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Influence of intensive coronary care acoustics on the quality of care and physiological state of patients

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Abstract

Aim of the study: To evaluate the possible role of room acoustics on patients with coronary artery disease and to test the hypothesis that a poor acoustics environment is likely to produce a bad work environment resulting in unwanted sound that could adversely affect the patients. *Methods and results:* A total of 94 patients admitted to the intensive coronary heart unit at Huddinge University Hospital for evaluation of chest pain were included in the study. Patient groups were recruited during bad and good acoustics, respectively. Acoustics were altered during the study period by changing the ceiling tiles throughout the CCU from sound-reflecting tiles (bad acoustics) to sound-absorbing tiles (good acoustics) of similar appearance. Patients were monitored with regard to blood pressure including pulse amplitude, heart rate and heart rate variability. The patients were asked to fill in a questionnaire about the quality of the care, and a follow-up of rehospitalization and mortality was made at 1 and 3 months, respectively. There were significant differences between good and bad acoustics with regard to pulse amplitude in the acute myocardial infarction and unstable angina pectoris groups, with lower values during the good acoustics period during the night. The incidence of rehospitalization was higher for the bad acoustics group. Patients treated during the good acoustics period considered the staff attitude to be much better than during the bad acoustics period. *Conclusion:* A bad acoustics environment during acute illness may have important detrimental physiological effects on rehabilitation.

Keywords: Myocardial infarction; Room acoustics; Reverberation time; Noise; Psychological stress; Pulse amplitude

1. Introduction

Patients with acute chest pain who are evaluated in the intensive coronary heart unit (CCU) are in general in a stressful situation. Apart from their medical condition with an activated sympathetic system, they may also be affected by several environmental conditions such as unexpected noise, long recognised to have a negative influence in the rehabilitation of patients [1]. The present study, which was performed in a university hospital, examined the possible role of poor vs. good room acoustics on the patients' cardiovascular condition. The hypothesis was that a poor sound absorption condition is likely to produce a bad work environment resulting in sounds that could affect the patients. It was expected that an environmental condition of longer reverberation time (poor room acoustics) would increase sound propagation and sound levels in the CCU, reduce speech intelligibility and thereby contribute to the generation of work-related noise, adversely affecting the patients.

In Huddinge University Hospital, patients are referred from a catchment area of 375,000 inhabitants. The Department of Cardiology serves as a referral clinic for two other hospitals for patients who need angioplasty, invasive electrophysiology and thoracic surgery. The comparatively small number of beds consequently enhances the turnover of patients in the unit. Six to seven patients with acute symptoms are daily admitted to the unit and the average observation time is 17 h. All patients are monitored with a computerised system for ECG monitoring and/or hemodynamics with different automatic alarms for critical values. Patients are, when needed, transported to the laboratories for

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further investigations, and laboratory testing is partly performed in a point-of-care setting in a corner of the central area. This and other logistics, such as regular cleaning of the patient rooms, exchange of beds and laundry, etc, gives the unit a noisy and somewhat turbulent atmosphere. Nearly 50 nurses are employed at the unit scheduled either for morning, evening or night shifts.

During the study period, the acoustic environment was changed in the patient rooms and the main work area of the unit where the staff makes most decisions and monitoring of the patients.

Physiological outcome parameters were heart rate, heart rate variability, systolic and diastolic blood pressure together with pulse amplitude which is the difference between systolic and diastolic blood pressure. The influence of the autonomic nervous system at the sinus node results in a beat-to-beat variation of heart rate which can be described as heart rate variability (HRV) [2]. Emotional and physical stress decreases HRV, and low HRV is an independent predictor of arrhythmic complications and death in patients with ischemic heart disease. These physiological parameters, previously shown to be sensitive to psychological arousal [2,3,4,5], were analysed in two patient populations during periods of bad and good acoustics.

The patients were also asked to fill in a questionnaire with a few questions about the quality of the care.

2. Methods

Data for this project were collected during regular weekdays and corresponding nights, but not during weekends since there were changes in staffing and other conditions during such periods. During the study period, the acoustics were changed in the patient rooms and central part of the unit where the staff makes most decisions and monitoring of the patients. The physical environment was manipulated in two steps. Firstly, remodelling of the ceiling took place changing the original tiles to sound-reflecting plaster tiles (13-mm solid painted plaster board tiles). One week before the final measurements, sound-absorbing tiles (40-mm Ecophon® ceiling tile) of nearly identical appearance replaced the plaster tiles. The sound-absorbing properties of the ceiling corresponded to class A (Acoustics-Sound absorbers for use in buildings) [6]. Both assessment periods (good and bad acoustics) lasted for 4 weeks.

