Noninvasive Assessment of Seasonal Variations in Cardiac Structure and Function in Cyclists

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SUMMARY Noninvasive studies were performed in 12 male bicyclists in the competitive season (CS) and in the resting season (RS) and in 12 matched control subjects to determine seasonal variations in cardiac structure and function in athletes and to compare the data with those of nonathletes. In athletes, peak oxygen uptake was 6% lower in the RS than the CS; the RS value was 40% higher than in nonathletes. The echocardiograms of athletes showed a higher left ventricular total diameter at end-diastole in the CS than in the RS; this difference was due to a greater septal and posterior wall thickness, with unchanged internal diameter. On the ECG, R-wave voltages were larger in the CS in leads I, V5, and V6. Athletes had greater left ventricular dimension and wall thickness than nonathletes, and their ratio of wall thickness to internal radius of the left ventricle was higher. Various echo- and mechanocardiographic indexes of left ventricular function were determined. During the RS, the athletes had a lower percent shortening and maximal velocity of left ventricular internal diameter, lower maximal and minimal velocities of the endocardium of the posterior wall, a longer preejection period and a larger ratio of preejection period to left ventricular ejection time. These findings are probably related to a greater left ventricular end-systolic stress, an index of myocardial afterload, in the RS.

We conclude that cyclists in the CS, compared with nonathletes, have greater left ventricular internal dimension and wall thickness, with similar left ventricular function. During the RS, internal dimension does not change from the level in the CS, but wall thickness is somewhat reduced and left ventricular function is slightly depressed, most likely because of a higher afterload in RS.

ENDURANCE TRAINING induces changes in cardiac structure, as revealed by the comparison of the hearts of athletes with those of nonathletes and longitudinal studies in which athletes and nonathletes are followed during training. Whereas radiologic examination of the chest can only demonstrate overall enlargement of the heart of endurance athletes, echocardiographic studies (except one) reveal that the left ventricular (LV) internal diameter is significantly increased, with an increase of septal and posterior wall thickness in most, but not all, reports. Calculated muscle mass was greater in athletes. Endurance training of nonathletes may affect LV dimension and wall thickness, although not consistently. Such differences are associated with higher voltages on the ECG in athletes or in nonathletes after training. The structural adaptation of the heart allows the generation of a higher stroke volume, which permits a higher maximal cardiac output and oxygen uptake. Some studies using echocardiography also suggest that ventricular performance is enhanced in athletes or in response to training, but others do not agree, or even report depressed LV function.

In most studies, athletes in their active season are compared with non- or less active subjects. Only two reports, one of oarsmen and one of swimmers and runners, describe the effects of training or detraining in athletes. In the present paper we report an extensive assessment of seasonal variations in cardiac function and structure in male Belgian cyclists using noninvasive techniques: ECG, echocardiogram, systolic time intervals (STIs) and bicycle ergometer stress testing. The data are compared with those of nonathletes, matched for age, height and weight to allow comparison of absolute data.

Materials and Methods

Twelve male cyclists, six professionals and six amateurs, ages 17–35 years, were studied. They had been involved in competitive cycling for an average of 7.6 years (range 4–10 years). They were all evaluated during the competitive season (CS) of 1981 (between May 15 and October 26). Data during the resting season (RS) were obtained in the previous winter in six (between December 12 and January 23), and in the winter after the CS in the other six (between November 27 and February 2). The athletes had ended the previous CS an average of 12 weeks before the RS evaluation (range 8–17 weeks). After ending the CS, they stopped all sports activities for 8 weeks (range 3–14 weeks); at the time of the RS investigation, some had taken up light power training and cross-country running, but not cycling. As revealed by questionnaire, an average of 18,250 km were covered in 1981 during training and competition (range 10,000–22,900 km) for the amateurs and 43,900 km (range 40,000–46,700 km) for the professionals. Twelve male nonathletes, matched for age, height and weight, were studied according to the same protocol between February 20 and November 2, 1981. They were not involved in competitive sports, but recreational sports activities were allowed, which...
they engaged in for an average of 1.5 hours a week (none in three, 1–2 hours in eight, and 6 hours in one subject).

