Successful Surgical Interruption of the Bundle of Kent in a Patient with Wolff-Parkinson-White Syndrome

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SUMMARY
Recurrent supraventricular tachycardia is a frequent complication in patients with the Wolff-Parkinson-White (WPW) syndrome. Our patient was unusual in that the arrhythmia was the predominant rhythm, and it was felt that the sustained tachycardia was responsible for signs and symptoms of congestive heart failure. The arrhythmia could not be controlled adequately with digitalis, quinidine, diphenylhydantoin, or propranolol. Atrial or ventricular pacing also failed to prevent recurrent episodes of tachycardia.

Physiological and pharmacological studies suggested that an anomalous pathway was responsible for the WPW abnormality and participated in a re-entrant circuit which sustained the episodes of tachycardia. Isopotential body surface mapping suggested anomalous ventricular excitation at the lateral aspect of the right atrioventricular groove. Epicardial mapping at the time of surgery was used to localize the earliest area of anomalous ventricular activation, and surgical transection of the atrioventricular junction at that point abolished the electrocardiographic features of WPW and the recurrent tachycardia. Five months after surgery neither the ECG features of WPW nor the tachycardia has recurred. The signs and symptoms of congestive heart failure have subsided, and the patient has returned to work.

Additional Indexing Words:
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Re-entrant pathway
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Congestive heart failure
Epicardial map

The Wolff-Parkinson-White (WPW) syndrome is a clinical disorder observed most commonly in patients with otherwise normal hearts.\(^1\) The electrocardiogram is characterized by an abnormally short P-R interval and a prolonged QRS complex with typical distortion of the initial portion.\(^1-3\) Many patients with the WPW syndrome are subject to frequent episodes of supraventricular tachycardia. Previous anatomic studies have suggested a definite, although variable, structural basis for the electrocardiographic abnormalities,\(^4-11\) and several investigators have suggested that these structural abnormalities also provide a basis for re-entrant tachycardia.\(^12-17\) This report describes a patient with type B WPW syndrome who had intractable episodes of tachycardia. Physiological and pharmacological studies suggested that re-entry was the basis of this arrhythmia. The approximate location of the anomalous pathway was determined from isopotential body surface maps and localized precisely by epicardial mapping at surgery. The region

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of the abnormal pathway was divided with subsequent abolition of both the electrocardiographic features of WPW syndrome and the episodes of tachycardia.

**Report of Case**

W.S. is a 32-year-old white fisherman who was referred to Duke Hospital for evaluation and treatment of a cardiac arrhythmia. Frequent episodes of rapid heart action were first noted when the patient was 4 years old. These episodes appeared to start spontaneously on most occasions, although exercise and emotional stimuli increased the frequency of attacks and rest seemed to decrease them. The episodes of tachycardia were relatively infrequent and well tolerated until 1965, when symptoms of congestive heart failure developed. An electrocardiogram showed recurrent episodes of supraventricular tachycardia, and chest x-rays showed cardiomegaly and pulmonary congestion. The patient was given digitalis and diuretics with subsequent improvement of the symptoms of heart failure; however, the episodes of tachycardia persisted, and he was unable to return to work. Six weeks prior to his admission to Duke Hospital, symptoms of heart failure recurred. Cardiomegaly and signs of congestion were noted, and an electrocardiogram revealed that the predominant rhythm was a supraventricular tachycardia. Sinus rhythm could be restored by carotid sinus massage. Digitalis and diuretics were given with improvement of the symptoms of heart failure; however, the recurrent episodes of tachycardia persisted.

