Occasional Review

Why blame the obstetrician? A review

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Summary and conclusions

Because of superficial thinking obstetricians have been blamed unjustifiably for causing brain damage, cerebral palsy, mental subnormality, congenital torticollis, and facial palsy. It is essential to look behind obvious difficulties in labour, such as abnormal presentation or anoxia, to the underlying causes, which are often genetic or social, or concern other prenatal factors.

Introduction

Some obstetricians deserve blame and so do some paediatricians—for no one is perfect. But obstetricians, midwives, and others are commonly blamed unjustly for a child's handicaps—for example, cerebral palsy, mental subnormality, "brain damage," facial palsy, congenital torticollis with sternomastoid tumour, and other conditions. Psychologists, psychiatrists, and others often refer to the "brain-damaged child" or to "minimal brain damage," implying blame on the person who delivered the baby. In a recent court case an obstetrician was even blamed by parents and expert witnesses for causing pectus excavatum (funnel chest), a condition that is a purely congenital deformity, often familial.¹⁻³

Factors influencing obstetric difficulties

The reason for such unjust blame is the common failure to look behind the events of birth to the causes of those events—namely, genetic, social, or other prenatal factors. For instance, paediatricians and others may still ascribe facial palsy at birth to forceps delivery. But Hepner⁴ showed long ago that forceps delivery is irrelevant. He found that the incidence of facial palsy in 159 normal babies delivered vaginally without the use of forceps was 6.3% and that in 716 babies delivered with forceps was 6.4%. In the 56 cases of facial palsy in both groups the side of the facial weakness was constantly associated with the side of the obstetrical presentation. Most were thought to be due to pressure of the facial nerve area against the sacral prominence during labour. Occasionally facial palsy does not resolve, probably because of agenesis of the facial nucleus.

The obstetrician is commonly blamed for congenital torticollis with sternomastoid tumour. Long ago it was suggested that pre-existing torticollis prevented the engagement of the fetal head, and caused breech presentation.^{5,6} Watson⁷ found that the flat side in plagiocephaly and unilateral dislocation of the hip and the convexity in scoliosis

and the sternomastoid tumour tend to be on the same side, and the unilateral bat ear on the opposite side. He thought that the sternomastoid muscle was shorter before birth, and was the cause of the breech delivery. The shortening was probably due to ischaemic fibrosis resulting from venous occlusion by persistent lateral flexion and rotation of the neck in utero. This had been suggested by Jones,⁸ the author of a book on torticollis. He wrote that "The remarkably mature fibrous tissue in material from neonates suggests that the disease begins well before birth and is probably the cause, not the result of obstetric difficulties." The fibrosis affects the whole length of muscle, even though clinically the tumour is localised.

When a child is mentally handicapped and has had convulsions in the newborn period many ascribe the handicap to the convulsions. It is surely obvious that one should look behind the convulsions to their cause—the anoxia, hypoglycaemia, underlying brain defect, or other factors. In the same way obstetricians are often blamed for stillbirths; but a high proportion of these are due to chromosomal defects incompatible with life, or to congenital abnormalities or underdevelopment of the brain.⁹

Cerebral palsy or mental subnormality is often ascribed to breech delivery. I saw a mentally subnormal child, with a single palmar crease and other abnormalities, who had been small for dates and whose mother had lost a considerable amount of weight in the last six weeks of full-term pregnancy: a paediatrician had confidently diagnosed birth injury because the delivery had been by breech. One would have thought that other factors were more likely. Cerebral palsy is associated with a somewhat higher incidence of breech delivery, but it is essential to look for the factors that caused the breech delivery. For instance, Lorber showed in Sheffield that infants with spina bifida were six times more likely to have been born by breech than others. Braun *et al*¹⁰ wrote that the factors causing breech presentation were smallness for dates, multiple pregnancy, bicornuate or double uterus, prematurity, hydramnios, oligohydramnios, and fetal abnormalities. The incidence of breech presentation was much higher in children with spina bifida, hydrocephalus, anencephaly, congenital dislocation of the hip, Riley's syndrome of familial dysautonomia, the Prader-Willi syndrome, trisomies, the Smith-Lemli-Opitz syndrome, the Cornelia de Lange syndrome, the Zellweger syndrome, Werdnig-Hoffmann's disease, myotonic dystrophy, and fetal alcohol syndrome.

