

parameter estimates and significance tests, are often difficult for non-statisticians to interpret. For this reason, graphical methods should be employed, to present the data in such a way that highlights and summarises the features relevant to the relationships in question. It is rare, and usually unhelpful, to present the 'raw data' of the study, especially when a large quantity of data is involved.

The graphs of our paper were presented in this spirit. The year was defined from November to October for the simple reason that a flu epidemic in November and December can only be related to a spring excess of schizophrenic births in the next, but not in the same, calendar year. This problem is highlighted by Dr Crow's erroneous comment about Fig. 1(b) and the flu epidemic of 1957. In fact, the Asian flu epidemic peaked in Autumn of 1957, and is therefore of potential relevance to the risk of schizophrenia in individuals born in the early months of 1958. Careful inspection of our Fig. 1(b) will reveal that the early months of 1957 did indeed experience a low rate of influenza deaths, but this was not so far for the later months of 1957. Indeed, the A2 influenza epidemic of that autumn was the most widespread epidemic to have occurred in the UK in the post-war period. The main reason that more influenza-related deaths were not recorded was that a similar strain of influenza had been common in the early years of the century, so that many older people, who are most likely to die following influenza, were immune to that strain of the virus.

No *P* value was presented for Fig. 2(b) because the issue of statistical significance was dealt with by statistical modelling, not graphical methods.

Our paper addressed a single, clear hypothesis, and we believe that it proceeded to examine the hypothesis in a reasonable and statistically correct manner. We are content to await the results of other epidemiological and experimental studies examining the prenatal influenza hypothesis, to see whether or not they confirm our findings.

- BARR, C. E., MEDNICK, S. A. & MUNK-JORGENSEN, P. (1990) Exposure to influenza epidemics during gestation and adult schizophrenia: a 40-year study. *Archives of General Psychiatry*, **47**, 869–874.
- CROW, T. J. & DONE, D. J. (1986) Age at onset of schizophrenia in siblings: a test of the contagion hypothesis. *Psychiatry Research*, **18**, 107–117.
- KNIGHT, J. (1991) Schizophrenia and influenza. *Lancet*, **338**, 390.
- MEDNICK, S. A., MACHON, R. A., HUTTENEN, M. O., *et al* (1988) Adult schizophrenia following prenatal exposure to an influenza epidemic. *Archives of General Psychiatry*, **45**, 189–192.
- TORREY, E. F., RAWLINGS, R. & WALDMAN, I. N. (1988) Schizophrenic births and viral diseases in two states. *Schizophrenia Research*, **1**, 73–77.

P. C. SHAM  
N. TAKEI  
G. MURRAY  
R. M. MURRAY

*Department of Psychological Medicine  
Institute of Psychiatry & King's College Hospital  
De Crespigny Park  
London*

E. O'CALLAGHAN

*Cluain Mhuire Family Centre  
Blackrock  
Co. Dublin  
Republic of Ireland*

### Preconscious perceptual processing

SIR: Klemperer (*Journal*, September 1992, **161**, 420) suggests that a failure of preconscious processing underlies *all* perceptual experiences in which the subject makes a faulty interpretation of an external stimulus.

In my paper I alluded to the possibility that the model of a cycle of false belief and misperception reinforcing one another, which I use to explain delusional misidentification, may be relevant to the development of some delusional disorders (see section Seeing and believing in the delusional disorders). However, this model is not able to explain all perceptual distortions; for example, it cannot explain the unusually vivid visual experience of a hypomanic patient.

I agree with Dr Klemperer that better understanding of the interpretative aspects of preconscious processing of perceptions is essential for our understanding of the psychopathology of perceptions.

It is possible that such processes may also be relevant to the development of hallucinations, and help to explain the temporal coincidence of the development of delusions and hallucinations, which appear otherwise to be quite independent of one another, which is commonly observed in psychotic relapses. The reasoning processes that underlie normal belief formation (Alloy & Tabachnik, 1984) and those that are involved in the interpretative aspects of preconscious perceptual processing are similar; there is a trade-off between prior expectation and the quality of current situational information or sense data. A single reasoning fault might result in interpretations being uncoupled from the evidence on which they should be based, to produce delusions if the evidence is current situational information, or hallucinations if the evidence is sense data.