In February 1968 the patient was evaluated in the Cardiology Clinic at Duke Hospital. Physical examination revealed a blood pressure of 110/90 mm Hg. There were frequent abrupt

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**Figure 1**

*Base-line rhythms. The 12-lead tracing on the left shows the Wolff-Parkinson-White anomalies. The tracing on the right was taken during one of the episodes of supraventricular tachycardia (paper speed, 25 mm/sec). The drawings at the bottom were made from vectorcardiograms taken during a period of anomalous conduction. F = frontal plane; H = horizontal plane; S = sagittal plane.*

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changes of both the radial and apical pulse rates from 70 to 150 beats/min. Examination of the chest revealed moderate cardiomegaly, although the lungs were clear to percussion and auscultation. Examination of the heart revealed an atrial gallop. No murmurs were heard and there was no evidence of peripheral edema. A chest x-ray showed moderate cardiomegaly and pulmonary congestion. The electrocardiogram showed sinus rhythm with a P-R interval of 0.12 sec, and QRS duration of 0.12 sec with a delta wave. Sinus rhythm was interrupted by frequent episodes of supraventricular tachycardia, during which the P-R interval was 0.20 sec and the duration of the QRS complex was 0.08 sec. The patient was placed on a maintenance dose of digoxin, and he was started on quinidine sulfate, 300 mg four times daily. This therapy was continued for 2 weeks with no decrease in the frequency of episodes of tachycardia, and he was subsequently hospitalized on the Cardiac Care Unit.

**Base-line Observations**

Continuous electrocardiographic monitoring demonstrated that the predominant rhythm was a supraventricular tachycardia. The tachycardia was present approximately 70% of the time and lasted for periods of minutes to several hours at a rate of 150 to 180 beats/min. When sinus rhythm was present, there was always anomalous ventricular activation. Exercise and excitement appeared to initiate episodes of tachycardia, although spontaneous onset of the tachycardia also was observed.

The two basic electrocardiographic patterns observed in this patient are illustrated in figure 1. In the tracings on the left, the abnormalities of the type B WPW syndrome are seen, including a short P-R interval, delta wave, and prolonged QRS complex. The vectorcardiogram shown below demonstrates that the initial vector was directed to the left in the frontal plane and anteriorly in the horizontal and sagittal planes. Characteristic initial slowing also was seen in all planes. The tracings on the right were obtained during an episode of supraventricular tachycardia. The QRS complex duration during the tachycardia was normal, and the P

**Figure 2**

Initiation and termination of the supraventricular tachycardia. Panels 1, 2, and 3 show spontaneous initiation of the tachycardia. Panels 4 and 5 show the response to Valsalva maneuvers.
waves appeared to be inverted in leads III and aVF. During the tachycardia the R-R intervals were slightly irregular. High speed tracings revealed that this irregularity was due to changes of the P-R interval on alternate beats, while the R-P interval remained unchanged.

**Onset and Termination of the Arrhythmia**

As illustrated in figure 2, premature ventricular beats (panels 1 and 4) and nodal or atrial premature beats (panels 2 and 3) initiated episodes of tachycardia. Retrograde P waves frequently followed the premature beats. Sinus beats with anomalous conduction were never seen to initiate episodes of tachycardia. Carotid sinus massage or a vigorous Valsalva maneuver (panels 4 and 5) were often effective in terminating the arrhythmia. There was gradual slowing of the tachycardia prior to its termination, accompanied by gradual lengthening of the P-R interval. The R-P interval did not change. The P-R interval of anomalous beats and the R-P interval during the tachycardia were essentially the same in duration. In panel 4, an escape beat is followed by a premature ventricular contraction which appears to re-initiate the tachycardia.

**Response to Pharmacological Agents**

The initial therapeutic approach was an attempt to determine whether the recurrent episodes of tachycardia were secondary to congestive heart failure. On a daily regimen of digoxin, low sodium diet, and furosemide (Lasix), minimal symptomatic improvement occurred. There were no changes in the frequency or duration of the tachycardia. Quinidine sulfate, 1.8 g daily in divided doses, was then given to maintain a blood level of 4.0 to 5.0 mg/L for several days. Again, no change in the frequency or duration of the tachycardia was noted. Diphénylhydantoin injected intravenously in 50-mg aliquots at 3 to 5-min intervals did not alter the attacks of tachycardia. Propranolol, 40 mg four times daily by mouth, slowed the rate of the tachycardia to 110 to 120 beats/min but did not reduce the frequency of attacks. The sinus rate also slowed, and long periods of asystole were noted when the rhythm reverted to a sinus mechanism.