Maternal and parental factors

The risk of cerebral palsy is six or seven times greater in multiple pregnancy than in the normal population, and this is commonly ascribed to the complications at birth—the breech presentation, prolapse of the cord, and other difficulties. Nevertheless, important factors are associated with multiple pregnancy—namely, increased incidence of toxæmia, hydramnios, congenital deformities, smallness for dates, placental insufficiency, bleeding during pregnancy, placenta praevia, and genetic factors.¹¹⁻¹⁴ Multiple pregnancy is more common in lower social classes, in short women, in primiparae or in multiparae after the fifth pregnancy, and in mothers over 35. The reason for preterm delivery in multiple pregnancy is not clear. In North Finland the incidence of conceptions of twins is higher in the light days and nights of summer than in the darkness of winter.

A third of all children with cerebral palsy are born prematurely. I observed that the incidence of preterm delivery in 762 children with cerebral palsy was 30%. Preterm delivery of severe degree carries a risk of mental subnormality, yet has many causes, such as social,

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nutritional, infective, and genetic factors, advanced maternal age, toxæmia, antepartum haemorrhage, previous induced abortion damaging the cervix, and other causes.¹⁵ Preterm delivery is much more common in lower social classes, as Dugald Baird showed 20 years ago in Aberdeen.¹⁶

Intrauterine growth retardation, causing smallness for dates, with a small placenta and especially oligohydramnios, is associated with an increased incidence of cerebral palsy and congenital anomalies. It is itself related to social factors, maternal malnutrition, toxæmia, smallness of maternal height, smoking in pregnancy, birth of previous low birth-weight infants, hypertension, and antepartum haemorrhage.¹⁷ Postmaturity considerably increases the risk of cerebral palsy and to some extent that of mental subnormality.¹⁸ The reason may lie partly in placental insufficiency.

Maternal age is associated with the incidence of cerebral palsy, the older mother, especially a primipara, being at risk. One factor may be the increased incidence of fibroids with age, with a risk to placental implantation, but maternal age is relevant for other reasons—such as social factors, the incidence of toxæmia, and preterm delivery—all of which are associated with cerebral palsy and mental handicap.

A major factor in the history of children with cerebral palsy and other handicap is relative infertility in the parent—that is, a longer than usual time before conception, together with a higher incidence of preterm delivery, abortions and threatened abortions, bleeding in pregnancy, or stillbirth—not only in the pregnancy that resulted in a handicapped child, but in the previous and subsequent pregnancy. Relative infertility has been noted in most studies of the aetiology of cerebral palsy in the past 20 years or more. For instance, Chefetz,¹⁹ in a study of the multiplicity of factors in 275 cases of cerebral palsy, found “reproductive insufficiency” in 78.4%. The incidence of bleeding in pregnancy was three times greater than in controls, and the incidence of placental complications was seven times greater. Denhoff and Holden²⁰ concluded that “one important area of research in the aetiology of cerebral palsy involves further investigation of reproductive physiology.” Ingram and Russell,²¹ investigating the prenatal history of 278 children with diplegia, found that as compared with controls there were more mothers over 30, fewer conceptions than in married childbearing women in Scotland, wider spacing of pregnancies, and more abortions, stillbirths, and neonatal deaths. Fewer than half of the conceptions resulted in the birth of healthy offspring surviving the first year, and the pregnancies contiguous to those resulting in diplegia were more often abnormal. They concluded that underlying abnormalities of the reproductive process were present, which caused both disorders of pregnancy and those of labour and delivery and the production of abnormal offspring. The main abnormalities of pregnancy were bleeding, toxæmia, prolonged labour, and breech and preterm delivery.

Bleeding commonly occurs in pregnancies leading to a child with cerebral palsy. Worcester *et al*²² found that 25% of severely malformed children at Boston had resulted from pregnancies in which bleeding had occurred. Latham *et al*²³ found that bleeding had occurred in pregnancy in 39.3% of 61 children with cerebral palsy. In nine of the 24 cases the bleeding occurred before the 20th week, in 10 after the 20th week, and in five before and after that time. Turnbull and Walker²⁴ and South and Naldrett²⁵ noted the association between bleeding in pregnancy and congenital abnormalities; the second group of workers²⁵ found that 1226 out of 7824 women in singleton pregnancies (15.7%) had vaginal bleeding before the 28th week. The mean birth weight was significantly reduced, more of the babies were preterm and small for dates, and, especially when bleeding occurred between the 16th and 28th weeks, more had congenital anomalies. Chefetz¹⁹ found that bleeding occurred three times more often in pregnancies resulting in cerebral palsy than in controls; and Hagberg *et al*²⁶ found that the incidence of bleeding was six times greater than that of women in Sweden generally. In the British Perinatal Mortality Survey²⁷ the influence of these various adverse prenatal factors, particularly the various indications of relative infertility, was convincingly shown in a detailed statistical analysis.

