SCIENTIFIC LETTERS

Myocardial infarction rates are higher on weekends than on weekdays in middle aged French men

Several studies have shown that the incidence of myocardial infarction (MI) does not follow a continuous pattern throughout the week, and that there is an increase in mortality on Mondays and possibly a decrease on Thursdays.[1] The reasons for such a pattern are not well understood, and several hypothesis have been made, namely a change from leisurely weekend activities to stressful work among working patients[1] or a Monday increase in the incidence of life threatening ventricular arrhythmias.[2] To our knowledge, no assessment of the weekly distribution of MI mortality has been performed in France. Thus, we used the data from the French national mortality statistics and from the French MONICA (monitoring trends and determinants in coronary disease) registers to assess the weekly pattern of incidence and mortality from MI.

Mortality data for coronary heart disease (ICD codes 410–414) was obtained from the French National Mortality Statistics (IN-SERM SC8, Le Vésinet, France) for years 1987 to 1997. The data were provided as number of deaths according to sex, year, 10 year age group, and day of the week. The corresponding population numbers were obtained from the French National Institute of Statistics and Economical Studies (INSEE, Paris, France). MI events were also collected from the three French MONICA registers (Lille, Strasbourg, and Toulouse) for both sexes, aged 35–64 years. Statistical analysis was performed using Epi-Info (CDC, Atlanta, USA) and SAS (SAS Institute, Cary, North Carolina, USA) statistical software. National mortality data were converted to mortality rates, which were then compared by Wilcoxon test. Data from the MONICA registers was analysed by χ^2. A further comparison between weekends and workdays was also performed. Significance was considered for $p < 0.05$.

The data from the French National Mortality Statistics contained over 17 000 coronary deaths. Average mortality rates for coronary heart disease by day of the week and for the period 1987 to 1997 are indicated in table 1. In men, significant differences were found for age groups 25–34, 35–44, and 45–54 years, higher rates occurring on Sundays and Saturdays, whereas no differences were found for older men (although with a tendency towards higher rates on Mondays) and for women (not shown). Interestingly, in men, the mortality pattern shifted from a higher mortality on weekends among young subjects to a higher mortality on Mondays among older subjects (comparison of daily number of deaths between 25–54 and 55–84 years: $\chi^2 = 11.64$, $p < 0.07$). Pooled data from the three French MONICA showed that, in men, MI events occurred more frequently on weekends and on Wednesdays, and that this difference was mainly caused by first time (incident) events, whereas no between-day difference was found for recurrent events.

Comparison between weekends and workdays showed incident cases to be more frequent on weekends ($\chi^2 = 7.75$, $p < 0.01$). For women, the number of events was too low to draw any valid conclusion.

Several limitations should be acknowledged regarding data from national mortality statistics. Although they are complete and represent all deaths which occurred in France, their accuracy is lower than mortality data derived from MI registers such as MONICA. Also, they do not provide information regarding survivors of MI. Nevertheless, those misclassifications, which are inherent in national mortality statistics, would tend to reduce differences between days of the week and thus the real differences might be even greater.

The effect of age on the weekly distribution of MI mortality has seldom been studied. The higher frequency of deaths on weekends found in this study is in agreement with some studies,[3] but not with others.[1] Although no clear-cut explanation can be provided, it is possible that young adults engage in strenuous activity during weekends, thus increasing the risk of MI.[4] This hypothesis is supported by the increase in incident (first time) MI cases on weekends than on workdays observed using MONICA data. Further, a possible explanation for the higher mortality rates on Mondays could be caused by a reporting bias, old subjects with a coronary episode on Sundays delaying their hospital admission to Mondays, thus increasing the number of events for that day. Nevertheless, since the MONICA data includes the precise timing of the MI event, such a hypothesis is unlikely.