**Response to Atrial and Ventricular Pacing**

Figure 3 illustrates the response to atrial and ventricular pacing. In panel A which is a rhythm strip during the tachycardia, the rate was regular at 150 beats/min, the QRS duration was 0.08 sec, and P and T waves were superimposed. Panel B illustrates the response to atrial stimulation. Atrial stimulation was initiated via a platinum-tipped electrode which had been positioned in the right atrium and attached to a Medtronic pulse generator. The large stimulus artifact (A) was produced by the unipolar mode of stimulation. Cardioversion occurred during stimulation of the atrium at 120 pulses/min, a rate slower than the tachycardia. The large arrow points to a T wave which does not have the usual superimposed P wave. The stimulus artifact preceding the large arrow distorted the last complex of the tachycardia and presumably resulted in atrial depolarization. Cardioversion appeared to be effected by the blocked atrial response. The next beat was a paced beat. This sequence was repeated on numerous occasions. Panel C illustrates that slow ventricular pacing also terminated the arrhythmia. Several randomly occurring stimuli can be seen prior to the point of conversion. Conversion occurred coincidently with the first propagated beat in response to the pacemaker. The pulse generator was then turned off. Two beats with inverted P waves and without anomalous conduction were followed by three beats with anomalous conduction. The tachycardia then recurred after either a premature atrial or nodal beat.

The atrium or ventricle was paced at rates varying from 75 to 120 beats/min in an attempt to prevent or reduce the duration of episodes of tachycardia. Although atrial and ventricular stimulation effectively terminated the tachycardia, pacing at the various rates did not eliminate the recurrent attacks. Rhythm strips during continuous pacing showed alternating periods of a paced atrial or ventricular rhythm, varying periods of tachycardia, and interruption of the tachycardia by pacing.

The rate of the tachycardia slowed from 165 (panel A) beats/min prior to propranolol to 132 beats/min (panel D) after intravenous administration of 10 mg of propranolol. This decrease in rate was due entirely to a lengthening of the P-R interval without a change of the R-P interval. The tachycardia terminated spontaneously in the middle of panel D. A sinus beat with aberrant conduction was followed by an atrial echo beat which propagated to the ventricles and then by sinus rhythm with aberrant conduction.

**Isopotential Body Surface Mapping**

An isopotential body surface map was recorded in an attempt to locate the anomalous pathway for atrioventricular (A-V) conduction. The procedure consisted of recording electrocardiograms from a total of 150 points over the entire thorax and upper abdomen. All data were recorded on magnetic tape and transcribed by an analog-to-digital converter at a sampling rate of 926 samples/sec. The digital tape was then analyzed by an IBM 360-75 computer, and the output was presented for each millisecond in a format which

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Figure 3

Termination of the supraventricular tachycardia by atrial and ventricular stimulation and by propranolol (Inderal). Panel A shows a rhythm strip of lead I. Panel B shows the response to atrial stimulation. The small arrows and A in panel B indicate the large artifact produced by unipolar stimulation with a hydrogen electrode. The large arrow in panel B points to the T wave of the last supraventricular tachycardial beat. Panel C shows the response to ventricular stimulation. The small arrows in panel C point to the small artifacts produced by bipolar stimulation. Panel D shows the response to intravenous propranolol, 10 mg. Simultaneous right atrial hydrogen electrode and lead I tracings are shown in panel D. Panels A, B, and D were recorded on photographic paper (1-sec time lines) and C on a standard electrocardiogram (paper speed, 25 mm/sec).

represented the thoracic distribution of the body surface voltage. A particular isopotential body surface distribution is observed in normal adults which correlates with wave fronts in the heart breaking through on the epicardial surface, specifically over the right ventricular free wall along the anterior descending coronary artery. This distribution consists of an anterior left precordial maximum (area of highest positive voltage recorded on the body surface) with two minimal areas of greatest negative voltage on the body surface located in the right upper anterior chest (fig. 4, left panel). Our patient's body surface
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map (fig. 4, right panel) for a comparable instant of time was distinctly abnormal with a discrete right anterolateral minimum extending into the anterior maximum. This suggested that wave fronts within the heart were breaking through on the lateral aspect of the right ventricle near the A-V groove. From the isopotential body surface distribution it appeared that the patient might have a surgically approachable anomalous pathway responsible for pre-excitation of a portion of the right ventricle.