Genetic and other factors

An important fact that indicates a prenatal and often genetic factor in mental subnormality and cerebral palsy is the high incidence of congenital anomalies. In my own series of 1068 mentally subnormal children without cerebral palsy, excluding mongols, cretins, and infants with hydrocephalus, 29.3% had major anomalies; while in 762 children with cerebral palsy, 7.5% had major anomalies, an incidence about three times that of the normal population, and a figure almost exactly the same as that of Latham *et al*.²³

Sigmund Freud²⁸ was one of the first to mention genetic factors in cerebral palsy. He suggested that genetic and other prenatal factors predispose to perinatal injury. Penrose²⁹ suggested that some cases of spastic diplegia with mental deficiency were due to a recessive gene. Gustavson *et al*³⁰ studied 43 families in Sweden with more than one affected child, and found that in 24 of the families the types of cerebral palsy (spastic, athetoid, ataxic) were different in the same family. In my own series of 762 cases of cerebral palsy, excluding kernicterus, 5.0% had an affected sibling. Ingram³¹ found that 10% of cases had an affected sibling or near relative. I have seen families with more than two affected children. In one family that I saw a mentally subnormal child was born with the spastic form of cerebral palsy. A doctor told the parents that this never happened again, but a second child was born, who was mentally subnormal and had athetosis. The parents were then told that the cerebral palsy was only due to birth injury, and that if a child were delivered by caesarean section it would be normal. As a result the third child was delivered by caesarean section. He was a mentally subnormal spastic. In my book *Recent Advances in Cerebral Palsy*³² I gave 21 references to papers on the genetic aspect of cerebral palsy.

Infections during pregnancy rarely cause cerebral palsy. The infections are mainly toxoplasmosis, cytomegalovirus infection, or rubella. Rubella in early pregnancy may cause extensive lesions in the brain, resulting in cerebral palsy.^{33–35} Hyperthermia has been implicated as a possible teratogenic agent.³⁶ Eight children born to women who had high fever at six to eight weeks of gestation were mentally subnormal and hypotonic and had fits, with slight distal limb defects; five children born to mothers who had high fever at seven to 16 weeks had arthrogryposis and serious neurological deficiencies, findings corresponding to those produced in experimental animals.

The only drug or toxic substance known to cause cerebral palsy in humans is methyl mercury, which was the cause of hundreds of cases of “Minamata” disease in Japan, in which pregnant women ate fish contaminated by mercury in the effluent of a factory in the Inland Sea. Similar cases have been reported in Iraq and elsewhere; Koos and Longo³⁷ suggested in their review that contamination of fish by mercury is such that pregnant women in America would be unwise to eat more than 350 g of fish weekly. Recently methyl dopa, taken between the 16th and 20th week of pregnancy, has been shown occasionally to reduce head size.³⁸ So many scores of drugs are now known to damage the fetus in utero that studies in experimental animals may well show that other drugs cause fetal brain damage.

Abnormalities of conception

An important but difficult topic for research is the relation between time of conception in the ovulatory cycle and the occurrence of handicaps in the child. Takala³⁹ investigated the sperm of 189 fathers of malformed children and found a “markedly high incidence” of abnormalities, especially that of dark-stained heads. Furuhrjem *et al*⁴⁰ showed that a high incidence of abnormal sperms was associated with abortions, stillbirths, and perinatal death. Legros,⁴¹ in a prospective study of 19 pregnancies in 12 couples, in which the man had a high proportion of abnormal sperms, found that 11 pregnancies resulted in stillbirths or neonatal deaths (of males); four children had congenital malformations and two had cerebral haemorrhage. Results of several studies^{42–46} showed a connection between mongolism and an overripe ovum or aging sperm. Lanman,⁴⁷ in a series of three articles, reviewed the extensive experimental and clinical evidence on the relation between time of conception and the occurrence of defects in the fetus.