Table 1
Clinical characteristics for study groups during bad and good acoustics

	Bad acoustics	Good acoustics	*p*
Number of patients	31	63	
Female	11 (33%)	26 (37%)	ns
Age	69 ± 11	66 ± 11	ns
Age > 60 years	21 (64%)	47 (75%)	ns
Beta-blockers	28 (90%)	52 (83%)	ns
Extra intravenous beta-blockers	10 (30%)	6 (10%)	< 0.01
Analgesics, sedatives	26 (84%)	44 (70%)	ns
Hospitalization time (days)	7 ± 4	5 ± 4	ns
Rehospitalization, 1 month	6 (18%)	6 (10%)	ns
Rehospitalization, 3 month	15 (48%)	13 (21%)	< 0.01
Subgroups of diagnosis			
Stable angina pectoris	11 (35%)	33 (52%)	ns
Unstable angina pectoris	4 (13%)	7 (11%)	ns
Acute myocardial infarction	16 (52%)	23 (37%)	ns
Out of whom			
Female	6 (37%)	7 (31%)	ns
Age (mean ± S.D.)	71 ± 11	68 ± 7	ns
Age > 60 years	12 (75%)	23 (100%)	< 0.05
Hospitalization time (days)	9 ± 5	7 ± 3	ns

Values are expressed as numbers, percent or mean ± S.D.

Table 2
Use of peroral beta-blockers in diagnostic subgroups during bad and good acoustics

	Bad acoustics	Good acoustics
AMI	15/16 (94%)	23/23 (100%)
Unstable angina	3/4 (75%)	4/7 (57%)
Stable angina	10/11 (91%)	25/33 (76%)

Values are expressed as numbers and percent.

Measurements of changes in acoustics showed that there was a drop in sound level of 5–6 dB in the two patient rooms that were subjected to measurements. In the main work area, however, the equivalent sound pressure level did not change considerably (57 dB (A) in bad acoustics vs. 56 dB (A) in good acoustics). After the application of the sound-reflecting plaster tiles, reverberation time [7] was reduced from 0.8 to 0.4 s in the main work area and from 0.9 to 0.4 s in the patient rooms. Speech intelligibility improved considerably both in the main work area and in the patient rooms, as indicated both by the RASTI method [8] and verbal reports from staff. Altogether, there was accordingly a considerable improvement of the acoustic environment. A separate study showed that there were marked effects on the perceived psychosocial work environment. Accordingly, the staff felt fewer demands and less irritation during the good acoustics period (Blomkvist et al., to be published).

A total of 94 patients were, after written informed consent, included in the study. Clinical characteristics for both study groups are described in Tables 1 and 2. The patient groups were analysed with regard to blood pressure date including pulse amplitude, heart rate and heart rate variability as a whole and in subgroups according to final diagnosis of stable angina pectoris, unstable angina pectoris and acute myocardial infarction. Blood pressures were assessed in the supine position by means of an automatic device (MIDA™, Ortivus Medical, Sweden).

The study was approved by the local research ethics committee at the Huddinge University Hospital.

Heart rate variability was analysed in the time domain, using SDNN (standard deviation of the NN intervals) and TINN (triangular index, in which the variability is calculated from the frequency distribution of all NN intervals). These measures are stable and insensitive to extra systoles

Table 3
Physiological parameters in the study groups of bad and good acoustics

	Bad acoustics	Good acoustics	*p*
Heart rate (beats/min)	68 ± 14	67 ± 14	ns
Heart rate variability			
NN (ms)	913 ± 183	920 ± 181	ns
SDNN (ms)	97 ± 45	95 ± 37	ns
TINN (ms)	345 ± 112	344 ± 155	ns
Systolic blood pressure, day	134 ± 20	134 ± 19	ns
Systolic blood pressure, night	139 ± 26	131 ± 22	ns
Diastolic blood pressure, day	76 ± 11	77 ± 10	ns
Diastolic blood pressure, night	77 ± 12	75 ± 10	ns
Pulse amplitude, day	59 ± 16	57 ± 15	ns
Pulse amplitude, night	54 ± 22	53 ± 21	ns

Values are expressed as mean ± S.D.; ns = nonsignificant.

and artefacts [2]. They estimate overall heart rate variability from long-time telemetric recordings. Patients with pacemaker rhythm or atrial fibrillation ($n = 2$) were excluded from analysis. Analysed time (hours) was 17.11 ± 4.77 and 14.17 ± 5.09 in bad and good acoustics, respectively.

Short time prognosis was evaluated as rehospitalization and mortality within 1 and 3 months.

The patients were also asked to fill in a questionnaire with a few questions about the quality of the care. Visual analogue scales were used for the ratings with scores ranging from 0 to 10. Six separate patients assessed the overall quality of care in the ward, staff attitude quality, wake-ups due to sounds, intelligibility of the staff's statements, sounds from the corridor and disturbances due to sounds.

3. Statistics

Data are presented as mean ± S.D. Differences between the two groups were tested by two-tailed *t*-test for unpaired samples. For nonparametric variables, the significance of differences was tested by means of Mann–Whitney *U*-tests. Differences in frequencies was tested with chi-square analysis. A *p*-value < 0.05 was considered significant.

4. Results

Table 3 shows the physiological parameters in the total groups of bad and good acoustics. There were no significant differences between the groups with regard to heart rate or its variability, blood pressure or pulse amplitude. These results remained unchanged when analysed in subgroups of different age and gender.