Each subject was investigated in the morning. A standard 12-lead resting ECG was recorded on an Elema-Schöndner Mingograf 34 three-channel electrocardiograph at a paper speed of 25 mm/sec. QRS voltages were carefully measured and the magnitude and direction of the mean electrical axis (frontal plane) calculated.19

Echocardiography was performed, always by the same investigator and with the subject rotated slightly on his left side, using a 2.25-MHz transducer and a commercially available Irex system II ultrasonic unit with photographic paper. Measurements were recorded at end-expiration. The transducer was angulated to ensure simultaneous visualization of the LV posterior wall (LVPW) and the interventricular septum (IVS) just below the mitral valve apparatus, i.e., at the level of the posterior chordae tendineae. Care was taken to obtain RS and CS echocardiographic records from the same interspace with identical transducer angulation. For analysis, comparable areas of the left ventricle were used. The echocardiograms were processed using a digitizer and computer program as described.20 The analysis of the echocardiograms was done on each of the three successive heart beats and the values were then averaged. Both LV dimensions and dimensional changes were studied. Wall and cavity measurements were made using the leading edge method (from the most anterior edge of endocardial and epicardial lines). The following echocardiographic measurements were obtained: (1) LV end-diastolic total and internal diameter (LVTdD and LVIDd), measured at the onset of the QRS complex, and LV end-systolic internal diameter (LVIDs), taken as the minimal LV dimension; (2) LVPW and IVS thickness at the onset of QRS (LVPWTd and IVStd) and the maximal thickness during systole (LVPWtS and IVSs); (3) maximal and minimal velocities (Velmax and Velmin) of the LV end and of the endocardium of the LVPW; Velmax of the LVPW and Velmin of the LVID occur during contraction and Velmin of LVID and Velmax of the LVPW during relaxation. The percent shortening of the LVID was calculated as [(LVIDd - LVIDs)/LVIDd] × 100. The cross-sectional area (CSA, cm²) of the LV wall in a transverse plane was determined from the echocardiographic dimension and wall thickness at end-diastole as \( \pi \left[ \frac{LVIDd}{2} + (IVStd + LVPWTd)/2 \right]^2 - \pi \left[ \frac{LVIDd}{2} \right]^2 \). This index of LV myocardial mass does not require major assumptions regarding ventricular geometry.21

End-systolic LV meridional wall stress (mm Hg), a quantitative index of myocardial afterload, was calculated as 0.334 × P × LVIDs/LVPWTs \[ 1 + \frac{LVPWTs}{LVIDs} \],22,23 where P = systolic blood pressure.

A carotid arterial tracing and heart sounds were recorded at end-expiration using Siemens-Elema-Schöndner equipment. STIs were computed from simultaneous recordings of ECG lead II, heart sounds (microphone 25 C) and a carotid arterial tracing (pulse transducer 860.2) on a four-channel recorder (Mingograf 81), with the subjects supine. Electromechanical systole was measured from the onset of the QRS complex to the high-frequency vibrations of the second heart sound (QS₂) and LV ejection time (LVET) from the upstroke to the dicrotic notch of the carotid tracing; the pre-ejection period (PEP) was calculated as (QS₂ - LVET). The data from three successive beats of each of three different recordings were averaged.

Each subject underwent a graded, uninterrupted exercise test on a bicycle ergometer (Siemens 380 B) until exhaustion. The initial external work load was 20 W and was increased by 30 W every 4 minutes. Pulmonary ventilation (V̇e) was continuously measured by a pneumotachograph, and oxygen uptake (VO₂) was determined from the measurement of oxygen in the expired air (Siemens FD 84). The respiratory equivalent for oxygen was calculated as V̇e/VO₂. Heart rate was calculated from the ECG.

All data were analyzed at the end of the data collection. The tracings were read in a random order. For statistical analysis, the data of the athletes in the RS and the CS were compared by paired t tests. The results of the nonathletes were compared with those of the athletes in RS and in CS by unpaired t tests. The STIs were related to the RR interval by regression analysis within each group; furthermore, individual STIs were reduced to the values at 1 second using the regression equation of the group. The dispersion of the data is given by the standard error of the mean.

Results

General Characteristics

Table 1 summarizes the characteristics of the subjects. The athletes did not differ from the nonathletes in age, height and weight. The athletes weighed less in the CS than in RS (p < 0.05). Resting blood pressure was not different. Peak oxygen uptake averaged 4.37 ± 0.13 l/min for the cyclists in the RS, which was significantly different (p < 0.001) from the value in nonathletes. Furthermore, VO₂ peak of the athletes was 6.2 ± 1.4% higher in the CS (p < 0.001). This was associated with a significantly lower submaximal heart rate in the CS than in the RS (11 beats/min less at 140 W, p < 0.01), but peak heart rate was also lower in CS (5 beats/min, p < 0.05). The peak heart rate of the control subjects (192 ± 2 beats/min) was not significantly different from that in athletes. The peak V̇e/VO₂ was not different in athletes during the CS compared with the RS, but was significantly higher in the nonathletes (p < 0.01).

