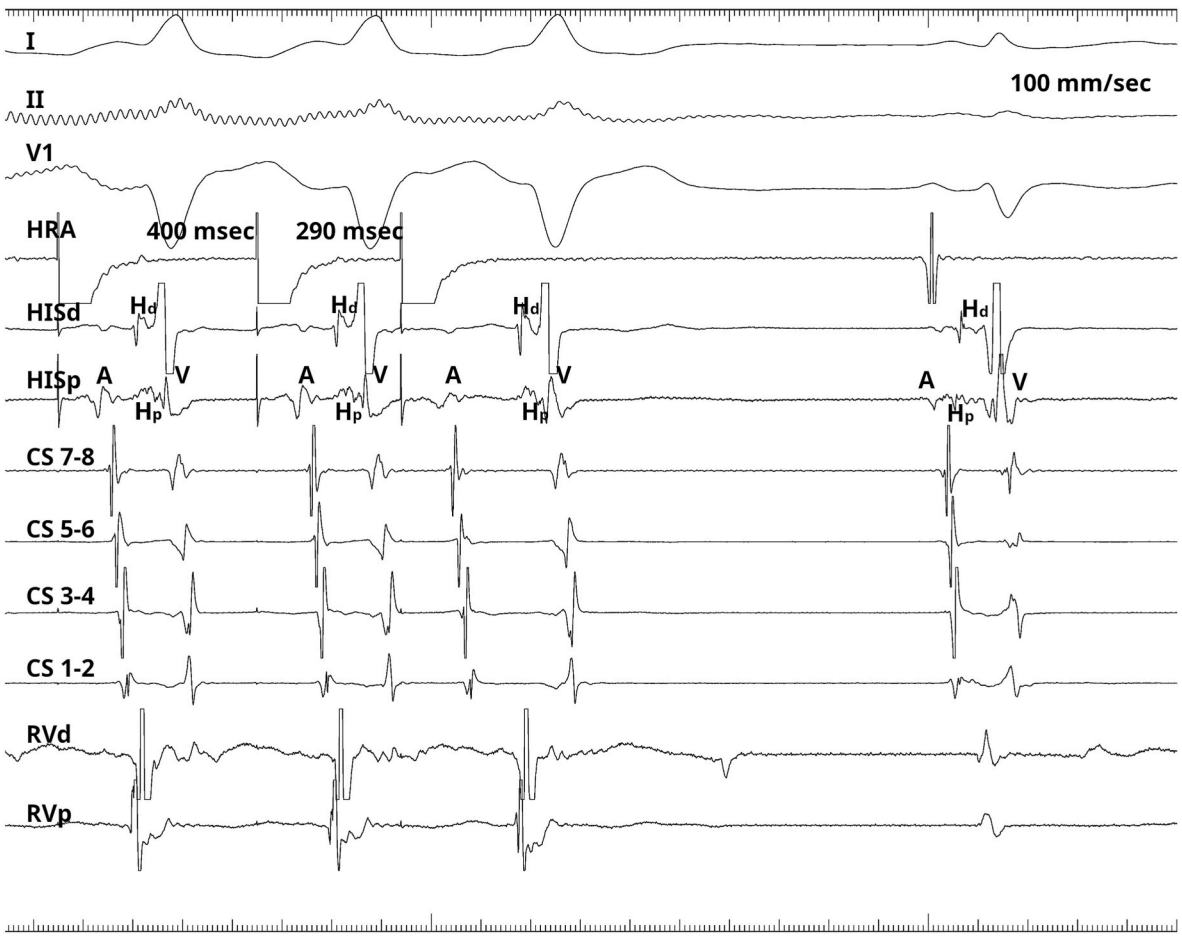

Fig. 3. When pacing from lateral high right atrium, pre-excitation with negative HV interval was demonstrated (S1 400 msec). With second atrial extra-stimulus (S2 290 msec), increment in stim-to-delta time was observed, suggestive of decremental pathway conduction. The HV interval during next sinus beat is positive. HRA: High right atrium; HISd: His-bundle distal EGM; HISp: His-bundle proximal EGM; CS 7,8: proximal coronary sinus EGMs; CS 1,2: distal coronary sinus EGMs; RVd: Right ventricle distal EGM; RVp: Right ventricle proximal EGM.

- These positive findings confirmed the presence and participation of a right-sided atriofascicular accessory pathway in the arrhythmia.

4.1. Final diagnosis and ablation

- Electrogram mapping of the right atrioventricular annulus identified discrete pathway potentials (M-potentials) at the 9 o'clock position of the tricuspid annulus.
- This patient had two different tachycardias, both involving the atriofascicular accessory pathway:
  - o A focal tachycardia due to enhanced automaticity from the pathway
  - o A macro-reentrant antidromic AVRT utilizing the pathway
- Radiofrequency ablation at this site resulted in the immediate termination of both tachycardias.

5. Why did the QRS morphology vary continuously during the focal automatic tachycardia?

Atriofascicular pathways are typically located at the posterior/lateral tricuspid annulus and insert directly into the right bundle. During automatic tachycardia or during AVRT, ventricular depolarisation occurs via retrograde conduction through the right bundle, which travels inside the moderator band towards the septum, subsequently to the left ventricle and/or to the atrium through the His bundle and AV node.