Epicardial Mapping at Surgery

At the time of surgery, bipolar and unipolar records, referenced against standard limb lead II of the electrocardiogram, were obtained from 40 to 60 points on the epicardial surface of the right and left ventricles and the right atrium. Records were taken while the patient exhibited anomalous conduction in the electrocardiogram. The results revealed that the earliest area of ventricular excitation occurred in a localized area, approximately 1.5 cm in width, at the extreme lower border of the right ventricle along the A-V groove (fig. 5). The epicardial wave front then moved leftward from this earliest area at the A-V groove across the right ventricle and lastly, across the left ventricle.

Surgical Division of the Anomalous Pathway

The patient was then placed on cardiopulmonary bypass. An incision was made in the right atrium parallel and anterior to the crista terminalis, thus exposing the interior of the

Figure 4

A characteristic body surface potential distribution in a normal adult (left panel) and in the patient with WPW type B (right panel) in selected instants of time. The dotted line indicates the calculated average potential line, and this has been labeled “O.” The solid lines denote positive values, and the interrupted lines indicate negative values. The (+) and (−) denote the highest (maximum) and lowest (minimum) voltage areas, respectively. The values of maxima and minima are indicated beneath each picture. The potential distribution shown for the normal adult is characteristic of the pattern which occurs simultaneously with epicardial breakthrough of wave fronts in the right ventricular free wall along the anterior descending coronary artery. The pattern consists of a maximum over the precordium with one minimum located in the right shoulder and a second minimum in the central sternal region. The potential distribution shown for the patient with WPW type B represents a comparable instant of time to that shown for the normal patient. An abnormal discrete right lateral minimum, which coincided with the delta wave on the electrocardiogram, is shown extending into the anterior maximum.
The epicardial excitation sequence (top views) of the patient with WPW type B prior to cardiac surgery. The bar graph below illustrates the time intervals encompassed for the epicardial areas indicated by the isochronous time lines as related to the onset of P wave. The white circles on the cardiac silhouette represent the recording sites. The isochronous time lines represent the epicardial areas undergoing excitation over a specific interval of time (constructed by connecting the points which were excited at similar times). In the lateral view, the posterior area of the left ventricle is indicated with a question mark to indicate that epicardial excitation times were not recorded from these posterolateral areas of the left ventricle. The earliest time of epicardial excitation (epicardial breakthrough) occurred about 110 to 115 msec after the onset of the P wave. Breakthrough occurred in a small area (approximately 1 cm) on the right ventricular free wall at the atrioventricular groove.

Figure 6

The heart at completion of cardiac surgery. The right ventricle occupies most of the picture with the outflow tract of the right ventricle labeled PA. The black circle indicates the area of the right ventricle along the atrioventricular groove which prior to surgery had been the area of earliest epicardial excitation. The sutures indicate the site of the incision.

The wave and appearance of a normal P-R interval and QRS duration. Figure 6 shows the heart following closure of the incision. The black dot indicates the point of earliest excitation.

Reexploration of the heart with a bipolar electrode following surgery revealed that the earliest area of activation was in a normal location on the right ventricular free wall adjacent to the anterior descending coronary artery (fig. 7).19, 20 The wave front then spread to encompass both right and left ventricles in a normal manner. The last area of activation recorded was at the right ventricular free wall along the A-V groove. This was the region which prior to surgery had been the area of earliest excitation.

Figure 8 shows a rhythm strip of lead II the evening following surgery and a 12-lead tracing 18 days after surgery. The P-R interval was 0.18 sec, and there were no delta waves. The vectorcardiogram at the bottom of figure 8 demonstrates an absence of the initial slow vector force which had been present before surgery. The patient’s postoperative course was essentially uneventful. Digitalis was discontinued and continuous monitoring revealed no recurrence of the WPW abnormality or paroxysms of supraventricular tachycardia. The patient has returned to our outpatient clinic 3, 5, and 8 weeks postoperatively. On each of these occasions electrocardiograms demonstrated a normal P-R interval.
and QRS complex. No episodes of supraventricular tachycardia have occurred since surgery. A chest x-ray 6 weeks after operation showed a decrease of heart size and clearing of the lung fields. The patient reported increasing exercise tolerance, is now on a daily exercise program, and has returned to work.