Electrocardiography

A comparison of voltage criteria in various leads (R waves in peripheral leads and V₅+6 and S waves in V₁-3) for the athletes in RS and CS revealed a significantly higher R wave in leads I, V₅ and V₆ in the CS (p < 0.05) (table 2). The R waves in V₅ and V₆ of the athletes in CS differed significantly from those of non-
athletes ($p < 0.05$), but there were no significant differences between the voltages in the athletes in RS and those in the nonathletes. The mean QRS axis and the mean QRS voltage were similar for the three groups.

**Echocardiography**

Tables 3A and 3B summarize the echocardiographic findings. LVTDD, LVIDd, LVIDs, IVSTD and LVPWTd were significantly larger in athletes than in the nonathletes ($p < 0.05$). In the CS, the athletes had a greater LVTDD than in the RS ($p < 0.01$) because of a thicker IVS and a thicker LVPW ($p < 0.05$) with unchanged LVIDd. The ratio of LV wall thickness to internal radius (h/R) at end-diastole, calculated as (LVPWTd + IVST)/LVIDd, was significantly higher in cyclists in the CS than in the RS ($p < 0.05$), and it was smaller in the nonathletes ($p < 0.05$). Calculated surface CSA had increased by 2.4 ± 0.6 cm² in the CS compared with the RS ($p < 0.01$), and was significantly smaller in the nonathletes ($p < 0.001$).

The indexes of myocardial function did not differ between athletes and nonathletes. However, during systole, the percent shortening of the LVID ($p < 0.05$), and the Velₘₜ of the endocardium of the posterior wall ($p < 0.06$) were smaller in the RS than in the CS in the athletes. During relaxation, both the Velₘₜ of the LVID ($p < 0.01$) and the Velₘₜ of the endocardium of the posterior wall ($p < 0.02$) were smaller in the RS. LV end-systolic stress was significantly higher in the RS than in the CS ($p < 0.05$).

**Systolic Time Intervals**

The relationships between the QS₂ and the RR interval and between LVET and the RR interval were significant for the controls, athletes in the RS and for the athletes in the CS ($r = 0.65-0.94; p < 0.05$). For the PEP, the relationship was only significant ($r = +0.62; p < 0.05$) for athletes in the RS. When the QS₂ and LVET, corrected to an RR interval of 1 second (QS₂ₑₛ and LVETₑₛ) according to the appropriate regression equation, were compared, no differences were found between the groups (table 4). PEPₑₛ in athletes in

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**Table 1. General Characteristics of the Subjects and Exercise Data**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Athletes RS</th>
<th>Athletes CS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24.4±1.6</td>
<td>—</td>
<td>23.6±1.4</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.79±0.02</td>
<td>—</td>
<td>1.78±0.01</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.5±1.9</td>
<td>72.0±1.5</td>
<td>70.4±1.3††</td>
</tr>
<tr>
<td>Resting systolic blood pressure (mm Hg)</td>
<td>124±3</td>
<td>127±2</td>
<td>123±2</td>
</tr>
<tr>
<td>Resting diastolic blood pressure (mm Hg)</td>
<td>83±3</td>
<td>79±3</td>
<td>81±3</td>
</tr>
<tr>
<td>Peak VO₂ (l/min)</td>
<td>3.04±0.11**</td>
<td>4.37±0.13</td>
<td>4.63±0.12§§</td>
</tr>
<tr>
<td>Peak VO₂ (ml/min/kg)</td>
<td>43.1±1.62**</td>
<td>60.9±1.5</td>
<td>65.9±1.3§§</td>
</tr>
<tr>
<td>Exercise time (min)</td>
<td>30.9±1.1†††</td>
<td>44.0±1.3</td>
<td>46.5±1.2§§</td>
</tr>
<tr>
<td>Peak VE/VO₂</td>
<td>35±1***</td>
<td>29±1</td>
<td>28±1</td>
</tr>
<tr>
<td>Heart rate, sitting (beats/min)</td>
<td>73±4§§</td>
<td>60±3</td>
<td>60±3</td>
</tr>
<tr>
<td>Heart rate, at 140 W (beats/min)</td>
<td>151±6**</td>
<td>115±4</td>
<td>104±3‡‡‡</td>
</tr>
<tr>
<td>Heart rate, peak (beats/min)</td>
<td>192±2</td>
<td>192±3</td>
<td>187±3†††</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.