Relative importance of injury at birth

It would be absurd to suggest that birth injury does not occur, but its importance has been exaggerated. Penrose,⁴⁸ in a study of 1280 mentally defective patients, found that in only 11 did the physical condition combined with the history warrant a diagnosis of birth injury as the main cause. Perlstein⁴⁹ wrote that “Faulty obstetric practice, formerly thought to be responsible for most of these cases, is now considered by some authorities to account for only three to five per cent of the children. Malformations are now believed to account for a much larger proportion than was previously thought. These malformations may be the result of morbid heredity or arrested development due to insult of the embryo in utero.” Dugald Baird¹⁶ investigated 64 children in a school for handicapped children in Aberdeen. An abnormality of pregnancy or labour was found in only seven, while an undoubted causal relation could be shown in only one. Roberts⁵⁰ studied 117 cases of intracranial haemorrhage in the new-

born; 38 had had a normal delivery without difficulty, and many of the others, though delivered by forceps or other methods, had presented no special problems. He concluded that many cases of cerebral palsy and other defects are not due to birth injury but to prenatal maldevelopment. In the discussion following the paper, Tyson said, "I fear that in the past many obstetricians have been falsely accused of poor obstetric technique."

Durkin *et al*⁵¹ studied 281 children with mental subnormality and cerebral palsy. There was the usual history of relative infertility, prematurity, or postmaturity, but in one-third there was no abnormality of labour, in one-third only trivial difficulties that normally cause no trouble, while in the remaining third considerable difficulties had been present. As I have said above, the causes of such difficulties are highly relevant. I have long been impressed by the fact that those babies who have a disastrous experience in delivery, with forceps delivery having failed or other problems, are not those that are found to have cerebral palsy or severe mental handicap. Others have made the same observations.⁵²⁻⁵³ Amiel-Tison⁵² followed 41 full-term newborn infants who had suffered severe birth trauma; on follow-up two to five years later none had cerebral palsy. In most cases of mental and physical handicap there is no evidence of birth trauma. Even if difficulties occurred at birth, one should try to determine the underlying reasons for the difficulties and the early factors that predisposed to problems or injury at birth.

Anoxia

Probably the most important factor in the aetiology of cerebral palsy—and an important factor in that of mental subnormality without cerebral palsy—is anoxia. Probably the main cause of perinatal death,⁵⁴⁻⁵⁵ it may have an obvious cause, such as obstruction of the umbilical cord, prolonged labour, or premature separation of placenta—but one must then consider what caused those conditions. Adamsons and Myers⁵⁶ suggested that prolonged uterine contraction was more relevant to the deprivation of oxygen to the fetus than the mere amplitude and frequency of contractions. Nevertheless, many babies are born with a low Apgar score with no obvious perinatal cause of anoxia and many handicapped children have experienced anoxia of a degree that others have experienced without any sequelae.

Furthermore, many follow-up studies have been performed on children who had severe anoxia at birth and who suffered no permanent ill effects. For instance, Apgar and her colleagues⁵⁷ investigated the capillary blood oxygen saturation of 243 randomly selected babies at intervals in the first three hours, and found no significant correlation between blood oxygen concentrations in the first three hours and subsequent Binet scores. Fraser and Wilks⁵⁸ in Aberdeen followed 40 severely asphyxiated babies and at school age found no important defects. Shipe *et al*⁵⁹ compared 33 children with an Apgar score of five or less with controls, and found no difference in later performance on psychological and personality ratings. Gordon *et al*,⁶⁰ in the collaborative perinatal study, found that most infants who had suffered perinatal anoxia were normal when re-examined at 8 months to 4 years. Hilary Scott⁶¹ examined 33 children in whom respiration had not been established by 20 minutes; three to seven years later three out of four were normal. Six had cerebral palsy, but four had suffered prolonged oxygen deficiency before labour. In a study that I performed on 60 severely asphyxiated infants, the Stanford-Binet tests at school age, done by an educational psychologist, gave a mean IQ score of over 100; none had cerebral palsy. But no one can say whether survivors who were apparently normal on follow-up had experienced subtle damage that was not identified, or would have been a genius but for the perinatal experience.

What we need to know is why some suffer sequelae of anoxia and others do not. The answer may lie in pathological studies. Myers⁶² and Brann and Myers⁶³ have shown that prolonged or repeated partial anoxia is the primary event that starts a vicious circle of brain swelling, leading to stasis of blood flow and cerebral necrosis; and that in animals repeated partial anoxia produces the pathological changes identical with those of cerebral palsy in the human, while complete anoxia does not.

