research strategy. An early example might be the incorporation of results from the cross-institutional study (described above) into the standards: robust results about what matters in therapeutic communities will be discussed at a stakeholder conference and incorporated by the democratic process.

This is more than an audit cycle because it will be producing new knowledge (about what defines a therapeutic community, and what is good practice within one) and bringing about structural change in systems (new ways of doing things). It is an action research project because it will gain its legitimacy through a consensual process involving all involved parties: all the stakeholders ‘own’ the emerging results. It is a coherent way of ensuring and improving quality while bringing about coordinated and research-based change. Furthermore, it institutionalises the process of change, so therapeutic communities become responsive to the superordinate systems upon which they rely for survival. They will need to continually negotiate their place among other communities and other treatment modalities by maintaining a culture of enquiry (Main, 1967; Norton, 1992) about their own practice.

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Grand rounds: catatonia

Classical cases of catatonia in schizophrenia have become much less frequent with the introduction of neuroleptic drugs (Blumer, 1997), and psychiatrists practising in Western countries may well have seen few cases. Neuroleptic malignant syndrome (NMS), however, continues to occur in between 0.02% and 0.4% of patients and may occasionally prove fatal (Addonizio et al, 1986). We report a case with features of both these conditions.

Case history

The patient, 51 years old, was first admitted to hospital in 1966 at age 19. He was noted to have a “fear of death” and “increased sexual desire”, and a “very abnormal electroencephalogram” and an encephalopathy resembling leucoencephalitis was suggested. Since childhood the patient was said to have had “tics and mannerisms”.

A diagnosis of schizophrenia was made in 1967, when he was 20 years of age, and he has needed over 14 further admissions presenting with a number of delusions. He has been an in-patient continuously since 1986, when he was aged 39. Over the years a wide range of antipsychotic medication (including chlorpromazine, thioridazine, trifluoperazine, perphenazine, haloperidol, fluphenazine and benperidol) has been required, often in substantial doses.

In recent times he has suffered persistent, though variable, symptoms of psychosis, stereotypies, mannerisms,
evidence of tardive dyskinesia and possible torsion
dystonia. His drug treatment prior to this exacerbation
consisted of fluphenazine decanoate depot, oral ben-
peridol and diazepam.
A dramatic change in his condition occurred in
October 1998. He became restless and self-care
worsened. He heard voices from God about damnation,
and felt guilty and worthless. He felt he might soon die.
He felt thoughts inserted into his mind, and experienced
the feeling of being pushed against the walls. Over 10
days he needed a total dose of 650 mg of zuclopenthixol
acetate intramuscular, with additional droperidol and
diazepam as required. He had a number of bruises from
falling and the need for restraint. His creatinine kinase
level was raised at 4960 IU/l, as was the neutrophil
count; heart rate was 116 per minute. He suffered
periods of screaming and apparent rigidity.
Transfer to the adjacent general medical unit was
undertaken. He remained ill, and very distressed. He was
sweating markedly and blood pressure was lable. His
limbs became increasingly rigid, and attempted move-
ment was very painful. For a time he was unresponsive,
moving only his eyes. Temperatures spiked, up to 38.9 °C,
and respiration was impaired. Cortisol levels were raised.
Analysis of the raised creatinine kinase level showed it to
be of muscle rather than cardiac origin. An Escherichia
coli urinary infection was identified and treated. A lumbar
puncture was undertaken, but the cerebrospinal fluid was
normal. Computerised tomograms of the head were also
normal. Benzodiazepines and anticholinergic medication
were required on a number of occasions.
The patient, however, remained significantly
distressed and delusional and required electroconvulsive
therapy (ECT). He improved swiftly and substantially,
returning to his state of health prior to this exacerbation.
He was prescribed risperidone, and for the emergence of
affective features, lithium was added. Following a relapse
requiring further ECT, clozapine was started and—
valproate later added following an epileptic fit. He has
also required low dose thioridazine and diazepam.
However, he is currently fairly well, with no active
psychoses or affective symptoms.

