statement for it is not unlike the statement that we made in the conclusions of our review on page 426. We stated: "There are at present no conclusive data in the medical literature to support the concept that ACBG is superior to adequate medical management to prevent or even ameliorate other signs and symptoms of ischemic heart disease, such as myocardial infarction, arrhythmia and congestive heart failure. The procedure therefore is not recommended for these purposes."

The main message that we hoped to emphasize in the review was that it is indeed surprising after 10 years of experience with between 300,000 and 400,000 patients that we do not know the answer to such important questions. In my judgment, many surgeons and cardiologists believe that surgery does prevent myocardial infarction. If the procedure did in a significant manner prevent myocardial infarction would one not expect it to be apparent after such an extensive and prolonged experience? As has often been said, "it did not take a randomized study to demonstrate the efficacy of penicillin in the treatment of pneumococcal pneumonia"... or... "to be different there must be a difference."

Our conclusion that ACBG should not be recommended for the purpose of prevention of a myocardial infarction still appears to be justified.

Dr. Hammermeister indicated that our discussion of his paper on the "Effect of Aortocoronary Saphenous Vein Bypass Grafting on Death and Sudden Death" may have been misleading. This was not our intent. Figure 1 in his paper showed the Seattle Heart Watch angiographic registry. Three and 56 deaths are identified as "procedural" among the patients reported as being dead in the medically and surgically treated groups, respectively. It is not clear, even now after rereading the manuscript several times, how one is to ascertain that the procedural deaths were excluded from the results of those patients not dying suddenly. Furthermore, the meaning of the classification is not clear even now; nor is it clear why there was such a striking difference, even considering the differences in the size of the two study populations, in the number of procedural deaths among surgical and medical patients. If we erred in the interpretation of this study we apologize.

It is difficult to comment on Dr. Hammermeister's disagreement about our concluding statement in the summary: "Available data in the literature do not necessarily indicate that the initial symptomatic improvement necessarily persists." The statement was supported by three references. A number of other well-evaluated studies could also have been cited. It is not surprising that the numbers would appear more or less impressive from one reader to the next. In our review, we attempted to point out where better results might be attained. Thus, in discussing the gradual clinical deterioration in the conclusions, we stated that, "It is thought that the deterioration in good results is due to progression of the disease and may therefore be preventable."

But the bottom line of the review is that Dr. Hammermeister and I may disagree about the meaning of statements about clinical deterioration because of the profound truth that is articulated in the first paragraph of the summary of the review: "Despite a decade of experience in aorto-coronary bypass grafting embracing 300,000 or more operations, indications for its use remain controversial. The controversy persists because of a lack of adequate controls with which to compare the clinical course of operated patients; only 1,248 have been reported who have been studied in a carefully controlled and random manner."

HENRY D. MCINTOSH, M.D.
JORGE GARCIA, M.D.
Watson Clinic
Lakeland, Florida
and
St. Luke's Hospital
Houston, Texas

References

Lown-Ganong-Levine Syndrome
To the Editor:
I have read with interest the article in the March issue of Circulation entitled "Characteristics of Atrioventricular Conduction and the Spectrum of Arrhythmias in Lown-Ganong-Levine Syndrome" published by Benditt, Pritchett, Smith, Wallace and Gallagher. They have presented 12 cases with various arrhythmias associated with a short P-R interval and normal QRS duration. In 1962 we published an article on the WPW pattern, which was an analysis of 15,000 routine consecutive electrocardiograms in male subjects age 18-24. The incidence of WPW pattern in that study was 46 in 15,000 routine electrocardiograms or 0.3%, only three of which revealed any evidence of rapid heart action. Consequently, we concluded at that time that the term WPW pattern should be used and that WPW syndrome be applied to those individuals who showed evidence of rapid heart action, and I think that this has generally been adopted since that time. Similarly, I would suggest that the LGL syndrome, as described in the article of March Circulation, page 454, should similarly be noted as LGL pattern, rather than syndrome, unless evidence of tachyarrhythmias have occurred. It may well be that later in life such patterns may develop tachyarrhythmias; however, at the age we studied there was very little evidence of any tachyarrhythmias. We also reviewed 2,322 routine normal electrocardiograms, of which 34 had a P-R interval equal to or less than 0.12 seconds. Eleven of these had a delta wave in leads I or II, but all had normal QRS duration. None of these revealed a history of or showed tachyarrhythmia. Our study, of course, is somewhat different in that we were reviewing routine electrocardiograms in healthy, young individuals, whereas in the WPW syndrome and LGL syndrome, tachyarrhythmias had occurred, and on reviewing the electrocardiogram in the absence of tachyarrhythmias this pattern was noted. I do think, however, we should make some difference between the pattern and the syndrome.

As a matter of fact, Burch's classification, published in the Annals of Internal Medicine in 1947, described Class 5 type WPW as a short P-R interval — less than 0.12 with the QRS normal. Burch
and Kimball described five types of WPW patterns, the fifth one being what is now known as the LGL syndrome.

I see many thousands of electrocardiograms in connection with the selection of Aircrew, and although in the recent years I have not done a careful study of these, I am sure that they are quite frequent, i.e., a short P-R interval of 0.12 or less with a normal QRS. If a delta wave is present, then I am more inclined to consider these as a WPW pattern.

