HISTORICAL NOTES

Wolff-Parkinson-White Syndrome: Long Follow-Up and an Anglo-American Historical Note

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A patient who developed palpitation in 1917 was later found to have the Wolff-Parkinson-White syndrome and survived to the age of 86, when he died of emphysema. Shortly before he first presented, a report of another patient had been published that can now retrospectively be recognized as containing the first tracings from a case of the Wolff-Parkinson-White syndrome; its coauthors were a distinguished American cardiologist, Alfred E. Cohn, who had worked with Sir Thomas Lewis, and his British research fellow, Francis R. Fraser.

The Wolff-Parkinson-White syndrome was first described as a clinical entity with the electrocardiographic features of a short PR interval and a wide QRS syndrome, with a tendency to paroxysmal tachycardia (1), after a comparison of tracings by Parkinson and White, when White was visiting London. Within a few years, it was appreciated that previously described cases (2) of paroxysmal tachycardia fell into the same category later described as preexcitation. The first case report, currently acknowledged as reflecting this entity is that of Wilson in 1915 (3), but even earlier, in 1914, Mines (4) had postulated atrioventricular reciprocating tachycardia involving the atrioventricular node and an accessory pathway. Over the years, the syndrome has been recognized as being essentially benign, although with the rare risk of sudden death when it is complicated by atrial fibrillation with rapid conduction of the impulse to the ventricles (5).

There are many individual reports of long-term follow-up, including several notable examples of up to 30 years in duration, recorded by White and Donovan (6). Of our first four patients with the Wolff-Parkinson-White syndrome who underwent electrophysiologic study (7), one was then 74 years old and had suffered from paroxysmal tachycardia for 54 years; in this report, his history is supplemented by the subsequent follow-up data, and some historical observations are made.

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Case Report

The patient had developed palpitation in 1917, when serving in the British Army from which he was then discharged, though the precise diagnosis at that time is uncertain (8). He remained well, apart from episodes of tachycardia, until 1957 when the Wolff-Parkinson-White syndrome was recognized. Response to prophylactic treatment with quinidine and digoxin was poor and only slightly better later, with practolol. At the age of 70, his dyspnea became worse and he was found to be emphysematous.

The electrophysiologic study (7) showed preexcitation with narrow QRS complexes during tachycardia. Tachycardia, whether induced or spontaneous, was promptly terminated with intravenous verapamil (9) and the arrhythmia was subsequently well controlled with oral verapamil, 240 mg a day, until shortly before the patient’s death at the age of 86. In the last 5 years of his life, his electrocardiogram also showed right bundle branch block, and he was finally very disabled by the effects of his emphysema. There was no autopsy.

Discussion

The patient. The patient believed that he had valve disease when discharged from the army, but his symptoms were typical of paroxysmal tachycardia (8) and the subsequent electrocardiograms confirmed the diagnosis. He had thus lived for 65 years after the first appearance of palpitation, and during most of the last 10 years of his life, while receiving verapamil, he was free from arrhythmia. While this is an exceptionally long period of follow-up, doubtless other such cases will be observed.
The earliest case? Two years ago, my attention was
drawn to an earlier report on paroxysmal tachycardia in
which, during sinus rhythm, the complexes showed PR
shortening, QRS widening and delta waves (Fig. 1) (10).
The first author of this report, Alfred E. Cohn, was well
known in the United States and Europe, but there was no
available record of his coauthor, Francis R. Fraser, and his
subsequent career.

The authors. Alfred E. Cohn (1879–1957) spent 2 years
in Europe after graduating from Columbia University, the
last few months at University College Hospital, London,
with Sir Thomas Lewis in 1909. Cohn, who brought the
first string galvanometer to the Western Hemisphere (11),
joined the staff of the Rockefeller Institute for Medical Re­
search in 1911; he and one of his research fellows, Fraser,
published at least three papers together, two being on dig­i­
tals. Several senior American cardiologists still have per­
sonal memories of Cohn, who subsequently had a distin­
guished career in clinical practice, but he made at least one
further practical contribution to electrocardiography in in­
roducing a “strap-on” electrode in 1920 to replace the
cumbersome baths in which the limbs were immersed (12).

The puzzlement about Cohn’s coauthor, Francis R. Fraser
(1885–1960) (Fig. 2), is explained by the fact that he was
a British graduate who spent 2 years in the United States
after graduating from the University of Edinburgh (13).
Returning to England in 1914 with the Harvard Medical
Unit, he joined the Royal Army Medical Corps, serving in
France and then at the Hampstead Hospital where Lewis
carried out his studies on the “effort syndrome” (8). After
the war, Fraser became assistant director of the University
Medical Unit at St. Bartholomew’s Hospital Medical Col­
lege and, in 1922, its director. Besides research on cardiac
dyspnea, his subsequent interests included the treatment of
pernicious anemia and diseases of the thyroid. In 1934, he
was appointed as the first director of the Department of
Medicine at the new Postgraduate Medical School of Lon­
don (now the Royal Postgraduate Medical School) at the
Hammersmith Hospital. He was knighted in 1944 and sub­
sequently played an important role in the postwar organi­

Figure 1. The first three complexes show sinus rhythm with a
short PR interval, slurring of the upstroke of the R wave, QRS
widening, ST depression and T wave flattening. The third such
complex is followed by a ventricular extrasystole which shows
concealed retrograde conduction into the atrioventricular node, so
that the ensuing sinus complex is conducted more slowly, initiating
paroxysmal tachycardia with narrow QRS complexes.

Figure 2. Sir Francis Fraser.
References