PAPERS AND ORIGINALS

Perceived noise in surgical wards and an intensive care area: an objective analysis

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Summary

An investigation of noise levels in a hospital ward, a cubicle off the ward, and an intensive therapy unit (ITU) showed that the noise levels in all three areas were higher than internationally recommended levels at all times of day. Loud noises above 70 dB(A) were common in all areas but especially the ITU. The noise pollution levels reached annoying values during the day in the ward and cubicle and during both the day and the night in the ITU. Equipment and conversations among the staff were the main causes of noise in the ITU.

These noisy environments are unlikely to help patients recover. Although measures designed to eliminate noisy surfaces will help, making staff aware of the noise they create and the effects it has may be much more effective in reducing noise pollution.

Introduction

From time to time clinicians, patients, and others have directed attention to problems of noise in hospital.¹⁻⁵ Studies by questionnaire in this hospital have shown that noise is often reported by patients as a problem that adds to their discomfort.⁶ We undertook an investigation to measure noise levels and so to assess whether there was an objective basis for complaint. The study was also designed to find the source of noise in a typical open general surgical ward and a mixed intensive care unit.

Background

Noise "pollution" in industry, at the roadside, and near airports has long been the object of study and legislation.⁷⁻⁹ Measurement is a complex subject, as recent experience with Concorde suggests. Though acoustic noise can be measured in absolute terms by the force per unit area that a sound wave produces, the use of derived scale (decibel; dB) of loudness rather than such a direct measure is necessary because of the very great dynamic range of the human ear.

In the Appendix we describe the origin of the decibel (dB) scale and the derivation of those other scales commonly used to assess noise.

FIG 1—Mean noise levels throughout 24 hours for three areas studied over five days.

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FIG 2—Mean curve ± 2 SD for each area studied.

We have chosen from among these the dB(A) scale because of its close relation to the curve that describes loudness and frequency for the human ear.¹⁰ We have also measured level of noise pollution⁸ (defined in the Appendix) to give some idea of the envelope of sound to which the hospital patient is exposed. In assessing the results we have obtained, it is sufficient to remember that a rise of 10 dB(A) implies a doubling of perceived noise intensity.

Methods

Noise levels were measured on the dB(A) scale using a Brüel and Kjaer piezoelectric microphone type 4117, which was positioned on the wall 0.5 m above a patient's head, a preamplifier and measuring amplifier type 2609, and a Devices mx4 pen recorder. The system was calibrated on the Brüel and Kjaer type 4230 sound level calibrator, which produces a 14-kHz tone of 84 dB. Because the weighting networks are the same at 1 kHz this calibrator may be used for all of them. The measuring amplifier had been adapted to give a logarithmic response so that the chart recorder could be calibrated linearly to a 10 dB/cm deflection. It was sited up to 20 m from the microphone and

measuring amplifier. Chart recordings were initially analysed in half-hour epochs to find the mean and its standard deviation of dB(A), level of noise pollution, and the number of discreet noises over 70 dB(A). Measurements were made during five 24-hour weekday periods at each site—an open Florence Nightingale ward, a cubicle in a cul-de-sac of this ward, and a general intensive care unit (ITU). Because visual inspection of the noise pattern produced showed a striking similarity for all periods, we pooled the data to give a profile for the area based on all the observations. Measurements are therefore shown as mean and standard deviations per half-hour epoch for five days.

FIG 3—Noise pollution levels for three areas.

FIG 4—Number of noises greater than 70 dB(A) per half hour.

Results

Fig 1 shows the mean level for each area and fig 2 the separate distribution profiles. During the daytime the average level was about 55 dB(A) in the ward, 50 dB(A) in the cubicle, and 53 dB(A) in the ITU. During the quietest part of the night, at 0400, the ward level was 35.5 dB(A), the cubicle level 31.5 dB(A), and the ITU level 42.5 dB(A). Noise pollution levels over the same period are shown in fig 3. Levels in both the ward and the cubicle in the daytime were about 65 dB(A), and these fell at night to 45 dB(A) and 38 dB(A), respectively. In the ITU from 0800 until 1600 hours average noise pollution was 72 dB(A), and between 1600 and 2400 it was 65 dB(A). From 2400 to 0600 pollution dropped slightly to 62 dB(A). Finally, fig 4 shows the number of loud noises above 70 dB(A) per half-hour; these generally followed the pattern of the noise pollution recording. From 0600 to 2400 there was a mean time between loud (greater than 70 dB(A)) noises of 3, 4, and 1.5 minutes in the cubicle, ward, and ITU respectively. At night loud noises were absent in the ward but occurred every nine minutes in the ITU. In all areas high levels at 0800 to 0900 were caused by vacuum cleaners.

