

Letter to the Editor

Intracoronary electrocardiographic deflections during transmural ischemia induced by percutaneous transluminal coronary angioplasty

Intracoronary (IC) leads have been shown to project local ischemia during percutaneous transluminal coronary angioplasty. Transient compromise in antegrade blood flow during balloon inflation causes alterations in the amplitude of the R wave and duration of the QT interval on the surface 12-lead electrocardiogram (ECG). In our article titled “R-wave Amplitude During Transmural Ischemia,”¹ we briefly presented the changes in R-wave amplitude recorded through an IC-lead during balloon inflation in 4 patients. Subsequently, 4 letters²⁻⁵ to the editor were published showing interest in our results and expressed legitimate criticism to the IC-tracing presented in our article.

We would like to dwell more on the method used for IC-ECG recording. Intracoronary recordings were obtained by connecting the lead to the proximal end of the guidewire outside the body. The conducting wire was inside the lumen of the angioplasty balloon with the tip of the wire in the coronary artery well beyond the site of balloon occlusion. The machine voltage was not changed any time during the recording. Even if this were to happen, this should not affect the data since the measurements were made using automated technology and therefore not influenced by the gain. The presented IC-tracing is perplexing as Madias⁴ mentioned. The amplitudes of the whole PQRST segments increased after balloon inflation; a finding likely to be related to physical (conductivity driven) rather than electrochemical factors. After this astute observation, we reviewed the IC-ECGs of all 4 patients and scrutinized the amplitudes in all beats at baseline and during balloon inflation. The amplitude of the R wave increased, the S wave regressed, and ST-segment elevation was witnessed with an increase in the T-wave amplitude in all 4 patients (Table 1); findings consistent with true ischemia. Further analysis of the deflections presented in the table reveal lower absolute increments in the R-wave and T-wave amplitudes in patients 2 and 3. This finding is likely explained by ischemic preconditioning due to prolonged occult ischemia that these patients were experiencing before presenting for percutaneous coronary intervention. Another explanation would be the variation in the amount of myocardium subtended by the blood vessel intervened upon which might vary between patients. And in our opinion, the amount of jeopardized myocardium reflects on the changes on the 12-lead ECG.

Table 1

The amplitudes (mm) recorded on the IC-ECG in the four patients at baseline and during balloon inflation (B1)

	P	Q	R	S	STJ	T
Patient 1						
Baseline	0.68	0	16.93	0	1.12	2.88
B1	0.34	1.22	71.88	0	13.90	56.60
Patient 2						
Baseline	1.41	17.03	149.76	0	3.75	-12.44
B1	1.22	15.37	159.38	0	5.22	-17.08
Patient 3						
Baseline	0.16	0	94.13	60.46	1.36	23.96
B1	0.13	0	101.99	37.13	13.61	48.75
Patient 4						
Baseline	3.80	2.87	32.84	51.58	3.56	19.86
B1	1.14	8.10	103.84	12.73	16.10	50.11

The IC-recording presented in our original paper was obtained during the first 10 seconds of balloon inflation where the wire could have been in contact with the myocardium causing the proportional deflection changes. This was not reproduced in any of the other patients nor with successive balloon inflations (Fig. 1). The IC-recording during the second 10 seconds of the first balloon inflation in patient 4 is presented in Fig. 2.

In summary, we appreciate the interest generated by our paper. The real electrographic changes very early in ischemia and during cessation of myocardial blood flow can be studied with our model in humans with very little risk to the patient, keeping in mind the limitations inherent to the patient population (ischemic preconditioning) and the short duration of allowable cessation of blood flow. Further studies will have to be done to determine the precise mechanisms of the observations we have reported rather than abandoning the whole R-wave/percutaneous coronary intervention model.