Complete recovery is far more likely in the human infant after an episode of complete anoxia during delivery than after repeated or prolonged anoxia. Sylvester⁹ reported that mature infants with fetal distress had a significantly smaller placenta (in relation to body weight) than those without. Hilary Scott⁶¹ named several causes of impaired placental perfusion: toxemia, hypertension, maternal illness and heart disease, and bleeding in pregnancy, early or late. Hagberg *et al*²⁶ in Sweden found that the association between fetal deprivation of

oxygen in early pregnancy (as in partial separation of placenta with bleeding) and anoxia or cerebral haemorrhage during delivery was significant. They suggested that "fetal deprivation of supply affects the developing brain not mainly as the single cause of brain damage, but perhaps more as the predisposing background factor for subsequent otherwise innocent insults or disturbances of brain homeostasis which themselves might be of minor importance." Fraser and Wilks⁵⁸ and others have suggested that an intrinsic defect of brain development in fetal life may be the cause of anoxia during labour, so that subsequent handicap is due not directly to the anoxia but to the pre-existing brain defect. This might act, for instance, by depressing the respiratory centre. Severe global brain damage, causing cerebral palsy and gross mental subnormality, is unlikely to be due only to an episode of acute anoxia during delivery. Previous brain defect is a much more likely cause, and this may have predisposed to anoxia in labour, the anoxia in labour adding to the subsequent handicap.

Interrelations between harmful prenatal factors

Many years ago Lilienfeld and others⁶⁴⁻⁶⁶ compared the prenatal history of children with cerebral palsy, mental subnormality, epilepsy, and severe behavioural disorders with that of controls. In all these conditions the incidence of the factors outlined above was high. The authors coined the oft-quoted expression "a continuum of reproductive wastage," with a lethal component consisting of abortions, stillbirths, and neonatal deaths, and a sublethal component consisting of cerebral palsy and other related conditions. These same prenatal factors are relevant not only to cerebral palsy, mental deficiency, and perhaps epilepsy, but to preterm delivery, intrapartum anoxia, multiple pregnancy, and breech delivery. These factors or insults occur in many different permutations and combinations, and they cannot be disentangled. The result may depend on the nature and severity of the insult and the exact duration of gestation when the insults occurred. Chefetz,¹⁹ in his 275 case studies of cerebral palsy, wrote: "It is unusual to find just one cause for a given case of cerebral palsy," and he added that he had shown statistically the importance of a multiplicity of factors.

I agree with Cardwell,⁶⁷ who wrote that "It is becoming increasingly apparent that one cannot study causative factors in cerebral palsy apart from the wider problem of the causation of abortion, prematurity, stillbirth, neonatal death and congenital anomalies. A close interrelationship between those conditions has been established." Emminger⁶⁸ in Augsburg, in a study of necropsy findings in 191 cases of rupture of the tentorium or falx cerebri, found that in 150 cerebral haemorrhage resulting from severe anoxia or convulsions had been suspected but not confirmed at postmortem examination. He found no connection between complications of birth or mode of delivery and rupture of the tentorium, and thought that anoxia during fetal development put the fetus at risk of injury at birth. "Prenatal lesions are the cause of neonatal anoxia, falsely ascribed to birth trauma. The primary cause is fetal maldevelopment, birth only a secondary factor." Steer and Bonney⁶⁵ suggested that "noxious influences at work during conception, during organ formation, during intra-uterine development," predisposed to difficulties in labour. Neel⁶⁹ referred to "Insults falling on prepared soil."

That insults to the developing fetus have occurred is often shown in the primary teeth. Pindborg⁷⁰ showed that in about a half of all children with cerebral palsy enamel hypoplasia and other changes indicated insults during fetal development, which included maternal infections such as rubella and irradiation. Cohen and Diner⁷¹ wrote that "dental enamel defects are definitive chronological markers of the probable time of an insult to a developing fetus." A similar and important observation has been made by Professor J L Emery at Sheffield.⁷²⁻⁷³ Histological studies of the costochondral junctions of full-term infants who died during labour in the newborn period have shown that 75% had disordered, arrested, or bizarre growth at the rib end during intrauterine development. He suggested that these children, as a result of insults during pregnancy, were at risk of further difficulties during labour, normal or otherwise.