The observed continuous variation in QRS morphology is therefore attributable to dynamic changes in the ventricular portion of this circuit (Fig. 5).

6. Perturbations in the right-sided circuit

Possible mechanisms of perturbation in the right ventricular part of the circuit are:

- **Retrograde Right Bundle Branch Block (RBBB):** Proximal or distal retrograde RBBB can prolong the VH interval during antidromic tachycardia, potentially explaining subtle QRS changes with a superior axis shift.
- **Different sites of automaticity in the atriofascicular pathway:** This is unlikely, as ablation at the annular end of the pathway typically abolishes automaticity completely.
- **Myocardial Insertion and intramyocardial delays:** This mechanism has been refuted. A VH interval during antidromic AVRT, that is shorter than the HV interval in sinus rhythm is incompatible with a myocardial insertion site, proving direct insertion into the right bundle branch [1]. Furthermore, earlier activation of the right ventricular free wall compared to the septum, along with a completely different QRS morphology during right ventricular septal pacing versus tachycardia, suggests the pathway inserts into the distal moderator band or papillary muscle [1].
- **Multiple Insertion sites:** The hypothesis of multiple atriofascicular accessory pathway insertion sites is possible but lacks histological confirmation.

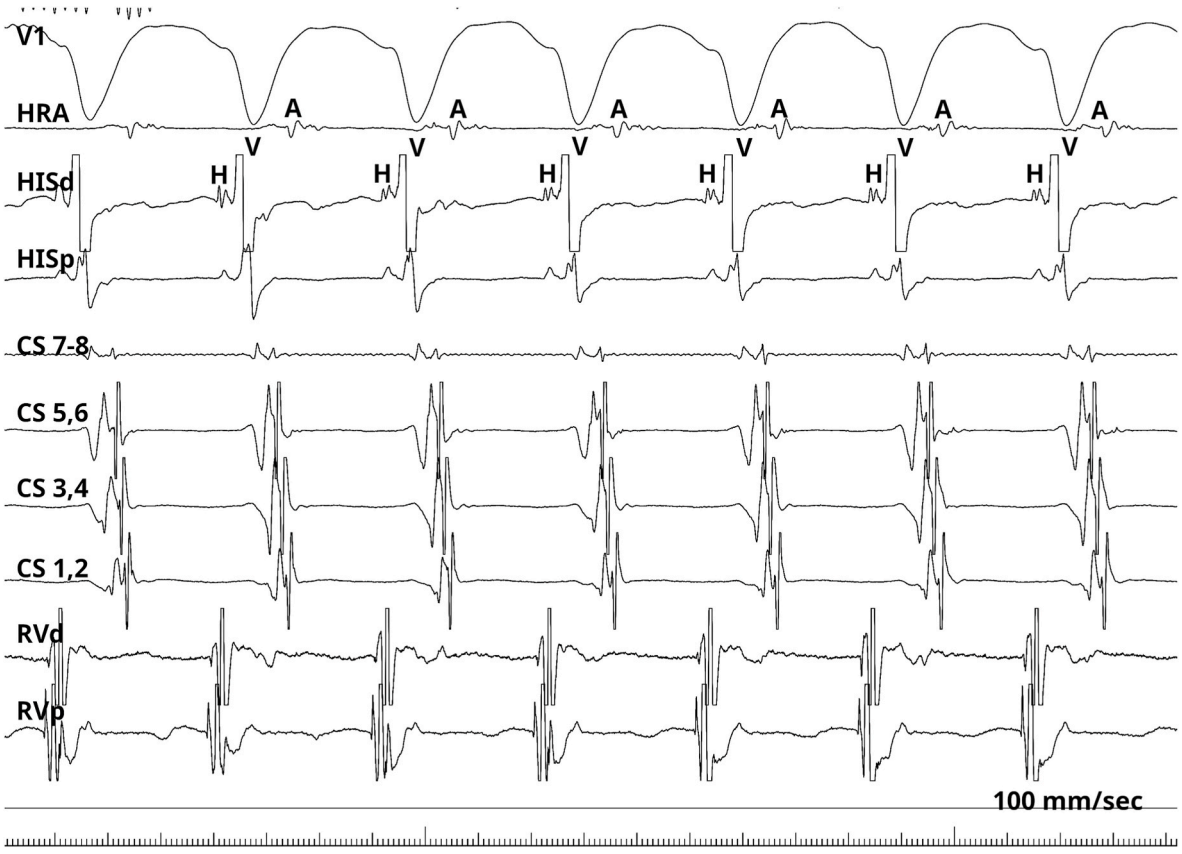

Fig. 4. A tachycardia with stable left bundle branch block QRS morphology with negative HV interval was induced with ventricular extra-stimulus. This was proven to be antidromic AVRT with atrial overdrive pacing. HRA: High right atrium; HISd: His-bundle distal EGM; HISp: His-bundle proximal EGM; CS 7,8: proximal coronary sinus EGMs; CS 1,2: distal coronary sinus EGMs; RVd: Right ventricle distal EGM; RVp: Right ventricle proximal EGM.