ALLOY, L. B. & TABACHNIK, N. (1984) Assessment of covariation by humans and animals: the joint influence of prior expectations and current situational information. *Psychological Review*, **91**, 112–149.

SIMON FLEMINGER

*Maudsley Hospital  
Denmark Hill  
London SE5 8AZ*

### Rapid tranquillisation

SIR: Pilowsky *et al* (*Journal*, June 1992, **160**, 831–835), in their survey of rapid tranquillisation, report that intravenous sedation with diazepam alone or in combination with haloperidol appeared to be more rapid and effective than other drugs given intramuscularly. Furthermore, staff expressed greatest satisfaction where a combination of an antipsychotic and a sedative were used. However, we are concerned about the dangers of intravenous injections in psychiatric settings. We note that one of the 60 patients in the study had a cardiorespiratory arrest, another collapsed with shallow respirations and a third had a transient tachycardia.

We would like to draw attention to the risks associated with emergency intravenous injections by describing two cases known to us. In the first, a general practitioner (GP) assessed an excited 23-year-old man threatening suicide after a furious row with his parents who objected to his bringing his girlfriend to the parental home at 3 a.m. He was given diazepam, 10 mg intravenously. As this apparently had no effect, the GP then gave chlorpromazine, 50 mg intravenously. The patient was brought into hospital unconscious, responding only to painful stimuli and with acute dyskinesias affecting his neck, trunk and limbs. It took two days for him to recover full consciousness. The ABPI data sheet compendium states that parenteral chlorpromazine can be administered only by intramuscular injection. Likewise, the British National Formulary (1991) and Gilman *et al* (1990) describe only intramuscular use. Martindale mentions that the injection can be given intravenously if it is diluted beforehand (Reynolds, 1989). How many doctors know this?

The second case involved a psychotic and disturbed young adult assessed at home by the GP and a psychiatrist. After intravenous injection of diazepam, 20 mg, and haloperidol, 20 mg, the patient had a fatal cardiac arrest. There was a history of drug therapy for asthma. Sometimes emergency treatment has to be started when the patient's mental state prevents the doctor taking an adequate history which would include details of past medical illness and recent drug (including illicit drug) use. Although these two cases

occurred in the community we do not believe that hospitals confer immunity from catastrophe.

Finally, we would like to point out that estimation of dose is based largely on guesswork. Dr Pilowsky *et al* used small bolus doses to avoid oversedation and undertreatment. However, we are concerned that with aggressive patients who are being restrained by staff and need to have a needle kept in a vein, there is a powerful incentive to get the matter over with quickly. But as Gilman *et al* (1990) write of intravenous injections, "once the drug is injected there is no retreat". While the same might be said of intramuscular injections, the effects are not so sudden. We think that the issue of intramuscular versus intravenous rapid tranquillisation should be addressed by a prospective study with random allocation of patients to either intramuscular or intravenous treatment groups.

BRITISH NATIONAL FORMULARY (1991) *British National Formulary*. London: British Medical Association and the Pharmaceutical Society of Great Britain.

GILMAN, A. G., RALL, T. W., NIES, A. S., *et al* (1990) *The Pharmacological Basis of Therapeutics* (8th edn). New York: Pergamon.

REYNOLDS, J. E. F. (1989) *Martindale. The Extra Pharmacopoeia* (29th edn). London: Pharmaceutical Press.

MARGOT QUENSTEDT  
ROSALIND RAMSAY  
MORRIS BERNADT

*Farnborough Hospital  
Kent BR6 8ND*

SIR: We found the paper by Pilowsky *et al* (*Journal*, June 1992, **160**, 831–835) on the use of rapid tranquillisation in a general psychiatric hospital compelling reading.

While psychological, behavioural and therapeutic restraint are acknowledged as alternative ways of managing aggressive patients, we were dismayed that no mention was made of the use of continuous observations. Shugar & Rehaluk (1990) found that continuous observation provides the essential ingredients of reduced stimulation, protection, intensive observation, and an opportunity for therapeutic contact and that its use forestalls and manages self-destructiveness, violence and over-stimulation of psychiatric in-patients. Our own study confirmed these findings. We studied consecutive acute psychiatric admissions in the Nottingham Health District, and found 14 documented incidents in a 28-day period. However, there were no untoward incidents when patients were observed on the most intense level of observation. In Nottingham this entails a designated nurse being in visual contact with the individual patient at all times.