ECG CASE STUDY

Poor R-Wave Progression

Ross MacKenzie, MD

Poor R-wave progression is a common ECG pattern, which is often inconclusively interpreted by medical directors. Although this ECG pattern is commonly attributed to anterior myocardial infarction, it may also be caused by left bundle branch block, Wolff-Parkinson-White syndrome, right and left ventricular hypertrophy as well as by faulty ECG recording technique. Failure to make a definitive interpretation of this pattern may result in a delay or loss of business.

Address: Ross MacKenzie Consulting, 2261 Constance Drive, Oakville, Ontario, L6J 5L8, Canada; e-mail: rossmackenzieconsulting@msn.com.

Correspondent: Ross MacKenzie, MD, FRCP(C), FACC; Division of Cardiology; Toronto General Hospital.

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CASE SCENARIO

A senior underwriter in your company has asked for your help in interpreting the electrocardiogram (ECG) in the Figure. He is requesting a second opinion on a large case and the competition is intense.

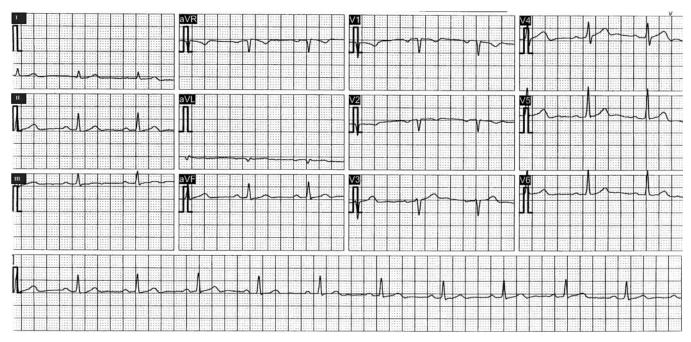
The electrocardiogram was done for age and amount by an industry paramedical company. The senior underwriter, who has taken an industry course on ECG interpretation, feels that the ECG is abnormal. A new medical director, who recently joined the medical department from clinical medicine, has also agreed that the ECG is abnormal. He has suggested repeating the ECG, as well as obtaining an old ECG and a stress myocardial perfusion study. The senior underwriter is under pressure to control underwriting expenses and is concerned about delaying the offer.

The applicant is a 44-year-old female nonsmoker. She is asymptomatic and there is no family history of heart disease. She is 5 feet 6 inches tall and weighs 156 pounds. Her examination and laboratory tests are normal.

What do you think of the ECG? Is it abnormal? If so, do the abnormalities have significant mortality implications? Can you make an underwriting decision now without additional requirements?

ECG INTERPRETATION

The prevailing rhythm is sinus in origin with an average ventricular rate of 64 per minute. The PR, QRS and QT intervals are normal. The QRS axis in the frontal plane is normal at approximately $+65^{\circ}$. P waves are negative in V_1 and V_2 and biphasic in V_3 . There is poor R-wave progression in leads V_1 – V_3



Applicant's electrocardiogram.

with a QS pattern in V_1 and V_2 and an embryonic r wave in V_3 of 1 mm in amplitude. The remainder of the ECG is unremarkable.

ECG ANALYSIS

Poor R-wave progression is an important ECG finding that may be associated with a number of cardiac conditions, which have mortality implications for the medical director. However, this ECG pattern can also occur in otherwise healthy individuals. The challenge for the medical director is to be able to recognize the significance of the pattern when it occurs in the context of an individual applying for insurance.

Before we begin our analysis, a few theoretical considerations are necessary to understand the genesis of the R-wave in the precordial leads. Ventricular depolarization normally begins in the middle-left of the interventricular septum, and travels in an anterior and left-to-right direction. This initial vector of electrical activity is seen by right-sided and midprecordial chest leads (V1–V3) as coming directly toward them resulting in a small r wave (referred to as a septal r wave). Poor R-wave progression may occur whenever these initial electrical forces are reduced in mag-

nitude or are directed posteriorly. Following activation of the septum, left ventricular depolarization dominates the remainder of the depolarization process. Although right ventricular depolarization is occurring simultaneously, right ventricular forces are dwarfed by the left ventricular events in the normal adult heart. The resulting vector will be seen by leads V_1 – V_3 to be moving directly away from them and a deep S wave is written on the ECG.¹

Following these theoretical considerations, let us now look at normal R-wave progression in the precordial leads. The normal chest lead ECG shows an rS-type complex in lead V_1 with a steady increase in the relative size of the R-wave toward the left chest and a decrease in the S wave amplitude. Leads V_5 and V_6 generally show a qR-type complex, with R-wave amplitude in V_5 often taller than V_6 because of the attenuating effect of the lungs. Normal variations include: narrow QS and rSr' patterns in V1 and qRs and R patterns in V_5 and V_6 .