Discussion

The International Proposal for Noise Assessment with Respect to Community Response suggests the following basic noise limits: daytime 45 dB(A), evening 40 dB(A), night 30 dB(A). In conformity with these, typical results for a living room in daytime are 40-50 dB(A) and for a domestic bedroom at night 20-30 dB(A). By contrast a London street corner averages 70-80 dB(A). To help the reader appreciate the meaning of these figures fig 5 shows the dB(A) scale with its familiar sources and the response they may invoke.

FIG 5—Causes of and response to noise with position of hospital areas superimposed.

Our results show that both the ward and the ITU were never as quiet as a bedroom at night and that throughout the 24 hours the ITU was as noisy or noisier than a living room. Loud noises above 70 dB(A) were common in both areas, and the pollution index was therefore also high—again particularly in the ITU, where sudden very loud surges took place against a comparatively low background.

From our observations during these studies it was possible to define with some confidence the major sources and pattern of noise. In the wards lights out at 2230 and lights on at 0630 were sharp cut-off and onset points. At night nurses' footsteps (on a sealed wood floor), coughing, snores and cries (60-70 dB(A)), and exogenous noises from sluices and from passing

traffic (50-55 dB(A)) were the major factors. By contrast, noisy equipment (particularly respirators) and conversation by the staff were most important in the ITU.

On the face of it, we must accept that, particularly in the ITU, this amount of noise is unlikely to be helpful to the patient's recovery. We have evidence that nearly all patients who enter hospital for surgery soon accumulate a considerable sleep deficit, which must be partly caused by the noisy environment.⁶ But to know objectively that a place is too noisy, as we now do for our wards and ITU, is not necessarily to have solutions to the problem. Much can undoubtedly be done by paying attention to physical details such as floor coverings, ceilings, and baffles.^{11,12} Nevertheless, the peaks of noise that contribute to the noise pollution index are essentially made by men and women. Particularly in the ITU, loud conversations tend to develop unchecked and noisy procedures seem to be regarded as a fact of life. We recommend that from time to time sound engineers should use the simple methods that we have used in all ITUs to show the staff how much they pollute the environment. Furthermore, the staff should be educated against noise pollution as part of good hospital procedure, especially those working in intensive care units.

We do not want to insist on absolute quiet, which may well be strange to patients who are used to a noisy background in their lives and who, when faced with stillness, are not at ease. We desire only to emphasise that the matter is worthy of continuous attention by all those engaged in the care of patients.

Appendix

Acoustic noise can be measured in absolute terms by the force per unit area that the sound wave produces. The smallest noise perceivable by half of the population of young adults is about 20 μ Pa-100 Pa. The measured sound pressure can be related to this base level by the expression:

$$10 \log_{10} \left(\frac{p^2}{p_{\text{ref}}^2} \right) = 20 \log_{10} \left(\frac{p}{p_{\text{ref}}} \right)$$

This is the decibel (dB) scale and is arithmetically convenient because for every doubling of perceived loudness there is an increase of 10 dB.

Such a scale of noise measurement suffers the disadvantage that the human ear has a limited range of frequency response, while the microphone is normally designed with a response that is the same over an extremely large frequency range. Also, the effect of noise is related to its interpretation by the brain; high frequencies (7.5-10 kHz) are therefore more annoying than lower ones and intermittent sounds more irritating than continuous ones, particularly if they cannot be localised and thereby masked out by the brain. Thus, to make noise measurements more meaningful in terms of annoyance, sound measurement equipment usually incorporates certain internationally agreed frequency weighted filters designated A, B, C, or D.⁹ To distinguish between noise measurements made with the different weightings and those made with instruments having a flat frequency response a suffix is used—for example, dB(A) for weighting scale A. dB(D) is used largely for the measurement of aircraft noise; dB(C) is flat over the human audible spectrum (20 Hz-20 kHz); dB(B) is slightly weighted against low frequencies; and dB(A) is heavily weighted not to respond to frequencies below 1 kHz. The last scale follows most closely the normal curve that relates loudness to frequency for the human ear and consequently gives figures that correspond most closely to subjective impressions of the loudness of acoustic noises. For this reason it is the weighting scale that is commonly used in assessing noise problems.