Discussion

In 1915, Wilson described a patient with frequent attacks of rapid heart beat and an electrocardiogram which demonstrated a short P-R interval and a wide QRS complex. In 1921, Wedd described a similar patient, and in 1930, Wolff and associates reported a series of 11 patients who shared similar clinical and electrocardiographic findings to those of the patients described above. They were young, healthy patients, usually with no underlying heart disease. They were prone to paroxysms of recurrent tachycardia and had the combination of a prolonged QRS complex and an abnormally short P-R interval. Many reports have followed, establishing this combination and referring to it as the Wolff-Parkinson-White syndrome.

Several theories have been proposed in an attempt to explain the electrocardiographic pattern in patients with WPW. The concept that the abnormal QRS complex is the result of a fusion beat was proposed by Hunter and associates. They suggested that anomalous complexes represented a double rhythm
produced by two interfering pacemakers, one located in the atrium and the other in one of the bundle branches. Sodi-Pallares and his associates\textsuperscript{22, 23} produced complexes resembling WPW during cardiac catheterization. They hypothesized that there were hyperexcitable areas on the right side of the interventricular septum in patients with the WPW syndrome that responded to the mechanical contraction of the atrium or to weak atrial action potentials. Prinzmetal and his associates\textsuperscript{24} proposed the theory of accelerated conduction through a normal A-V transmission system. They observed that artificial stimuli would produce QRS complexes resembling WPW complexes once the His bundle had been cut. Langendorf and co-workers\textsuperscript{25} pointed out that the artificially induced QRS complexes were only superficially similar to WPW complexes, since there was usually an absence of the delta wave. Furthermore, they noted that an irritable ventricular focus would not account for the fact that the QRS complex during episodes of tachycardia was usually of normal form and duration. Holzman and Scherf,\textsuperscript{26} and Wolferth and Wood\textsuperscript{27} suggested that the short P-R interval and delta wave might result from early excitation of the ventricle by an impulse which passed via an accessory atrioventricular pathway.

In 1893, Kent\textsuperscript{28, 29} described muscular bridges connecting the right atrium and right ventricle in a variety of mammalian species. Later studies by Kent\textsuperscript{30-35} described a band of muscle fibers at the right lateral atrioventricular junction. He reported that when all other atrioventricular connections were divided, the muscular bridge between the atrium and ventricle could conduct impulses in both an antegrade and retrograde direction. In subsequent years there has been considerable disagreement as to whether the communication described by Kent exists in the adult human heart. Truex and associates\textsuperscript{8} were able to demonstrate muscular bridges between the atrium and ventricle in human hearts only up to the age of 6 months. James\textsuperscript{36} has described specialized atrial fibers which bypass the upper portion of the node, and Mahaim and Clerc\textsuperscript{37} demonstrated connections between the A-V bundle and the upper part of the ventricular septum. It is apparent from previous anatomic studies that there are a variety of possible pathways, adjacent to, or at a distance from, the A-V node which could serve as a pathway for rapid conduction from atrium to ventricle. The histological studies of Ohnell,\textsuperscript{4} and Wood and associates\textsuperscript{5} are particularly relevant since they demonstrated abnormal muscular bridges of the type described by Kent in two patients with the WPW syndrome. Lev\textsuperscript{38} recently reviewed the anatomic reports of several patients who had had WPW syndrome. A Kent bundle was found in all of these patients with one exception, and in that patient copious Mahaim fibers were present.

There is increasing evidence not only that an accessory pathway does exist which is responsible for the classical electrocardiographic abnormalities, but that this pathway also plays a central role in the initiation and support of episodes of paroxysmal tachycardia. The response to atrial pacing and induced premature beats has been used recently in an effort to understand the electrophysiology of the anomalous pathway. Lau and associates\textsuperscript{39} observed that the P-R interval in patients manifesting the WPW abnormality did not progressively lengthen as the atrium was paced at increasing rates. They concluded that the anomalous pathway was not subject to the normal physiological delay which characterizes the A-V node. Durrer and his colleagues\textsuperscript{17, 40} have suggested that episodes of tachycardia are supported by a re-entry pathway involving the atrium, antegrade conduction through the A-V node and His bundle to the ventricles, and then a return to the atrium by retrograde conduction over the bundle of Kent. This hypothesis was tested and borne out by the response to induced atrial or ventricular premature beats during supraventricular tachycardia.