Discussion
Catatonia was first described by Kahlbaum in 1874 (Kahl-
baum, 1973). It is seen in affective and schizophrenia
disorders, and is characterised by abnormalities of move-
ment including posturing, stereotypies and waxy flex-
ibility. Language abnormalities may also be present and
include echolalia and verbigeration (Murray et al, 1997). In
the past chronic catatonia states were common sequelae of
encephalitis lethargica; now that this is no longer
epidemic, catatonia has lessened in frequency in psychia-
tric hospitals (Johnson, 1993), but is still reported to
occur with regularity in district hospital units. Treatment is
to avoid neuroleptics, possibly prescribe benzodiazepines
and ECT is of value.
The NMS was first described in the English language
literature by Delay and Deniker in 1968. The onset is some
2–28 days after receiving neuroleptics. Muscular rigidity,
akinia, pyrexia, clouding of consciousness and auto-
nomic changes may be present – the latter including
hypertension, sweating, pallor and urinary incontinence;
there may be a neutrophilia, raised creatinine phospho-
kine (CPK; an enzyme released in muscle damage) and
raised potassium levels (Murray et al, 1997).
It may last 5–10 days after drug discontinuation,
but longer with depot preparations. There is no treat-
ment proven to be effective, but bromocriptine and
dantrolene have been used. It has been more recently
suggested that CPK levels are raised in catatonic
patients, and this is related to dyskinetic movements
(Northoff et al, 1996). The coexisting E. coli urinary tract
infection from which this patient suffered did complicate
the clinical picture, and may have contributed to the
pyrexia and neutrophilia.
Forty years ago, here in Dumfries, Mayer-Gross, in a
text influential in its time, drew attention to coexisting
physical illnesses: he felt that they were the immediate
cause of death in such cases, and the patients’ abnormal
behaviour impeded their diagnosis (Mayer-Gross et al,
1960; Kellam, 1987). Again, more recently, it has been
suggested that NMS is not an unitary syndrome, but
could represent extrapyramidal side-effects (EPS) compli-
cated by medical disorders – with dehydration, infection,
pulmonary embolism and rhabdomyolysis being common
complications of untreated EPS (Levinson & Simpson,
1986). Features common to both catatonia and NMS are
increasingly recognised, with the NMS felt to closely
represent advanced catatonia; similar syndromes with
unexplained fulminating hyperpyrexia, usually in the
setting of catatonia, have been noted since the beginning
of the 19th century, well before the development of
neuroleptics (Kellam, 1987). A common neurochemical
basis has been postulated for catatonia and NMS: a
massive blockade of dopamine receptors, previously
hyper-stimulated by psychoses or by dopamine blocking
drugs (Osman & Khurasani, 1994), and both share a
common beneficial response to ECT. Patients with past or
present catatonic symptoms are particularly vulnerable to
NMS (Blumer, 1997).
This is a complex and partially understood area, but
one that deserves a high level of awareness both owing to
the morbidity and mortality that may occur, and to the
fact that treatment may be effective. While some litera-
ture suggests that lethal catatonia often requires neuro-
leptic treatment (Castillo et al, 1989), it is more widely
held that, as mentioned in a review article: “Although the
mental blockade effected by routine early prescription of
neuroleptics appears to prevent emergence of the
previously plentiful catatonic symptoms in schizophrenia,
once catatonic signs are already present the neuroleptics
must be avoided because of the risk of severe catatonia
with NMS. For the same reason, neuroleptics should be
prescribed with caution for patients with a history of
catatonia.” (Blumer, 1997)
This patient had no history of catatonia, but there is
the possibility of one of a movement disorder, and more
recently he had suffered from a degree of tardive
dyskinesia. Conversely, this patient did have frequent
symptoms of psychoses over recent years requiring active
treatment. Clozapine, which this patient is now receiving,
has been shown to be of value after an episode of NMS
(Weller & Kornhuber, 1992). However, atypical antipsy-
chotics including clozapine, olanzapine and risperidone
can also cause NMS and catatonia. Clozapine is not
available on the Japanese market owing to reports of
malignant hyperthermia.

Despite the decline in the prevalence of classical
catatonia, we should remain alert to it, and to NMS. While
remaining aware of the benefits that traditional neuro-
leptics have brought, and their continuing value as part of
a package of treatment in the emergency situation, we
must continue to use them with caution. When catatonia
does occur, a prompt response and liaison between
psychiatrists and physicians is essential.

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With the population of India recently increasing to over
1 billion, the need for personnel to cater to the health
services is ever increasing. This article will touch upon
various aspects of psychiatric training in India and briefly
cover some aspects of psychiatric practice and services in
the country.

Health care services and medical training
India comprises 28 states, with each state having its own
health care delivery service. The central government’s
Ministry of Health and Family Welfare is responsible for
policy development and there are a large number of
centrally funded health care delivery institutions. India has
a large government funded health care system, although
standards of care and funding vary. The private sector has
been increasing year by year and there are many inde-
pendently practising general practitioners and specialists
and a network of private hospitals. The government
funded service functions on a three tier level, with
primary health centres covering a defined area. The
secondary level comprises various district hospitals. The
tertiary level are various medical colleges and centrally
funded hospitals with subspecialities.

India has one of the largest number of doctors in
training in the world, with 17,000 students entering
medical school every year. Medical graduates train in
state or centrally funded medical colleges. There are
about 162 medical colleges (Ministry of Health and Family
Welfare website: http://mohfw.nic.in). Medical training
in government funded colleges is relatively cheap.
Competition to enter medical training is high, with a few
hundred thousand students competing in a common
entrance examination. Additionally, there are 49 private
medical colleges and the number is increasing. These
colleges have their own entrance system and fees can be
high.

Training to be a psychiatrist
Doctors train for 4- and a-half years, after which they do
a year of internship (equivalent to house officer) and are
ready for postgraduate training. Emphasis on training in
psychiatry during undergraduate training is dismally low;
the Medical Council of India guidelines show that
students are required to participate only in a 2-week
programme of clinical postings, excluding a number of
theory lectures. Also, the staff teaching the undergraduates
are relatively junior and not fully trained in teaching
methodology, curriculum planning and use of teaching
aids (Alexander & Kumaraswamy, 1995). Those interested
in gaining experience can work as junior residents