Coronary Artery Anatomy in Left Bundle Branch Block

By Henry De Mots, M.D., Josef Rösch, M.D.,
and Shahbudin H. Rahimtoola, M.B., F.R.C.P.

SUMMARY

It has previously been reported that the length of the left main coronary artery is short in patients with left bundle branch block (LBBB) and that an unexpectedly large number of LBBB patients had dominant left coronary arterial distribution. The present coronary arteriographic study of 13 patients with LBBB revealed a mean length of the left main coronary artery (± 1 sn) of 10.9 ± 6.0 mm, a measurement which was not significantly different (P > 0.5) from that of the left coronary arteries in 78 patients in a control group (10.0 ± 3 mm). The arterial distribution patterns showed a contribution of the right coronary artery to the posterior descending artery in 12 of the 13 patients with LBBB. Coronary artery anatomy does not appear related to the presence of LBBB.

Additional Indexing Words:
Coronary arteriography  Left main coronary artery  Coronary arterial distribution pattern

Left bundle branch block (LBBB) is found in association with a variety of cardiac lesions including coronary artery disease, hypertension, valvular heart disease, myocarditis, and various cardiomyopathies. Fibrosis of the conducting system has been found by most investigators to be the histologic abnormality in patients with LBBB. Ischemia is a possible common pathogenetic mechanism producing fibrosis.

Lewis et al. found that the length of the left main coronary artery (LMCA) was less than 6 mm in all but one of 12 patients with LBBB and was longer than 7 mm in a control group of 25 patients. This data suggest that the cardiac diseases associated with LBBB were not etiologically important or that they were important only when the LMCA was short. Because significant etiologic and prognostic implications are raised by these observations we studied a group of patients with LBBB in whom coronary arteriography had been performed. We were unable to confirm their findings.

Materials and Methods

The electrocardiograms of patients studied consecutively with coronary arteriography were examined for the presence or absence of LBBB. LBBB was diagnosed if the QRS interval was 0.12 sec or longer and there was a broad R wave not preceded by a Q wave in I, aVL, and V6 with secondary ST and T wave abnormalities. The patients had been studied because of suspected or known coronary artery disease, in some as a screening procedure prior to valve replacement. Coronary arteriography was carried out using the Judkins technique and radiographs made in the right anterior oblique (RAO) projection were used to measure the length of the LMCA. In each instance, the radiograph showing the longest length was chosen to obviate the problem of shortening during systole. The length of the LMCA was corrected for X-ray magnification by comparing the actual and projected catheter widths. The arterial distribution pattern and the presence or absence of coronary artery disease was noted.

Thirteen patients with LBBB were identified. Seven were women and six were men. Their mean age was 54 years (range 44–71). Five of the patients had only coronary artery disease. Of the remaining eight, two had isolated valve disease, one had combined coronary and valvular disease, two had hypertensive heart disease, two had cardiomyopathy, and one patient had no demonstrable abnormality at cardiac catheterization and coronary arteriography. The control group of 78 patients was drawn from 13 groups of six patients each. The three patients immediately before and the three

From the Division of Cardiology, Department of Medicine and the Department of Radiology, University of Oregon Medical School, Portland, Oregon.

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Reprint requests to: Henry DeMots, M.D., Division of Cardiology, University of Oregon Medical School, 3181 S.W. Sam Jackson Park Road, Portland, Oregon 97201.

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after the index patient with LBBB comprised these groups.

Results

The left coronary artery of a patient with a long LMCA and LBBB is shown in figure 1. Figure 2 demonstrates a patient with normal conduction and a very short LMCA. The distribution of lengths of the LMCA in all patients is shown in figure 3. The mean length (± 1 sp) of the LMCA in patients with LBBB was 10 ± 6 mm and in the control group was 10.9 ± 3.6 mm. The difference between the two groups was not statistically significant ($P > 0.5$). The right coronary artery was dominant in nine, the left in one, and a balanced system was found in three patients.

Discussion

There was no significant difference in the lengths of left main coronary artery (LMCA) in patients

![Figure 1](image)

*Figure 1*

This long (18 mm) left main coronary artery was present in a man with coronary artery disease and aortic valvular disease. The patient developed left bundle branch block at a time remote from his surgery following a progressively severe intraventricular conduction delay associated with left ventricular hypertrophy.
with and without left bundle branch block (LBBB). The arterial distribution pattern of patients in our series does not differ from those described by other workers.

In contrast to the patients described by Lewis et al., all of the patients with no discernable LMCA or a common ostium were in the control group rather than the group with LBBB.

We are unable to confirm the previously described angiographic patterns. Though the mean values of the lengths of the LMCA in patients in the two control groups were similar, the distribution curve differs in the complete absence of short LMCA in patients in their control series. The small number of patients in their group may explain this difference. There is no apparent reason for the difference in length between the two LBBB groups though each group is relatively small.

Correction for X-ray magnification by measurement of catheter diameters is not entirely satisfactory. However, our study was a retrospective one designed to compare the LMCA length distribution curve in patients with LBBB and a group of controls rather than to establish standards for the length of the LMCA. Because the ratio of tube to film distance was constant and that of image to film distance varied only a little, the error introduced by this factor was small and should not affect the results in a systematic fashion. Further, because a similar method was used in the work referred to above, a difference in techniques cannot explain the differences in the observed lengths of the LMCA.

Lewis et al. speculated that the association between a short LMCA and LBBB could be explained by greater shearing forces imposed during systole in the short arteries. This, in turn, might compromise flow through the early septal branches of the left coronary system and thus produce ischemia and fibrosis of the left bundle branch. However, in patients with LBBB, disease of the left bundle is diffuse rather than accelerated and well localized in a portion of the conducting system as would be expected if only flow through the small septal arteries were compromised.

Frink and James reported a dual blood supply to the anterior half of the left bundle in four of ten hearts. In these same hearts, the posterior division
of the left bundle was supplied completely either by the right coronary or by both the right and left coronary arteries in nine of ten. Therefore, a mechanism involving only the left coronary system would not be expected to produce LBBB unless there were no contribution to septal flow by the right coronary artery, a circumstance found in only one of our 13 LBBB patients.

It is therefore likely that the presence or absence of LBBB in a given patient is related to apparently random and unpredictable localization of lesions occurring in a number of clinical entities. The etiology of LBBB does not appear to be influenced by a congenital predisposition associated with the length of the LMCA or the distribution pattern of the coronary arterial system.

References

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