Controls vs athletes RS:

* $p < 0.05.

† $p < 0.01.

‡ $p < 0.001.

Controls vs athletes CS:

§§ $p < 0.05.

¶ $p < 0.01.

** $p < 0.001.

Athletes CS vs athletes RS:

†† $p < 0.05.

‡‡ $p < 0.01.

§§§ $p < 0.001.

All other comparisons: NS.

Abbreviations: VO₂ = oxygen uptake; VE/VO₂ = respiratory equivalent for oxygen; RS = resting season; CS = competitive season.
The purpose of the present investigation was to study seasonal cardiac variations in cyclists and to compare these data with a control group of nonathletes matched for age, height and weight. All athletes were investigated during one competitive season; to avoid systematic errors due to adaptation to the investigative procedures, half were studied during the previous and half during the following RS.

The VO₂ for a graded, uninterrupted exercise test until exhaustion was about 6% lower for athletes in the RS than in the CS, but was still 40% higher than that of nonathletes. No attempt was made to determine true VO₂max. The peak heart rate of 192 beats/min and the Vₑ/VO₂ of 35 of the nonathletes indicate that they performed up to exhaustion. The athletes obtained a peak heart rate in the RS identical to that of nonathletes, but it was significantly lower, by 5 beats/min, in the CS, despite a similar peak Vₑ/VO₂ in the RS and the CS. It is thus possible that the intensive training of the CS decreases peak heart rate.24 Furthermore, the lower peak Vₑ/VO₂ in athletes compared with the control subjects suggests a lesser metabolic acidosis in athletes.

During the RS, the heart size of the athletes was significantly less than during the CS. The 1.9-mm decrease of total LV end-diastolic diameter was entirely due to reductions of IVST and PWT, with unchanged LVID. The unchanged LVID may have reflected that heart rate was not different in the RS and the CS at the time of the echocardiographic recording; the duration of diastolic filling, which is inversely related to heart rate, is indeed considered a major determinant of end-diastolic volume. Other studies on training or detraining of athletes and of nonathletes found significant changes of wall thickness10–12 and/or LVID10–13 or no changes at all.14,15

In both the RS and the CS, the LVID and the wall thickness of the athletes’ hearts were significantly larger than those of the nonathletes, in agreement with most previous studies of athletes engaged in mainly isotonic sports activities, such as runners,1–3 skiers,1–3 basketball players2 and oarsmen.10 Nishimura et al.2 studied three age groups of Japanese cyclists. The younger group, ages 20–29 years, which is similar to the age of our athletes, showed dilatation and no thickening of the ventricular wall, but both the septum and

### Table 3A. Echocardiographic Data on Cardiac Structure

<table>
<thead>
<tr>
<th>Heart rate (beats/min)</th>
<th>Controls</th>
<th>Athletes RS</th>
<th>Athletes CS</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd (mm)</td>
<td>63.8 ± 2.8**¶</td>
<td>54.9 ± 2.2</td>
<td>52.5 ± 1.7</td>
</tr>
<tr>
<td>LVIDs (mm)</td>
<td>49.4 ± 2.9**¶</td>
<td>55.0 ± 0.9</td>
<td>55.0 ± 1.0</td>
</tr>
<tr>
<td>IVSTd (mm)</td>
<td>32.1 ± 0.74¶</td>
<td>37.5 ± 0.8</td>
<td>36.5 ± 1.0</td>
</tr>
<tr>
<td>IVSTS (mm)</td>
<td>9.1 ± 0.25**¶</td>
<td>11.5 ± 0.4</td>
<td>12.3 ± 0.4††</td>
</tr>
<tr>
<td>LVWTd (mm)</td>
<td>17.6 ± 0.52**¶</td>
<td>18.0 ± 0.6</td>
<td>19.4 ± 0.5††</td>
</tr>
<tr>
<td>CSA (cm²)</td>
<td>17.6 ± 0.52**¶</td>
<td>24.5 ± 1.1</td>
<td>26.8 ± 1.1††</td>
</tr>
</tbody>
</table>

See table 1 for significance of comparisons.