Our patient demonstrated many of the features that have been described in patients with WPW syndrome. He is a young man
with no apparent underlying heart disease. The electrocardiographic pattern demonstrated the classical changes of WPW (that is, short P-R interval, abnormal QRS configuration [delta wave], and initial slow vector by vectorcardiogram). Several preoperative observations suggested that an anomalous pathway for A-V transmission was present and that this pathway was responsible for the WPW abnormality as well as the recurrent tachycardia. The electrocardiogram always showed a prolonged QRS complex and delta wave in the presence of sinus rhythm, although the QRS was of normal form and duration during episodes of tachycardia. Isopotential body surface mapping showed that during the inscription of the delta wave an abnormal minimum developed over the right anterior chest in a position which suggested early epicardial excitation in the region of the right A-V groove. During episodes of tachycardia, there was retrograde activation of the atria. The R-P interval during the tachycardia was equal to the P-R interval during sinus rhythm with anomalous conduction. Furthermore, during the tachycardia, variations of the R-R interval resulted from changes of the P-R interval while the R-P interval remained constant. These observations suggested to us that during the tachycardia the impulse was transmitted from atrium to ventricle over the normal A-V transmission system and then returned to the atrium over the anomalous pathway, thereby establishing a re-entry circuit.

The concept proposed above is supported by other observations in our patient. Propranolol slowed the tachycardia from 150 to 110-120/min by prolonging the P-R interval, although neither the R-P interval of the tachycardia nor the P-R interval during sinus beats was affected. The ability of a single, appropriately timed atrial or ventricular premature beat to terminate the tachycardia is also consistent with a re-entry mechanism.41 Finally, the definitive evidence was the dramatic elimination of the WPW anomaly and recurrent paroxysmal tachycardias by surgically interrupting a segment of muscle which connected the atrium and ventricle at the point where pre-excitation was demonstrated by epicardial mapping.

Recurrent episodes of tachycardia in our patient were not controlled by therapy for congestive heart failure or by combinations of digitalis, quinidine, and Dilantin. Propranolol slowed but did not eliminate the tachycardia and accentuated the period of asystole after the tachycardia. Atrial and ventricular stimulation, although effective in terminating the arrhythmia, did not prevent its recurrence. We concluded that the tachycardia was the principal cause of incapacitation and that its control was imperative. Evidence that a surgical approach might be successful has been provided by recent observations. Durrer and Roos42 described the pattern of epicardial excitation of the ventricle in a patient with WPW syndrome. The earliest point of excitation was found near the right lateral part of the anterior atrioventricular sulcus, 10 msec following the P wave. Burchell and associates43 found a similar pattern of early excitation in a patient with WPW syndrome who was undergoing repair of an atrial septal defect. They abolished the WPW phenomenon by injecting 1% procaine into the ventricular muscle at the area of pre-excitation. A 1-cm cut was made parallel to the atrioventricular ring in an attempt to interrupt the Kent bundle. At the end of the surgical procedure, the electrocardiogram showed a return of WPW complexes. Burchell and his colleagues proposed that a larger incision, perhaps separating the right atrium and ventricle, should successfully interrupt the anomalous pathway. This report provides additional evidence that in certain patients with WPW syndrome, an anomalous pathway can be localized by electrophysiological techniques. Surgical interruption of this pathway was accomplished in our patient, and his subsequent clinical course indicates the feasibility of this approach for the permanent control of refractory tachycardia in selected patients with type B WPW syndrome.
It should be emphasized again that available anatomic studies indicate considerable variation in the structural basis for preexcitation and tachycardia in patients with WPW syndrome. A surgical approach similar to that described above would not be feasible if the aberrant pathway had not been accessible at the right lateral border of the heart. The ability to perform detailed epicardial mapping studies at the time of surgery was indispensable to precise localization and interruption of the accessory bundle.

References


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