Noise quoted in terms of mean dB(A) gives no indication of the variability of the level. But the level of noise pollution (LNP) is an index that includes a factor for variability: $LNP = L \text{ mean} + 2.56 \text{ SD}$. The 2.56 constant has been found

empirically to give the "best fit" when compared with subjective assessment of noise annoyance.⁸⁻¹⁰

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References

- ¹ Falk, S A, and Woods, N F, *New England Journal of Medicine*, 1973, **289**, 774.
- ² Woods, N F, and Falk, S A, *Nursing Research*, 1974, **23**, 144.
- ³ Ragan, C A, *Medical Times*, 1974, **102**, 17.
- ⁴ *British Medical Journal*, 1973, **4**, 625.
- ⁵ Whitfield, S, *Nursing Times*, 1975, **71**, 402.
- ⁶ Murphy, F, Bentley, S, and Dudley, H A F. In preparation.
- ⁷ Korsten, C W, and Vanos, G J, *Proceedings of the Conference on Control of Noise*. London, HMSO, 1962.
- ⁸ Robinson, D W, *The Concept of Noise Pollution Level*, NPL Aero Report Ac 38. London, National Physical Laboratory, 1969.
- ⁹ Robinson, D W, *An Outline Guide to Criteria for the Limitations of Urban Noise*. CP No 1112. London, HMSO, 1970.
- ¹⁰ Broch, J T, *The Application of the Brüel and Kjaer Measuring Systems to Acoustic Noise Measurements*, p 32. Naerum, Denmark, Brüel and Kjaer, 1971.
- ¹¹ Central Health Services Council. Standing Nursing Advisory Committee. *The Control of Noise in Hospital*. London, Ministry of Health, 1961.
- ¹² Ministry of Health, *Hospital Design*, note 4. London, HMSO, 1966.

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Cimetidine for duodenal ulceration in patients undergoing haemodialysis

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Summary

Peptic ulcer is a common problem in advanced renal failure, but most drugs for ulcers are hazardous in this condition. In a small open study cimetidine was given to nine patients with acid hypersecretion and endoscopically diagnosed duodenal ulceration who were undergoing haemodialysis. The patients obtained good pain relief and suffered no serious side effects. Both basal and stimulated acid output fell considerably and the plasma gastrin response to food increased during treatment. Two patients with recurrent vomiting during haemodialysis had a striking response to cimetidine, which suggested that such vomiting may be acid-mediated in some patients.

These preliminary results suggest that cimetidine may prove to be an advance in the management of peptic ulcer in uraemic patients.

Introduction

Peptic ulceration is unusually common in chronic renal failure,¹ and this poses a difficult problem, since many drugs for ulcer may have undesirable side effects in the uraemic patient. The H₂-receptor antagonist cimetidine is excreted mainly by the kidneys, but in patients on regular dialysis a reduced dose will produce adequate blood concentrations without the risk of accumulation.² Furthermore, an acid-lowering drug is a logical approach in the uraemic patient, as pyloroduodenal ulcer pre-

dominates in this condition, and gastric hyperacidity is strongly implicated.³ We present here our preliminary findings in patients undergoing renal dialysis who suffered peptic ulceration and were treated with cimetidine.

Patients and methods

Nine patients were treated with cimetidine. All had end-stage chronic renal failure (creatinine clearances <3 ml/min) requiring support by regular haemodialysis, and all had endoscopically proved duodenal or pyloric ulceration with gastric acid hypersecretion (peak acid output >45 mmol (mEq)/h for men, >30 mmol/h for women; or basal acid output >5 mmol/h).

Cimetidine was given for six weeks in a reduced dose of 400 mg/day on non-dialysis days and 800 mg/day on dialysis days, as a single haemodialysis is known completely to clear the drug from the blood. Maintenance treatment of 200 mg/day was continued thereafter.

ASSESSMENT

The symptomatic response was recorded at the end of six weeks, and repeat endoscopy carried out in those who had an ulcer crater before treatment. (Repeat endoscopy was not carried out in those who initially showed chronic scarring and deformity). All patients had two pentagastrin tests and two standard meal tests to assess, respectively, the effect of treatment on gastric acidity and plasma gastrin response to food. The tests were performed before treatment and again at the mid-point of the initial six-week course. A 200 mg dose of oral cimetidine was given one hour before the repeat test.

Gastric acidity was measured using the standard method of intramuscular pentagastrin stimulation (6 µg/kg body weight), and plasma gastrin was measured by radioimmunoassay.⁴ (Normal values for plasma gastrin in this laboratory are 0-150 ng/l.) The standard meal used consisted of 50 g carbohydrate, 18 g protein, and 20 g fat given as lean cooked ham, white crustless bread, butter, unsweetened orange juice, and a cup of tea with milk. The test was carried out after an overnight fast of 10 hours, and blood samples were withdrawn through an indwelling needle inserted into a forearm vein.

The following haematological and biochemical indices were measured on all patients before and after six weeks' treatment: total and differential white cell count, platelet count, and serum creatinine and transaminase concentrations.

The significances of differences were calculated using the paired *t* test.

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