**Abbreviations: RS = resting season; CS = competitive season; LVIDd = left ventricular end-diastolic total diameter; LVIDs = LV end-diastolic internal diameter; LVIDs = LV systolic internal diameter; IVSTd = interventricular septal wall thickness at end-diastole; IVSTS = interventricular septal wall thickness during systole; LVWTd = LV posterior wall thickness at end-diastole; LVWTs = LV posterior wall thickness during systole; h/R = ratio of wall thickness to internal radius at end-diastole; CSA = cross-sectional area.

### Table 3B. Echocardiographic Data on Cardiac Function

<table>
<thead>
<tr>
<th>During systole</th>
<th>Controls</th>
<th>Athletes RS</th>
<th>Athletes CS</th>
</tr>
</thead>
<tbody>
<tr>
<td>% shortening of LVID (%)</td>
<td>35.0 ± 1.3</td>
<td>31.8 ± 1.2</td>
<td>33.6 ± 1.2††</td>
</tr>
<tr>
<td>Velₘᵢₙ ᵄ LVID (mm/s)</td>
<td>−114 ± 5</td>
<td>−107 ± 4</td>
<td>−107 ± 5</td>
</tr>
<tr>
<td>Velₘᵃₓ ᵄ LVPW (mm/s)</td>
<td>76.7 ± 5.3</td>
<td>62.5 ± 5.9</td>
<td>74.9 ± 2.3¶¶</td>
</tr>
<tr>
<td>During relaxation</td>
<td>181 ± 10</td>
<td>164 ± 5</td>
<td>189 ± 7††</td>
</tr>
<tr>
<td>Velₘᵢₙ ᵄ LVPW (mm/s)</td>
<td>−162 ± 9</td>
<td>−144 ± 9</td>
<td>−172 ± 9††</td>
</tr>
<tr>
<td>LV end-systolic stress (mm Hg)</td>
<td>53.0 ± 1.9</td>
<td>60.7 ± 3.8</td>
<td>50.9 ± 2.6††</td>
</tr>
</tbody>
</table>

See table 1 for significance of comparisons; ‡‡p < 0.06 between RS and CS.

Abbreviations: RS = resting season; CS = competitive season; LVID = left ventricular internal dimension; Velₘᵢₙ = minimal velocity; Velₘᵃₓ = maximal velocity; LVPW = left ventricular posterior wall; LV = left ventricular.

the RS was significantly greater than that in athletes in the CS and in the control group (p < 0.05), but the value was not different between the athletes in CS and the nonathletes. The PEP/LVET tended to be higher in athletes in the RS than in the CS (p < 0.06); furthermore, the PEP/LVET in athletes in the RS was greater than the ratio in the nonathletes (p < 0.02), whereas the ratio was not different between athletes in the CS and nonathletes.

### Discussion

The purpose of the present investigation was to study seasonal cardiac variations in cyclists and to compare these data with a control group of nonathletes
the posterior wall were thickened in 40–49-year-old athletes. Our data are, however, in agreement with and very close to the findings in another study of young Belgian cyclists. The difference between Belgian and Japanese cyclists may relate to the younger age at which our cyclists started their sports activities. Indeed, the younger group of Japanese cyclists, whose average age was 25.2 years, had an athletic history of only 5 years, whereas our athletes, average age 23.6 years, had been involved in competitive cycling for 7.6 years. It is also possible that competitive cycling is more demanding in Western Europe than in Japan.

The ratio of the wall thickness to the internal radius of the left ventricle is of particular interest. The ratio was significantly higher in the cyclists, both in CS and in RS, than in the control group, and the ratio had increased from the RS to the CS in the athletes. This disproportionate increase of wall thickness to internal dimension suggests that both volume and pressure overload affect the left ventricle of these athletes. Cycling involves isometric exercise of the muscles of the arms and the upper part of the body; this type of exercise primarily causes thickening of the ventricular wall in relation to the increase of lean body mass, without an increase in size. Furthermore, systolic blood pressure is high during cycling and therefore elevated for many hours a day during CS; during the exercise test in the CS, systolic pressure averaged 200 ± 4 mm Hg at 70% of peak VO₂.

In the CS, the ECG of the athletes showed significantly higher R waves than in RS in leads I, V₅, and V₆. This finding is compatible with the significant difference in calculated CSA of the LV wall. The R waves in V₅ and V₆ of athletes in CS differed from the values of nonathletes, but the latter were not different from those of athletes in RS. In general, our electrocardiographic data are in agreement with the follow-up of normal subjects or athletes during a training period, and with data comparing athletes in their competitive season with nonathletes. Also, in agreement with Van Ganse et al. but not with Parker et al., the QRS axis of athletes was not different from that in controls.