At some point, generally around the V_3 or V_4 position, the QRS complex changes from predominately negative to predominately positive and the R/S ratio becomes >1. This

is known as the transition zone. In some normal individuals, the transition may be seen as early as V_2 . This is called early transition. At times, transition may be delayed until V_4 to V_5 . This is called delayed transition.²

The normal R-wave height in V_3 is usually greater than 2 mm. If the height of the r wave in leads V_1 to V_4 remains extremely small, we say there is "poor R-wave progression." In the literature, definitions of poor R-wave progression have been variable, using criteria such as R-wave less than 2–4 mm in leads V_3 or V_4 and/or the presence of reversed R-wave progression defined as R in $V_4 <$ R in V_3 or R in $V_3 <$ R in $V_4 <$ R in $V_5 <$ rank in $V_5 <$ R in $V_7 <$ or any combination of these.

Both the senior underwriter and the new medical director are concerned about the possibility of a previous anterior myocardial infarction. With myocardial necrosis due to myocardial infarction, a certain amount of myocardial tissue becomes electrically inert and unable to generate depolarizing forces. The remaining forces of ventricular depolarization now become accentuated (since they are no longer opposed) and the resulting vector of depolarization is reoriented in the direction away from the zone of necrosis (in the direction of the unopposed forces). In anterior myocardial infarctions, this produces Q waves in the right and midprecordial leads (V_1-V_4) . However, in a significant number of patients the Q waves do not persist. With previously documented anterior myocardial infarction, the reported estimate of poor Rwave progression on subsequent ECGs varies between 20% and 30%.^{3,6,7} The average length of time for the complete disappearance of the abnormal Q waves is 1.5 years.7 In addition, the magnitude of the subsequent leftward forces is less than in patients with poor Rwave progression from other causes. On the ECG, this results in a diminuation of R-wave amplitude in standard lead I. As a consequence, up to 85% of patients with old anterior myocardial infarction and poor R-wave progression have either an R-wave in lead I of 4 mm or less or an R-wave in lead V₃ of 1.5 mm or less. Absence of these amplitude

criteria makes old anterior myocardial infarction unlikely with only a 10%–15% false exclusion of old myocardial infarction.³ In the setting of poor R-wave progression, the additional presence of ST-T wave abnormalities, would provide support for a diagnosis of old anterior myocardial infarction.

Could our applicant have had an anterior myocardial infarction in the past? It's possible, as her ECG does meet some of the criteria noted above. However, as always, it is important to analyze the ECG in the context of the situation where it was taken. Using Bayesian analysis, the prevalence of coronary artery disease in an asymptomatic 46-year-old female insurance applicant with no risk factors is approximately 1%.8 Therefore, the likelihood, that such an individual's ECG actually represents an old myocardial infarction is very low.

What other possibilities could be responsible for the poor R-wave progression in our applicant's ECG? A number of other conditions result in a relative decrease in the amplitude of anteriorly directed cardiac electrical forces. Some, such as left bundle branch block, left anterior fascicular block, Wolff-Parkinson-White syndrome, certain types of right ventricular hypertrophy (especially that associated with chronic pulmonary disease), and left ventricular hypertrophy are easily recognized.^{3,5} Less common causes of poor Rwave progression include spontaneous pneumothorax, corrected transposition of the great vessels and congenital absence of the pericardium.9

Since none of the above conditions are present, could the poor R-wave progression in our applicant be the result of faulty ECG recording technique? The exact placement of electrodes for ECG recording depends on the skill of the person performing it (eg, nurses, technicians, medical students). Placement of the precordial electrodes is especially subject to variation and is the most common cause of variability in ECGs done for insurance applicants. Precordial electrodes are commonly placed either too high or too low or are hor-

izontally displaced from their anatomically defined sites.¹⁰

If unipolar electrodes are placed on every area of the chest—both front and back—the complexes recorded form a pattern in which only predominately negative QRS complexes are recorded over the upper portion of the chest, and only predominately positive complexes are recorded over the lower portion.3 Poor R-wave progression is frequently caused by a relatively high placement of the right and midprecordial electrodes. The high position of the electrodes as a cause of poor Rwave progression can be confirmed by recording normal R-wave progression from lower and proper locations on the thorax. When poor R-wave progression is due to anterior myocardial infarction, poor R-wave progression or QS waves will persist in the lower leads.9 The possibility of faulty technique in the recording of our applicant's ECG is supported by the negative P waves in V₁ and V₂ and the biphasic P waves in V₃. Normally, the P wave is biphasic in V₁ and upright in $V_2 - V_6$.