Discussion

In this review I have discussed only some of the conditions that the obstetrician is unjustifiably blamed for causing. I have not reviewed factors in the care of the newborn which are relevant to cerebral palsy and other handicaps. I have suggested that, as a

result of superficial thinking, obstetricians receive a great deal of unjustifiable blame for causing physical and mental handicaps, and especially "brain damage." They are blamed for causing cerebral palsy and mental subnormality when abundant evidence shows that the causes are far more complex and that many interrelated damaging factors underlie these conditions. Some of these factors may lead to obstetrical difficulties or predispose to additional damage at birth, whether the birth is normal or not.

Psychologists, child psychiatrists, and often paediatricians label many children as "brain damaged," much to the distress of parents and to the children's disadvantage. In the United States scores of thousands of children are said to be suffering from "minimal brain dysfunction" or "minimal brain damage," a ragbag diagnosis into which over 100 symptoms (such as "over-activity") have been put.⁷⁴ Inevitably the term "brain damage" implies blame on the person who delivered the baby, though some psychologists have denied this, claiming that "brain damage" may occur at any time during the pregnancy. That may be, but parents inevitably assume that the damage was done at birth. If a tragedy befalls any of us we feel better if it could not have been avoided than if it was due to someone's carelessness. The diagnosis has no possible validity, for psychological and other tests cannot establish the diagnosis in a child who survives. Even in the newborn period it is difficult to establish the diagnosis of cerebral haemorrhage, other than a subdural effusion, in a child who survives; and without a lumbar puncture, which itself is often difficult to interpret, the diagnosis cannot be made.

In recent years British obstetricians and neonatologists have received blame indirectly for causing hundreds of cases of cerebral palsy unnecessarily because of failure to apply modern techniques, and for causing our perinatal and infant mortality to be significantly worse than that of other countries in Europe. Those who make such charges pay no attention to the great difficulty of making comparisons among countries because of important differences in definitions,⁷⁵ or to the different distribution of social class and differences in the incidence of prematurity and congenital abnormalities, the effects of immigration, and many other factors. For instance, the incidence of low birth weight babies varies considerably between countries, and this inevitably affects perinatal and infant mortality. Whereas in Scandinavia roughly 4% of all births are infants of low birth weight, the figure for England and Wales is approximately 6.5%, and is still higher in America. At different stages of gestation the mean weight varies—for example, figures calculated by Rantakillio⁷⁶ in Finland show that at 32 weeks' gestation the mean weight is around one pound (454 g) more than that in Britain, a difference that would be highly relevant in comparing survival rates. Furthermore, closer inspection of the British figures shows that our neonatal mortality, for which obstetrical management is so important, compares favourably with that of other European countries, while our postneonatal mortality lags behind, probably mainly for social reasons.

As for the charge that the number of handicapped children born in Britain could be appreciably reduced by applying modern methods of management, that may be so. Nevertheless, Turnbull⁷⁷ wrote that studies in two units in Cardiff, one with an extremely conservative approach to advances, and the other using more radical policies, could show no significant differences in outcome. In Sheffield Kelso *et al*⁷⁸ studied 504 pregnancies in a controlled trial of fetal monitoring versus intermittent auscultation. They found no difference in neonatal deaths, Apgar score, maternal or neonatal mortality, or blood-gas concentrations. They concluded that continuous heart monitoring in labour had led to neither beneficial nor deleterious results.

That which is most up to date and new is not necessarily the best.

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MATERIA NON MEDICA

An inland lifeboat

What do you do to transfer a patient in urgent need of treatment from her home 25 miles to the hospital, 25 miles from the coast inland, when the roads are blocked by snow drifts and no transport can get through, when the trains are halted by iced points, and when the local RAF helicopters are grounded because of poor visibility? Recently, when Norfolk experienced the worst weather conditions since 1947, such a problem arose: three patients in urgent need of haemodialysis were marooned in Great Yarmouth and unable to reach the Norwich renal unit. Road and railway communications were blocked, and helicopters at RAF Coltishall, far from being able to take off in the blizzard conditions, could not even be brought out of their hangers. The lifeboatmen at Gorleston, with the same courage and resourcefulness which produced Norfolk's best-known lifeboatman, Henry Blogg of Cromer, answered the emergency call and brought the three patients inland up the river Yare to Norwich. As the coxswain said: "It was pretty dodgy: it's not surprising really, whoever heard of a lifeboat going on a twenty-five mile trip inland? We couldn't have done it without radar. We hit packs of ice and debris, and in places it was difficult to spot where the river went because of flooding." After a two and a half hour journey the three patients arrived in Norwich in fine form and treatment started soon after.—D B HAMILTON (senior medical registrar, Norwich).