Several indexes of LV function, derived from echo- and mechanocardiographic measurements, were studied. When athletes were compared in the CS and in the RS, the echocardiogram revealed a slightly but significantly reduced fractional shortening of the LVVID in the RS, together with a lower velocity of the posterior wall during contraction. During relaxation, the velocities of the LVVID and of the posterior wall were smaller in the RS than in the CS. As for the STIs, the PEPₗ, and the PEP/LVID were higher in athletes in the RS. LV function is the result of the interactions of several variables, such as preload, afterload and the myocardial contractile state. The athletes’ LVVID, as an expression of preload, was similar in the RS and in the CS. However, LV meridional end-systolic stress, a quantitative index of myocardial afterload, was higher in the RS than in the CS. This may explain the differences in the various indexes of LV function. Therefore, these differences cannot be interpreted as evidence of changes in myocardial contractile state from the CS to the RS.

Since the echocardiographic indexes of athletes are not significantly different from those of nonathletes, as in most other studies, it is difficult to interpret the seasonal variations in athletes. However, the findings that the echocardiographic data of nonathletes are closest to those of athletes in the CS and that PEP/LVID is similar in nonathletes and athletes in CS, but higher in athletes in the RS, suggest that the detraining during the RS induces an increase of LV end-systolic stress and a decrease of ventricular function, rather than the inverse. However, differences in end-diastolic diameter and heart rate may hamper the comparison between athletes and nonathletes.

The data on cardiac function could be in agreement with the finding that older Japanese cyclists who reduced their activity to 1–2 hours of daily training have lower ejection fractions than age-matched sedentary subjects and that former athletes with only light leisure physical activity at the time of investigation have a longer PEP than nonathletes with similar activity level, however, did not observe changes of ejection fraction during detraining of runners. To our knowledge, no echocardiographic longitudinal follow-up of high-level athletes who stopped their sports activities is available. It would be interesting to know whether these changes of LV end-systolic stress and of ventricular function after interrupting intensive training and competition are transient or permanent.

Acknowledgments

The authors gratefully acknowledge the collaboration of Y. Van Mol, M.D., sports medical attendant of the cyclists, the willingness of the athletes and the control subjects, the valuable advice of H. Ector, M.D., and the assistance of R. Dewaele, M.D., R. Nuyts, J. Delsupehe and J. Romont.

References

The Diagnosis of Acute, Recurrent, Deep-vein Thrombosis: A Diagnostic Challenge


SUMMARY Recurrent venous thrombosis presents a diagnostic challenge. Venography, impedance plethysmography and fibrinogen leg scanning all have potential limitations, and their role in this context has not been evaluated. We performed a prospective cohort study evaluating impedance plethysmography and leg scanning, plus venography, using outcome on long-term follow-up as the end point in 270 patients with clinically suspected recent deep-vein thrombosis. Anticoagulant treatment was withheld in the 181 patients negative by noninvasive testing and was given in patients positive by impedance plethysmography if leg scanning was positive or if intraluminal filling defects were detected by venography. The validity of this approach was tested by long-term follow-up. Three of 181 patients (1.7%) negative by noninvasive testing had a recurrence, compared with 18 of 89 (20%) with positive findings (p < 0.001). Our objective diagnostic approach has high clinical utility; an objective rationale for withholding or giving treatment was established in 95% of patients.

THE PATIENT with clinically suspected recurrent deep-vein thrombosis presents a diagnostic challenge for the clinician because the accuracy of currently accepted tests for deep-vein thrombosis has not been formally evaluated in this context. Furthermore, although the clinical diagnosis of recurrent deep-vein thrombosis is thought by many to be nonspecific, this has not been formally documented. Based on the findings of extensive prospective clinical trials of objective testing, combined impedance plethysmography and leg scanning plus venography are now recognized as accurate, clinically useful tests 14 in patients with their first episode of clinically suspected venous thrombosis. The role of these tests, however, has not been firmly established in patients with clinically suspected recurrent deep-vein thrombosis. Careful evaluation of these diagnostic tests is necessary because each objective test has potential limitations in diagnosing acute recurrence in the presence of previous venous disease.
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R Fagard, A Aubert, R Lysens, J Staessen, L Vanhees and A Amery

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