scan published in their article, we suggest an alternative explanation. The enlarged distance from the temporal tip to the inferior frontal gyrus is consistent with the radiological diagnosis of 'open operculum'.

The diagnosis of open operculum suggests an underlying mechanism distinct from that which results in atrophy. Tatum *et al* (1990) suggest that an open operculum is the result of abnormal neuronal migration during the first part of gestation. They gave case reports of four infants with open opercula. In one infant, examined neuropathologically, they also detected abnormalities in the formation of the cerebral cortical gyri (i.e. macrogyria and micropolygyria). Another infant had Acardi syndrome, known to be associated with micropolygyria and heterotopias. These cerebral cortical anomalies are most likely the result of abnormalities in neuronal migration *in utero* (Larroche, 1984).

We recently detected similar cerebral cortical anomalies consistent with defects in neuronal migration in seven of 13 high functioning autistic males (Piven *et al*, 1990). We performed magnetic resonance imaging (MRI) on 13 autistic men and 13 controls (comparable on age and IQ) and found five autistic individuals with polymicrogyria, one with macrogyria, and one with schizencephaly. In another preliminary study, Berthier *et al* (1990) reported developmental cortical anomalies in two subjects with Asperger's syndrome studied with MRI. One subject was also noted to have a MRI scan consistent with opercular dysplasia.

Thus, the radiological diagnosis of open operculum is consistent with our studies of radiological abnormalities of the brain in autism and Asperger's syndrome. Further, the clinical course which would be expected with a developmental brain abnormality is more consistent with the natural course of both Asperger's syndrome and autism than that which would be expected with the occurrence of atrophy.

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Bipolar affective disorder and anoxic brain damage

SIR: Collins & Jacobson (*Journal*, May 1990, **156**, 736–740) discussed the case of a bipolar patient who developed mild brain damage from anoxia after attempting suicide by hanging. The discussion helped resolve many of the complexities of the diagnosis and treatment of this patient who had concomitant neurological and functional abnormalities. We suggest an additional interpretation of the patient's condition, which may contribute to an understanding of the evolution of her symptoms.

The authors reported that during the two months following the suicide attempt Ms A showed mutism and poor self-care. Although these symptoms can be attributed to depression, they occurred within a constellation of several other neurological abnormalities. Also, the psychiatric staff did not observe frank depressive symptoms. The patient was free from vegetative symptoms of depression. Her sleep and appetite were unremarkable and there was no diurnal variation in her behaviour. It is probable that the patient's depression improved for a period of a few months after her suicide attempt. Although one cannot rule out the effect of the electroconvulsive therapy that the patient was receiving before the suicide attempt, it is possible that the suicide attempt itself had an impact on the patient's mood.

Improvement of mood after a failed suicide attempt is a fairly common but probably temporary event. Lesse (1967) described the positive effects of suicidal behaviour on the mood. These 'apparent remissions' after suicide attempts can be attributed to the cathartic effect of suicide and its role in stimulating an empathic and supportive environment and to the emotional impact of the survival.

We previously reported the observation of manic and hypomanic switches immediately or shortly after a suicide attempt by hanging in four depressed patients. Two of these patients were previously diagnosed as being bipolar (Bourgeois *et al*, 1985). In our review we cited the observation of the 19th-century psychiatrists that melancholics who survived suicide by hanging had their mental state improved. These authors were attributing the improvement to asphyxia. Moreover, 'carbonarcose' or anoxic shock (coma induced by letting patients inhale a mixture of 30% carbon monoxide and 70% oxygen) was

practiced by several psychiatrists in 1946 as a method of treatment of depression (Bourgeois, 1967). More recently, endogenous depressed patients were found to have a lower response to carbon dioxide (CO_2) inhalation. Damas-Mora *et al* (1978) hypothesised that depressed patients have hyposensitivity to CO_2. However, the relationship between anoxia, CO_2 sensitivity and depression is still speculative. Also, it is reported that during periods of acute ischaemia there is a progressive increase in calcium entry inside the cell, an increase in catecholamine release, and a rapid accumulation of cyclic AMP (Palmer, 1985). These changes may play a role in precipitating a mood change after suicide attempt by hanging.

In summary, Ms A probably had a partial and temporary change of depressive symptoms after her suicide attempt. This increased the complexity of her clinical picture. Whether the effect is biological, psychological or both, such possible effects need always be considered in the evaluation of the mental status of depressed patients who fail in their suicide attempt. Further research needs to be conducted on the effect of failed suicide on the mood as well as on the possible interesting relationship between anoxia and CO_2 in depression.

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Hyperinnervation of orbital frontal cortex in schizophrenia

SIR: Waddington (*Journal*, May 1990, **156**, 615–619) is rightly intrigued by Schetzman *et al*'s (1988) findings of increased resting glucose consumption (hypermetabolism) in the frontal lobe of neuroleptic-naive schizophrenics. This contrasts with several earlier reports of hypofrontality in previously treated schizophrenics. Dr Waddington discusses the speculation that hyperfrontality could indicate hyperinnervation of frontal cortex. He points out that this is a variant of the hypothesis that schizophrenia involves a fault in the normal developmental process of synaptic elimination. However, rather than discussing the relevant neurochemical evidence, Waddington reviews studies of the peripheral neural cell adhesion molecule (NCAM) whose relationship to the impaired synaptic elimination story seems obscure.

We presented direct evidence compatible with an excessive glutamatergic innervation of orbital frontal cortex in schizophrenia (Deakin *et al*, 1989). *In vitro* binding of ^{3}HD-aspartate, a ligand that binds to glutamate uptake sites, and of ^{3}H-kainate, a glutamate receptor ligand, were both significantly increased in orbital frontal cortex from left and right sides of schizophrenic brains obtained at post-mortem. We have since found increased ^{3}H-TCP binding to another type of glutamate receptor which is again localised to orbital frontal cortex.

Independent corroboration of increased frontal cortical ^{3}H-kainate binding is described in a Japanese brain series (Toru *et al*, 1988). Furthermore, G. P. Reynolds (personal communication, 1989) has recently found increased concentrations of glutamate itself, which are localised to orbital frontal cortex in schizophrenic samples.

We pointed out that an increased number of glutamate synapses in orbital frontal cortex could explain increased ligand binding to pre- and post-synaptic sites (Deaken *et al*, 1989). Since the changes were bilateral, we further suggested that an excessive mutual innervation of one orbital frontal cortex by the other could be the origin of the additional synapses. We suggested that the cause was an arrest in the normal developmental process which eliminates immature transient projections. We agree with Dr Waddington that more imaging studies in drug-naive subjects are needed to identify disease-related changes in cerebral function, but direct studies of the neurochemical anatomy of the brain are needed to identify the primary pathology.

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