We conclude that, for French young men, MI incidence and mortality is higher on weekends than on workdays, whereas for older subjects a trend towards higher MI mortality on Mondays is found. The reasons for such a discrepancy await further investigation

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Table 1 *Average mortality rates for coronary heart disease for period 1987 to 1997, by day of the week, in men*

Age group	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday	Test
25–34	3.1	2.3	2.9	2.7	2.6	3.5	4.1	*
35–44	23.1	20.6	22.4	21.0	22.4	24.5	27.7	***
45–54	71.8	65.3	63.4	66.6	67.1	71.3	75.2	*
55–64	196.9	188.0	183.5	186.5	187.5	190.7	194.2	NS
65–74	528.8	513.4	509.9	511.8	508.5	498.2	492.2	NS
75–84	1297.9	1269.8	1252.5	1275.6	1271.9	1256.4	1247.3	NS

Results are expressed as average rates for 1000000 inhabitants. Analysis by Wilcoxon test separately for each age group: *$p < 0.05$; ***$p < 0.001$; NS, not significant.

Myocardial integrated ultrasound backscatter in patients with Duchenne's progressive muscular dystrophy

Duchenne's progressive muscular dystrophy (DMD) is a genetic muscular disorder that causes degeneration and atrophy of skeletal and cardiac muscle. Histologic changes in the heart of patients with DMD include fibrosis, degeneration, and fatty infiltration. Fibrosis begins in the outer half of the left ventricular posterior wall, which is a relatively specific finding for DMD.[1]

Ultrasonography can be used to characterise changes in the myocardium at the cellular level. Unprocessed radiofrequency signals, redirected back to the transducer, provide quantitative information about intramural architecture in terms of ultrasonic integrated backscatter (IBS). Two of the IBS parameters can be measured: the magnitude of cyclic variation (CV) and IBS intensity. Changes in the CV are caused by variations in myocardial collagen, water content, myofibril orientation, and myocardial contractility. Myocardial IBS intensity is correlated with the collagen content or the degree of the fibrosis in the myocardium.[2 3] In this study, we investigated whether myocardial ultrasound IBS is useful for the early detection of myocardial involvement in patients with DMD.

Twenty five patients with DMD were enrolled in this study. The mean patient age was 17.6 (2.7) years. Fourteen healthy individuals were included as an age matched control group. A previously described protocol was used for the measurement of myocardial IBS.[4] In the parasternal long axis view of the left ventricle, an elliptical shaped region of interest was positioned in the inner and outer halves of the left ventricular posterior wall, respectively. The CV of the inner and outer halves of the left ventricular posterior wall were calculated as the difference between the end diastolic (peak) and end systolic (nadir) IBS values (CVin and CVout, respectively). The difference in the CV (dif CV) was calculated by subtracting the CVout from the CVin. Mean values for the IBS intensity in the inner and outer halves of the left ventricular myocardium were automatically displayed (IBSin and IBSout, respectively). The corrected mean myocardial IBS values for the inner and outer halves of the left ventricular posterior wall (cIBSin and cIBSout, respectively) were measured by subtracting

Table 1 Echocardiographic and integrated backscatter variables

	Control group (n=14)	*Patients with DMD (n=25)*	*p Value*
Age (years)	16.1 (4.8)	17.6 (2.7)	NS
Weight (kg)	49.5 (9.4)	35.8 (11.6)	< 0.001
Heart rate (bpm)	68 (11)	86 (13)	< 0.001
LVd (mm)	43.8 (6.9)	48.9 (9.4)	< 0.005
SF (%)	35.6 (3.8)	22.8 (9.7)	< 0.001
IBS parameters			
CVin (dB)	9.6 (2.4)	6.1 (2.1)	< 0.001
CVout (dB)	8.4 (1.9)	4.5 (2.1)	< 0.001
dif CV (dB)	1.3 (1.3)	1.5 (1.4)	NS
cIBSin (dB)	11.4 (3.5)	15.3 (4.6)	< 0.01
cIBSout (dB)	14.3 (4.3)	25.2 (6.3)	< 0.001
dif IBS (dB)	2.9 (1.8)	9.9 (5.0)	< 0.001

Data presented are mean value (SD). LVd=left ventricular internal dimension at end diastole; SF=shortening fraction of the left ventricle; CVin=cyclic variation for the inner half of the left ventricular posterior wall; CVout=cyclic variation for the outer half of the left ventricular posterior wall; dif CV (difference between CVin and CVout)=CVin−CVout; cIBSin=mean value of IBS for the inner half of the left ventricular posterior wall − mean value of IBS for the left ventricular cavity; cIBSout=mean value of IBS for the outer half of the left ventricular posterior wall − mean value of IBS for the left ventricular cavity; dif IBS (difference between IBSout and IBSin) = cIBSout − cIBSin

the mean IBS value for the left ventricular cavity near the left ventricular posterior wall from the IBSin and IBSout. The difference in the IBS intensity (dif IBS) was also calculated by subtracting the cIBSin from the cIBSout.