DISCUSSION

Poor R-wave progression is a common ECG pattern in which the expected increase of R-wave amplitude in successive right and midprecordial leads does not occur. It occurs in about 10% of hospitalized adult patients and as many as 8% of patients examined for coronary artery disease.⁵ Poor R-wave progression was the sixth commonest abnormal ECG pattern in a series of 19,734 ECGs collected consecutively by Metropolitan Life Insurance Company over a period of 5 ¼ years.¹¹

Despite its prevalence, the definition and risk selection implications of this entity are unclear and controversial. Medical directors often report this pattern with qualifiers such as suggestive but not diagnostic of, consistent with, cannot rule out, possible, or probable anterior myocardial infarction. In an insurance risk selection setting, the lack of a defin-

itive decision frequently results in a delay or loss of important business.

Attempts have been made to improve the predictive accuracy of poor R-wave progression for the diagnosis of anterior myocardial infarction by refining the ECG criteria.^{3,4,12} In these studies, the presence of anterior myocardial infarction was determined with various techniques, including post-mortem studies, vectorcardiography, planar thallium scintography and contrast ventriculography. A recent systematic evaluation of commonly used and previously published criteria for poor R-wave progression was reported by Gami et al⁵ using dual isotope gated single emission computed tomography (SPECT) as the gold standard. This technique is very sensitive for myocardial infarctions and misses only 5% of subendocardial infarctions identified with contrast-enhanced magnetic resonance imaging.¹³ These investigators found that only a small percentage of individuals who met various criteria for poor R-wave progression (a proportion no different than would be expected by chance) had myocardial infarction. They concluded that although poor R-wave progression may occur in the presence and as a consequence of anterior myocardial infarction, the association is not strong and does not differ from that predicted by chance alone. It occurs with sufficient frequency in individuals without anterior myocardial infarction that it does not provide useful discriminant value. They recommended against making specific comments about the presence of anterior myocardial infarction on the basis of the ECG finding of poor Rwave progression.

REFERENCES

- 1. Gauer K, Curry Jr RW. *Clinical Electrocardiography*. Cambridge, Mass: Blackwell Scientific Publications; 1992:87–92.
- 2. Goldberger, AL. *Clinical Electrocardiography: A Simplified Approach.* 5th ed. St. Louis: Mosby, Inc; 1994: 37–40.
- 3. Zema MJ, Kligfield P. ECG poor R-wave progression. *Arch Intern Med.* 1982;142:1145–1148.
- 4. DePace NL, Colby J, Hakki AH, et al. Poor R-wave

- progression in the precordial leads; clinical implications for the diagnosis of myocardial infarction. *JACC*. 1983;2:1073–1079.
- 5. Gami AS, Holly TA, Rosenthal JE. Electrocardiographic poor R-wave progression: analysis of multiple criteria reveals little usefulness. *Am Heart J.* 2004;148:80–85.
- 6. Chou TC, Knilans K. *Electrocardiography in Clinical Practice*. 4th ed. W.B. Saunders; 1996:131.
- 7. Kaplan BM, Berkson DM. Serial electrocardiograms after myocardial infarction. *Ann Intern Med.* 1964;60:434.
- 8. MacKenzie BR. Coronary Heart Disease. In: Brackenridge RDC, Elder WJ. *Medical Selection of Life Risks*. 4th ed. Macmillan Reference Ltd; 1998:329.
- 9. Fisch C. The electrocardiographic QS pattern in leads V₁ and V₂. *ACC Current J Rev.* 1993;May/June: 72–73.

- 10. Lateef F, Nimbkar N, Da ZK, et al. Vertical displacement of the precordial leads alters electrocardiographic morphology. *Indian Heart J.* 2003;55: 339–343.
- 11. Ferrer MF. Electrocardiographic variations, arrythmias, pacemakers. In: Lew EA, Gajewski J, eds. *Medical Risks: Trends in Mortality by Age and Time Elapsed.* New York: Praeger; 1990:7.1–7.64.
- 12. Warner RA, Reger M, Hill NE, et al. Electrocardiographic criteria for the diagnosis of anterior myocardial infarction: importance of the duration of precordial r waves. *Am J Cardiol.* 1983;52:690–692.
- 13. Wagner A, Mahrholdt H, Holly TA, et al. Contrastenhanced MRI detects subendocardial myocardial infarcts that are missed by routine SPECT myocardial perfusion imaging. *Lancet*. 2003;361:374–379.