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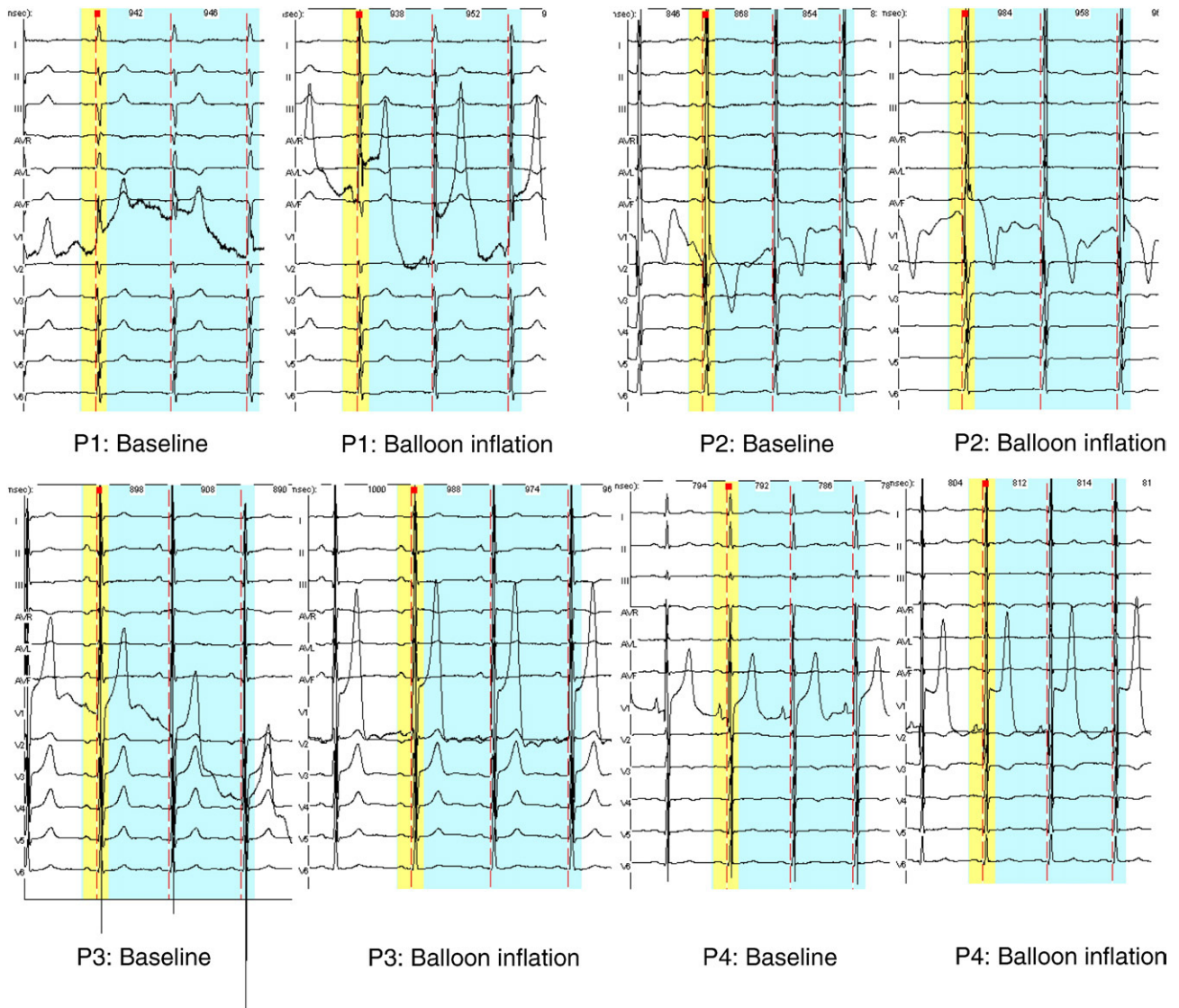
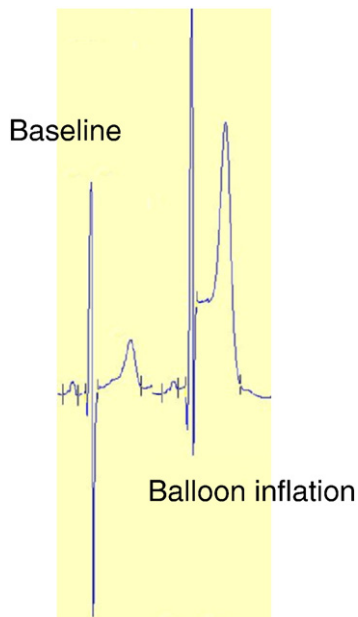


Fig. 1. The change from baseline obtained on an IC-tracing from all four patients during balloon inflation (the amplitudes of the PQRST segments are presented in the table above). Color illustration online.



References

1. Sinno MC, et al. R-wave amplitude changes measured by electrocardiography during early transmural ischemia. *J Electrocardiol* 2008;41:425.
2. Birnbaum Y. Understanding the dynamic electrocardiographic changes that occur during ischemia. *J Electrocardiol* 2008;41:431.
3. Childers R. R wave amplitude change with ischemia. *J Electrocardiol* 2008;41:433.
4. Madias JE. R-wave amplitude augmentation with myocardial ischemia: a conundrum defying complete resolution. *J Electrocardiol* 2009;42:296.
5. Zhong-Qun Z, Shu-Yi D, Chong-Quan W. Use of intramyocardial conduction delay during early transmural ischemia to interpret electrocardiograms. *J Electrocardiol* 2008;41:434.

Fig. 2. The change magnified in one beat obtained from patient 4 at baseline and balloon inflation obtained during the second 10 seconds of the first balloon inflation. The amplitude of the R wave increased, the S wave regressed, ST-segment elevation was witnessed with an increase in the T-wave amplitude, all consistent with true ischemia. Color illustration online.