also relieve the pain, but the effect is usually delayed. For these reasons, we do not hesitate to use one or two intravenous doses of steroids. If the pain persists, the patient is placed on a three-day tapering course of steroid therapy or is given aspirin. Since there is some evidence that a prolonged course of steroid therapy might interfere with wound healing,¹ we have not continued a course of steroid therapy for longer than three days.

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REFERENCES

Left Anterior Hemiblock or Inadvertent Lead Misplacement?

To the Editor:

In regard to the article entitled “Left Anterior Hemiblock Concealing Diaphragmatic Infarction and Simulating Anteroseptal Infarction” by Dhingra and co-workers,¹ which appeared in the June 1975 issue, we analyzed the tracings presented in the report, with particular attention to Figure 1B, which is supposed to represent the main thrust of the article. We wish to emphasize several points of interest. It is strange that all the precordial leads are very similar; there are Qr complexes in leads V₁ to V₄ and a QS configuration in lead V₅ with elevated ST segment and negative (ischemic) T waves. There are no significant changes in the P-wave morphology in the precordial leads, while such changes are evident in the first and last electrocardiograms. The mean AQRS in the frontal plane is at −45°, pointing upwards and to the left; the AT is directed at +50° downwards and to the left. These frontal vectors are definitely incompatible with the mean axis in the horizontal plane, as calculated from the precordial leads, where AQRS is directed approximately at −175° with the terminal portions pointing at +90°, while the horizontal AT points at −100° strongly to the right.

The authors of the article try to explain these patterns by left anterior hemiblock producing a block in the septal activation simulating an anteroseptal infarction. Although we recognize that a delay in the septal activation could lead to a Q wave in leads V₁ and V₄, this does not explain the pathologic Q waves in leads V₁ to V₄. This behavior suggests an extensive anterior infarction (with extension to the free wall of the left ventricle); and, therefore, it cannot be explained by the same phenomenon. The existence of a “block” in the anterolateral portions of the left ventricle would have to be admitted as a less-than-satisfactory explanation.

Since this ECG can induce wrong concepts about the behavior of left anterior hemiblock, we believe that it is pertinent to emphasize the discrepancies.

We think that there has been a technical error when obtaining the ECG. We can’t state which of some possible variants produced the tracing. It may have happened that the chest lead was placed on the left leg, whose electrode was used to register the precordial leads with the electrocardiographic selector switched on V or CF. This type of technical error could produce the ECG shown, i.e., a simulated left anterior hemiblock and the extensive anterolateral infarction. We reproduced a tracing of a patient with an inferior infarction while switching the leads as described and obtained a very similar pattern (Fig 1). Another possibility, which seems less likely, is that the activation process of the heart changed while all the precordial leads were being registered.

Intraventricular blocks have been an interesting subject in electrocardiography while they may conceal or simulate other conditions;²³ however, in the present case, one should be cautious in accepting the facts as presented by the authors in view of the aforementioned comments.

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REFERENCES
**FIGURE 1.** A (upper). Electrocardiogram of patient with old diaphragmatic infarction. B (lower). Electrocardiogram of same patient with chest lead placed on left leg and with left-leg electrode used for chest leads. Note simulation of left anterior hemiblock concealing diaphragmatic infarction and appearance of extensive anterolateral infarction. CF leads (lower) magnify anterior ischemia and necrosis.

...electrocardiographic and vectorcardiographic diagnosis of the left anterior subdivision block isolated and associated with RBBB. Am Heart J 83:447, 1972


**To the Editor:**

We thank Drs. Horwitz and Medrano for calling our attention to the possibility of a technical error on our part while obtaining the electrocardiogram (Fig 1B) published in our recent article (Chest 67:713-715, 1975). There does appear to be an incompatibility between the frontal and horizontal planes. We repeated the experiment of Horwitz and Medrano in our recently reported patient (now with old diaphragmatic infarction) and were able to reproduce identical electrocardiographic changes by switching the leads as described. Thus, the interpretation of inadvertent lead misplacement is almost certainly the correct explanation for the unusual electrocardiographic changes described in our patient. To further support this, we would point out that the ECG in question was taken by one of our house staff at 1:00 AM, increasing the likelihood of human error. We commend Horwitz and Medrano for their astute observations.

This case emphasizes the importance of correct lead placement and the need for utmost care in interpreting sudden major transient changes in QRS morphology. The principles elucidated by Horwitz and Medrano are certainly useful in detecting this type of technical mishap.

In view of this communication, we would like to take the opportunity to propose a revised title for our recent “Electrocardiogram of the Month,” this being “Inadvertent Lead Misplacement Simulating Left Anterior Hemiblock, Concealing Diaphragmatic Infarction, and Simulating Anteroseptal Infarction.”

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450 COMMUNICATIONS TO THE EDITOR