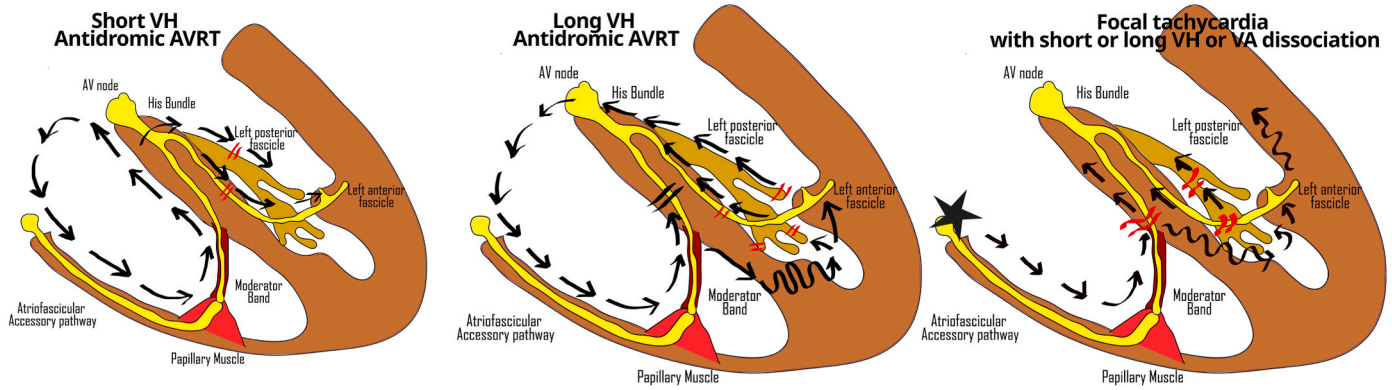

Fig. 5. Circuits of antidromic atriofascicular tachycardia with short VH AVRT, long VH AVRT and automatic atriofascicular tahcycardia (left to right). Activation pattern of left-sided fascicles and possible locations of conduction block (red) is demonstrated.

In summary, retrograde RBBB is a common phenomenon that leads to VH prolongation or dissociation, and subtle changes in QRS morphology but the multiple QRS morphologies and axis shifts observed in short-VH antidromic AVRT and automatic tachycardias cannot be fully explained by perturbations confined to the right-sided circuit.

7. Perturbations in the left-sided circuit

Perturbations within left ventricular circuit are illustrated in Fig. 5.

- In **short-VH** tachycardia (without retrograde RBBB), left ventricle is depolarised by anterograde conduction via the left anterior and posterior fascicles. Blocks within these fascicles could produce different QRS morphologies.
- In **long-VH** tachycardia (with retrograde RBBB), the left-sided conduction system is activated retrogradely, which can yield a multitude of QRS morphologies.

Therefore, the wide range of QRS morphologies observed during short-VH or long-VH, antidromic or automatic, atriofascicular tachycardia can be explained by the differing activation patterns of the left ventricle.

8. Question 6. Why do atriofascicular pathways show enhanced automaticity?

The atrio-annular portion of these fibres is known to contain cells with pacemaker-like properties, often referred to as an "accessory AV node." While clear anatomical or embryological evidence for this hypothesis is lacking, the electrophysiologic features—including decremental conduction, discrete potentials, and adenosine sensitivity—strongly support it [2]. In most of the reported cases, enhanced automaticity has been observed at the time of or immediately following catheter ablation. Spontaneous automaticity prior to ablation, sufficient to cause symptomatic arrhythmia, is rare. In a series of 40 patients, Sternick et al. reported spontaneous activity in 5 (12.5%) cases [3].

9. Conclusion

Atriofascicular accessory pathways are most commonly associated with antidromic AVRT but can also be a source of automatic tachycardia. This presentation can be easily confused with ventricular tachycardia and should be considered in the differential diagnosis of a broad-complex tachycardia. Furthermore, the varying QRS morphology observed in both short and long VH tachycardias is likely attributable to conduction blocks (anterograde or retrograde respectively) within the left fascicles.

10. Editorial commentary (Yash Lokhandwala)

The ECGs here are a classic example of trying to make sense of a bizarre rhythm by first establishing a central ‘foundation’ and then building around it. The QRS complex morphology in this case is the bedrock. The initial r and the rS in V1 are too sharp to be of ventricular origin. The QRS in the 12 leads then conforms to a typical LBBB pattern. But more QRS complexes than P waves would indicate a ventricular origin, in this case the right bundle branch. The slightly varying QRS morphologies in the limb leads have been lucidly explained.

Since Mahaim-like pathways are known to have automatic potential, now the ECG seems to fall in place. There is a continuously ongoing erratic automatic rhythm generated by the Mahaim-like pathway. Hence instead of trying different anti-arrhythmic medications, the decision to perform an electrophysiologic study is perfect. The repose to right atrial pacing is then nearly confirmatory, while the antidromic tachycardia is the icing on the cake.

Disclosures

None.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the author(s) used Deepseek v3.2 in order to check the grammar of the manuscript. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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