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 accessory fibres that bypass all, or part of, the AV node\cite{1}. In some cases there may be a congenitally malfunctioning AV node\cite{2}. Whatever the pathophysiology, conduction is passed from the atria to the bundle of His without the delay usually incurred at the AV node.

Diagnostic criteria include PR interval of no more than 120 ms, normal QRS complex duration, a normal or inverted P wave, 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\cite{3}. 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)\cite{1}.

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\cite{3}. It is however very rare, with prevalence estimated to be less than one in a million\cite{4}.

**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 less 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 when symptomatic.
- 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. A longer recording of a week increases the chances of recording an episode.

**Management**

- Referral to a cardiologist is required to try to obtain a definite diagnosis.
- Echocardiogram and electrophysiological studies may be performed in an attempt made to find a cause for the SVT and assess the risk of sudden cardiac death.
- 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. It has been used to control symptoms in pregnancy\cite{3}.
- Dual AV sequential demand pacemakers have been used for this condition where medication has failed to give control\cite{5}.
• People with LGL syndrome need special care and management during anaesthesia as they are at high risk of peri-operative arrhythmias which may be life-threatening\(^2\). Certain drugs and anaesthetic agents should be avoided, and specialist cardiology input is helpful.

**Prognosis**

The syndrome can produce ventricular fibrillation and sudden death\(^7\). However, this is unusual, and it is normally far more benign and can be controlled by pharmacological means.

**Historical**

LGL is named after three American cardiologists. Bernard Lown was born in 1921, William Ganong was born 1924 and died in 2007, 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, the group was awarded the Nobel Prize for Peace. He developed the direct current defibrillator.

**Further reading & references**

- Lown-Ganong-Levine Syndrome; ECG Library
- Lown–Ganong–Levine syndrome; ECG Course 115.0 | The EKG Guy, YouTube, January 2018

4. Lown–Ganong–Levine syndrome; Orphanet rare diseases

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