Although the follow-up is by no means complete, we have found very little evidence of tachyarrhythmias in the classical WPW pattern. Admittedly, the studies were done in younger men from age 18-24 and it may well be that tachyarrhythmias may develop at a later age in both the WPW pattern and the LGL pattern, however, I thought it of interest to point out that the WPW pattern without symptoms or arrhythmias and particularly the LGL pattern might not carry a poor prognosis as far as tachyarrhythmias are concerned. Of course, a long follow-up study will be necessary. Nevertheless, these patterns appearing in healthy, normal, young people are likely of no significance and particularly the LGL pattern. We are, of course, dealing with different groups. The patient who is found to reveal tachyarrhythmias may well show the WPW or LGL pattern in an electrocardiogram taken during a normal period; whereas we were dealing with normal, healthy, young men whose routine and consecutive tracings were recorded in connection with the R.C.A.F. Aircrew Selection Program.

G. W. MANNING, M.D.
University Hospital
London, Ontario

References

To the Editor:
Benditt et al.1 reported that their stimulation studies in patients with the Lown-Ganong-Levine (LGL) syndrome were done by “delivering impulses of 2 msec duration employing the minimum impulse intensity permitting consistent capture.” A recent observation in our laboratory, however, indicates that pacing by the lowest possible stimulus strength permitting consistent capture may give rise to errors in measurements of electrophysiological parameters. This is demonstrated in the accompanying figure 1, which was recorded from a patient with the Wolff-Parkinson-White syndrome and a right sided accessory pathway. As shown in panel A, stable right ventricular stimulation could be obtained by pacing the ventricle with impulses of 2 msec duration and with the same strength as the value found for the diastolic stimulating threshold which was 0.5 mA. The surprising finding was that following a ventricular extra-stimulus with a V_{1}V_{2} interval of 460 msec, the corresponding A_{1}A_{2} interval measured 430 msec, suggesting that following the ventricular premature beat retrograde, conduction over the accessory pathway was shortened by 30 msec. A more careful analysis of the His bundle lead, however, shows that latency time after the basic pacing stimulus artifact (V_{1}) is longer than following the extrastimulus (V_{2}): 120 msec vs 90 msec.

As shown in panel B, by increasing the stimulus strength to twice diastolic threshold (1 mA), latency time after V_{1} and V_{2} had the same value, resulting in identical V_{1}V_{2} and A_{1}A_{2} intervals. The different values for latency time after the basic and the extrastimulus during programmed cardiac stimulation with strengths just above threshold can be explained on the basis of changes in cardiac dimensions in relation to prematurity, allowing a better and closer contact between the stimulating electrodes and the cardiac tissue.

In our patient, the effect of latency became very evident because in cases with ventriculo-atrial conduction over an accessory pathway, no change in ventriculo-atrial conduction time is expected following premature ventricular activation. In patients with retrograde conduction over the AV node, however, artificial measurements due to latency cannot be so readily identifiable.

Although we do not know how often the problem illustrated in the figure occurs, our observation supports pacing of the heart at twice the diastolic threshold value for accurate measurements during programmed stimulation studies.2

HEIN J. J. WELLENS, M.D.
JERÓNIMO FARRÉ MUNCHARAZ, M.D.
FRITS W. BÄR, M.D.
Annadal Hospital
Maastricht, The Netherlands

References

The authors reply:
To the Editor:
The authors would like to thank Dr. Manning for his comments regarding our recent article.1 We agree with him that patients manifesting a short P-R interval on ECG, but who do not have a history of “rapid heart action,” should not be classified as Lown-Ganong-Levine (LGL) syndrome. In fact, the description of the LGL patient population in our study specified that each patient had to meet the following criteria in addition to having a short P-R interval (Methods, paragraph 1, p 454):
1) a positive history of paroxysmal rapid heart action.
2) electrocardiographically documented tachyarrhythmias (atrial fibrillation or flutter, narrow QRS complex tachycardia, ventricular tachycardia or ventricular fibrillation).

As Dr. Manning points out, patients frequently manifest short P-R intervals on ECG with no history of tachyarrhythmia. We prefer to read these tracings as having a “short P-R interval,” making no reference to either LGL ‘pattern’ or syndrome, since even raising the issue may do more harm than good.

We would also like to point out that in our opinion Dr. Manning’s comparison of findings in Wolff-Parkinson-White syndrome (a condition with a well-defined anatomic substrate,4 and LGL syndrome (a condition in which the anatomic basis is less well understood) will only tend to promote the misconception that these syndromes have a comparable anatomic and pathophysiological basis.

The observation reported by Dr. Wellens and his colleagues is interesting but would have been demonstrated more convincingly had an electrogram from the right ventricle been recorded. In addition, we note that following the increase in stimulus intensity (panel B), the QRS morphology in lead V_{1} has been altered markedly. It is possible that the pattern of ventricular activation has changed in association with the increased stimulus intensity, thereby changing the ‘apparent’ duration of latency recorded on the electrograms provided.

DAVID G. BENDITT, M.D.
EDWARD L. C. PRITCHETT, M.D.
WILLIAM M. SMITH, M.D.
ANDREW G. WALLACE, M.D.
JOHN J. GALLAGHER, M.D.
Duke University Medical Center
Durham, North Carolina
Lown-Ganong-Levine syndrome.
G W Manning

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