Comparison of the IBS parameters between patients with DMD and the control group is summarised in table 1. In the control group, CVin was significantly higher than CVout (9.6 (2.4) dB *v* 8.4 (1.9) dB, p < 0.01). The intensity of cIBSin was significantly lower than that of cIBSout (11.4 (3.5) dB *v* 14.3 (4.3) dB, p < 0.001).

Both CVin and CVout were significantly lower in patients with DMD than in the control group (6.1 (2.1) dB *v* 9.6 (2.4) dB for CVin, 4.5 (2.1) dB *v* 8.4 (1.9) dB for CVout, p < 0.001 for both). The intensities of cIBSin and cIBSout were significantly greater in patients with DMD than in the control group (15.3 (4.6) dB *v* 11.4 (3.5) dB for cIBSin, p < 0.01: 25.2 (6.3) dB *v* 14.3 (4.3) dB for cIBSout, p < 0.001). The dif IBS was also significantly greater in patients with DMD than in the control group (9.9 (5.0) dB *v* 2.9 (1.8) dB, p < 0.001).

When the normal range for the shortening fraction of the left ventricle was defined as the mean (2 SD), nine patients with DMD had a normal shortening fraction (28%). Both CVin and CVout were significantly lower in patients with DMD with normal shortening fraction than in the control group (7.6 (1.0) dB *v* 9.6 (2.4) dB for CVin, p < 0.05:

5.4 (1.4) dB *v* 8.4 (1.9) dB for CVout, p < 0.001). There was no significant difference in cIBSin intensity between the two groups. In contrast, cIBSout was significantly higher in patients with DMD than in the control group (22.8 (7.3) dB *v* 14.3 (4.2) dB, p < 0.005). The dif IBS was also significantly greater in patients with DMD than in the control group (8.9 (5.3) dB *v* 2.9 (1.8) dB, p < 0.001). These data indicate that IBS intensity was increased and the magnitude of CV was decreased in the outer half of the left ventricular posterior wall in patients with DMD, even in the setting of normal global cardiac function (shortening fraction). When the mean (2 SD) in the control group is considered the upper limit of normal for dif IBS, 6.5 dB> dif IBS is abnormally high. Among the nine DMD patients with a normal left ventricular shortening fraction, six patients had an increase in dif IBS.

Fibrotic changes do not always occur homogeneously throughout the ventricular wall in myocardial diseases. In the setting of idiopathic dilated cardiomyopathy, the endocardial half of the myocardium generally has more severe fibrosis. In contrast, cardiomyopathy caused by DMD is unique in that fibrosis begins in the outer half of the myocardium.[1] Thereafter, small areas of fibrosis may be identified in the ventricular septum, near the right ventricular cavity. Eventually, there is a diffuse transmural fibrosis. The echo amplitude of the outer half

of the left ventricular posterior wall is often increased on conventional two dimensional echocardiography in patients with DMD.[5] We were able to evaluate quantitatively the characteristics of the myocardium in patients with DMD by analysing myocardial IBS.

Suwa and colleagues reported that patients with dilated cardiomyopathy who responded to β blocker treatment had a lower IBS intensity of the left ventricular posterior wall compared to non-responders.[2] They concluded that left ventricular myocardial IBS intensity provides useful information for predicting the degree of myocardial fibrosis and the response to β blocker treatment in patients with dilated cardiomyopathy. We believe that measurements of IBS variables, especially the dif IBS, are useful for the early detection of the myocardial involvement in patients with DMD, even if conventional echocardiographic findings (such as shortening fraction) show normal values. Early detection of myocardial changes in patients with DMD might be important, because earlier treatment might be more effective in preventing the development of myocardial fibrosis.

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