When the groups were subdivided according to degree of disease, there were significant differences between the good and the bad acoustics period with regard to pulse amplitude in the acute myocardial infarction and unstable angina pectoris groups. The lower values were found in the good acoustics period during the night, as described in Table 4. For the other studied variables, there were no significant differences between the myocardial infarction patients in the two acoustics groups.

There was a higher incidence of rehospitalization at both 1 and 3 months in the group of bad acoustics compared to that in good acoustics. This difference was statistically significant at 3 months ($p < 0.01$). Early mortality, which was very low in both groups, did not differ between the acoustics periods.

Table 5 shows the results of the comparisons between the two conditions with regard to patient ratings. The findings for the total group and the myocardial infarction patients are very similar. Separate analyses were not performed for the unstable angina pectoris group because these subgroups were very small. The most striking finding is that patients in the good acoustics period considered the staff attitude to be much better than during the bad acoustics period. There was also a tendency for the patients to overhear sounds from the corridor and to wake up due to disturbing sounds more often during the bad acoustics period.

Table 4
Pulse amplitude (mm Hg) during bad and good acoustics

	Bad acoustics	Good acoustics	*p*
Stable angina pectoris	57 ± 15	63 ± 18	ns
Unstable angina pectoris	78 ± 9	59 ± 12	0.03
Acute myocardial infarction	62 ± 19	49 ± 17	0.04

Values are expressed as mean ± S.D. A *p*-value < 0.05 was considered significant.

Table 5
Mann–Whitney *U*-tests comparing bad with good acoustics for total groups (T) and myocardial infarction groups (AMI)

	z(T)	*p*(T)	*z*(AMI)	*p*(AMI)
Health care in general	− 2.00	0.046	− 1.93	0.054
Staff attitude quality	− 2.90	0.004	− 2.62	0.009
Waking due to sounds	− 1.98	0.048	− 1.92	0.054
Can hear what staff say	− 0.01	0.99	− 0.87	0.39
Sounds from corridor	− 2.03	0.04	1.86	0.06
Disturbed by sounds	− 1.58	0.12	− 0.47	0.64

Minus indicates that the score is better in the good acoustics period.

5. Discussion

The main finding in the present study is that in the myocardial infarction patients, there is a statistical association between acoustics and patient pulse amplitude during the night in the intensive coronary care—with higher pulse amplitude in worse acoustics. This finding seems to be associated with a more serious degree of disease. In the literature, pulse amplitude has only been used to a limited extent as a biological stress marker [9]. The differences in pulse amplitude during bad and good acoustic may influence stroke volume, and if so, this may in part be due to an increased activity of the beta-2 receptors of the sympathetic nervous system during the period of bad acoustics [10]. It is reasonable to think that a bad atmosphere resulting in raised voices will have a greater influence on patients if the room acoustic conditions are poor (long reverberation time). This may also be more obvious during the night period when the normal daily background noise is low and a sudden increase in noise can be even more stressful. The average observation time for all patients in the CCU is 17 h, but effects of room acoustics on pulse amplitude come more or less momentarily. Therefore, differences in time spent in the unit or total hospitalization time is not important for the results in this study.

The use of peroral beta-blockers was equal in the two groups, but there was a significantly higher frequency for the need of extra intravenous beta-blockers in the group during bad acoustics ($p<0.01$). Medication with beta-blockers can be considered to be a potentially important confounder. Nearly all the patients were on peroral beta-blockers, and extra intravenous treatment was given, in this clinical setting mostly on the indication of pain This may explain why there was no difference in heart rate or in heart rate variability between the two groups. It has previously been shown that the use of beta-blockers can increase heart rate variability through the effects over the sinus node [11]. The difference in pulse amplitude was observed despite the beta blockade in both groups. This may indicate that the effect of acoustics on pulse amplitude is due to a non-chronotropic mechanism.

It may seem surprising that there were no significant findings for pulse amplitude during the day. One possible explanation could be that the patients woke up due to noise less frequently in the night during the good acoustics period with less arousal effects on blood pressure. This is in line with the findings of Berg [12], who showed that increased sound absorption contributed to a better acoustic environment by reducing sound-induced sleep fragmentation.

Since the patients represent an urban population in Sweden, they were relatively old. It could be argued that the sound environment is unimportant for such patients. However, the proportion of patients with hearing aids was similar in the two groups. In addition, it could be argued that some hearing devices are very sensitive to loud sounds. Hence, this group of patients may be even more sensitive to this kind of external influence than other patients.

The two patient groups in the study are not randomized. During the good acoustic period, all patient rooms and the central part of the ward were modified, and a randomized inclusion could therefore not be made. However, as can be seen in Table 1, the study groups did not differ in basal clinical characteristics or subgroups of diagnosis. Still, there was a significant difference in pulse amplitude between groups of bad and good acoustics together with a significantly greater need for extra intravenous beta-blockers during bad acoustics. These observations together indicate that a poor acoustic environment during acute illness may have important physiological consequences. As rehospitalization rate at 3 months was significantly higher in the group of bad acoustics, it could not be excluded that this also may have a negative impact on the rehabilitation period.

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