Lown-Ganong-Levine Syndrome

The Lown-Ganong-Levine (LGL) syndrome is one of the pre-excitation syndromes of which Wolff-Parkinson-White (WPW) syndrome is the best known. In WPW syndrome there is an accessory pathway for conduction, called the Bundle of Kent, that bypasses the atrioventricular (AV) node. No such pathway has been identified for LGL. Theories to explain the condition have suggested possible intranodal or paranodal fibres that bypass all, or part of, the AV node.[1]

Diagnostic criteria include PR interval of no more than 120 ms, normal QRS complex duration, and paroxysmal supraventricular tachycardia (PSVT) but not atrial fibrillation or flutter.

The condition was first described in 1952 before the advent of electrophysiological testing, and some people dispute its existence as an entity.[2] Where there is a short PR interval but no history of supraventricular tachycardia (SVT), it is probably just a variation of normal. Where arrhythmias have been investigated in people with the diagnostic criteria, another cause has often been found.

Sometimes the duration of conduction through the AV node is fast and this is called enhanced atrioventricular nodal conduction (EAVNC).

Although tachycardia, along with increased stroke volume, enables cardiac output to meet demands in exercise, a very fast tachycardia is inefficient and may cause compromise. The ventricles do not have adequate time to fill in diastole and this may reduce cardiac output. Tachycardia reduces the duration of both systole and diastole but it is diastole that is reduced more. Around 75% of the blood flow to the right ventricle and 100% of the blood flow to the left ventricle occur during diastole. Hence, there is less time to perfuse the myocardium at a time of increased metabolic need.

Epidemiology

Lown suggested that 17% of people with a PR interval of less than 120 ms would have the condition.[2]

Presentation

History

The history is of bouts of tachycardia that may present as rapid palpitations. It most often starts in early adulthood but can present in childhood. It tends to get less frequent with passing years. In the otherwise healthy person there is probably no other feature but, where the heart and circulation are already compromised, perhaps from coronary heart disease, this can produce angina pectoris, shortness of breath and heart failure. There may well be light-headedness and dizziness due to hypotension.

Examination

There is usually no abnormality to be found between attacks, although some people have a resting sinus tachycardia. During an attack the pulse rate may be 200 beats per minute or sometimes even higher.

Investigations

- A 12-lead ECG is required. The PR interval should be no more than 120 ms and with no delta wave. A normal QRS is essential for diagnosis and a delta wave suggests an accessory pathway and a diagnosis of WPW syndrome.
- If possible, try to encourage the patient to come in during an attack so that an ECG can be recorded during one.
- Check U&E, calcium and magnesium. Check TFTs.
A Holter monitor may be used to record the heart rate. Ask the patient to note the time that an attack starts and stops. The old machines had memory for only 24 hours of monitoring but modern machines can record for a full week, greatly increasing the chance of recording an episode.

Management

- Referral to a cardiologist is required to try to obtain a definite diagnosis.
- Electrophysiological studies may be performed and an attempt made to find a cause for the SVT.[3]
- There is no aberrant bundle to ablate as in WPW syndrome.
- Beta-blockers such as metoprolol or atenolol may be useful and slow AV conduction.
- Non-dihydropyridine calcium-channel blockers such as verapamil may slow AV conduction and can be used to treat an acute PSVT. Verapamil plus a beta-blocker may produce complete heart block and so they should not be used together.
- Digoxin can also decrease conduction in the AV node.
- If drugs fail to give control, it can be treated by pacemakers. [4] Dual AV sequential demand pacemakers are used where there is an enhanced AV node pathway.

Prognosis

The syndrome can produce ventricular fibrillation and sudden death.[5] However, it is normally far more benign and can usually be controlled by pharmacological means.

Historical

Bernard Lown was born in 1921, William Ganong was born 1924 and Samuel Albert Levine was born in 1891 and died in 1966. The occurrence of frequent paroxysms of tachycardia in patients with a short PR interval and normal QRS duration had been described by Clerc et al in 1938 but it was the Americans who achieved the immortality of an eponym. Bernard Lown was a founder of International Physicians for the Prevention of Nuclear War and, in 1985, he was awarded the Nobel Prize for Peace. He is Professor Emeritus of Cardiology at Harvard.

Further reading & references

- Lown-Ganong-Levine Syndrome; ECG Library


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