The duck on the turbulated tricycle

I was disappointed to miss the clockwork monsters—Alain Bourbonnais's "turbulents"—actually moving. They are among the many memorable exhibits at the Hayward Gallery's "Outsiders" exhibition (on until 8 April). Working from no cultural tradition, and seeking no recognition, these are artists who create an "art without precedent" or *Art Brut*. At least 24 of the 42 artists here were psychotic, though this does not apply to all Outsider exhibitions.

These works, claims the catalogue, are "about intense experience which, if we are receptive, we will recognise as our own." Does this apply to, say, Friedrich Schröder-Sonnenstern's paintings celebrating the monstrousness of the human body, or to Francis Marshall's hideous figures (made of nylons and rubbish from the banks of the Seine tied together with string) suggesting the banality and squalor of French rural life? However that may be, much of the work is thought-provoking and intriguing, and sometimes beautiful with a surrealist improbability or monumental detail and symmetry.

These artists, we are told, "dared" to be original, to reject cultural traditions. Many of them had little choice, having deprived or humble backgrounds and sometimes bizarre careers. Henry Darger worked all his life as a lavatory cleaner; Schröder-Sonnenstern, in and out of prison and asylum, was a thief, smuggler, confidence trickster, fortune teller, and leader of a mystical sect. A few did have cultural backgrounds, which they later rejected. Louis Soutter, for instance, did thousands of drawings and silhouette finger paintings with a primitive

energy that broke right away from his early art studies. But by this time he was mentally ill. Presumably kicking over the traces, artistically speaking, becomes easy—or inevitable—for the psychotic, at least for those who have the talent. Think of Louis Wain and his cats: his anthropomorphic and social cats and the increasingly stylised cat faces of his schizophrenia.

Apart from an altered mental state, however, the psychotic artist has a vast emptiness: the emptiness of institutional life, usually, as well as of social withdrawal—with no "persons from Porlock" to interrupt his art. Many of these artists spent decades in institutions; because of his violence Adolf Wölfl spent his last 23 years in a one-man cell that he crammed with manuscripts and drawings. And many of the others have led isolated lives. To most of us our involvements and our full lives are precious; and we would dismiss as one-sided Francis Marshall's saying that "Rules, restrictions, work torture people, . . . deform them, crush them, rot them." But I think this exhibition is good for us in pointing to possibilities we should not lose sight of, and to our need for some empty spaces in which to explore and be in touch with ourselves.—DAPHNE GLOAG (London).

The science and art of government among the Scots

1979 is the quatercentenary of the publication of George Buchanan's *Dialogus de Jure Regni apud Scotos*. Although the book was badly received by King James VI (to whom it had been dedicated) and by the Establishment, and was condemned by the Scottish Parliament, banned after the restoration of Charles II, and ordered by the University of Oxford to be publicly burned by the common hangman, the book had a profound influence on political thinking of the time.

The book had been published in Scotland just after the Scottish people had forced Mary Queen of Scots to abdicate in favour of her infant son James VI, and Buchanan argued the right of the people to bring the King to order. This at the time was a remarkably novel principle and appealed to those seeking a more democratic parliamentary system. Indeed, in England the Parliamentarians frequently cited Buchanan to justify their own proceedings against their King, Charles I. And the ideas Buchanan enunciated were woven later into the constitution of the United States, which was brought home when the will of the American people forced the resignation of their president, Mr Richard Nixon over the Watergate affair. The *Dialogus* of George Buchanan has been ignored in English universities and, surprisingly, has been neglected in Scottish universities, but in European seats of learning it is considered a major classic in the art and science of good government.

George Buchanan has been maligned by several British historians, but this Gaelic-speaking Highlander was a great Latin scholar, poet, historian, (he wrote a massive history of Scotland), reformer in education (he reformed St Andrews University), religion, and politics. He died in 1582, penniless, his grave being marked by a simple *throckstone* in Greyfriars Kirkyard, Edinburgh.—W WATSON BUCHANAN (Hamilton, Ontario).