send anyone the full list, which is too long to reproduce here. The copies of discharge summaries and notes in our possession total four shelf-inches, A4 size.

We hope this obituary is premature. It was once said that old patients with Munchausen's syndrome were like old soldiers: they never died but just faded away. Stewart McIlroy taught many lessons to those who were deceived, not least being the lesson that we are not always the astute physicians we should like to believe.

Postscript

Munchausen's syndrome has been much discussed since Asher first described it.[2] The difficulty of arranging psychiatric treatment is well known: the patients will not stay long voluntarily, and it is usually impossible to justify detention under the sections of the Mental Health Act.[3] The concept of a general register has been rejected as unethical, and the black book kept in casualty departments is circumvented if (*a*) a new name is used and (*b*) the symptoms are convincing—and they usually are, especially if there is a genuine underlying medical condition.[4]

Our thanks are due to the many physicians and medical records staff who replied to our requests for information during the past four years.

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Neural-tube defects: importance of a history of abortion in aetiology

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Summary and conclusions

The overall incidence of anencephaly and spina bifida (ASB) in 69 056 pregnancies was 4·7/1000 births. ASB was more common (8·4/1000 births) among children of mothers who had had two or more abortions, but the increased risk was confined to spina bifida. A history of abortion was more common in older women and women of higher parity, but this was not matched by a similar increase in the incidence of ASB. The incidence of ASB was related to social class, but the prevalence of previous abortions was similar in all classes.

The results suggest that expectant mothers with a history of two or more abortions have an increased risk of producing a child with spina bifida. The abortions are considered to be a manifestation of previous abnormal conceptions rather than the primary cause.

Introduction

Clarke *et al*[1] suggested that trophoblastic "rest" material remaining after a miscarriage may be associated with the subsequent delivery of a child with anencephaly or spina bifida (ASB), postulating that the rest material interfered with normal formation of the neural tube during early fetal life. This suggestion was confirmed by Gardiner *et al*,[2] who found that patients whose previous conception had ended in spontaneous abortion had a significantly higher incidence of infants with congenital malformation than patients whose previous conception had resulted in a normal live-born child. Their study, however, was limited by small numbers. I have tested the rest hypothesis by looking at factors that influence the incidence of both ASB and abortions in an overall population.

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Patients and methods

Records were obtained from the Cardiff Births Survey (CBS) of all mothers who had given birth in Cardiff hospitals during 1965-76. An association was sought between ASB and a history of abortion. Although the CBS does not give detailed information on all immediate past conceptions or distinguish spontaneous from therapeutic abortion, this is not critical in testing the rest hypothesis, which postulates that it is the presence of trophoblastic material that is relevant, not the type of abortion. Further data linked the association between a history of abortion and ASB and, respectively, social class, maternal age, and parity—all three factors known to be causally related to neural-tube defects.[3 4]

Results

A total of 69 056 mothers delivered 70 871 infants. Of these, 331 had ASB (146 anencephaly, and 185 spina bifida), an overall incidence of 4·7/1000 births. The following analysis is of singleton births only.

Table I shows the incidences of ASB according to the past history of abortion. Of all 321 infants with ASB, 248 (77·3%) were born to mothers who had never had an abortion. Forty-eight mothers (15·0%) had had one abortion, and 25 (7·8%) two or more. The increased incidence of ASB after one abortion was not significant, but the increased incidence of spina bifida after two or more abortions was highly significant ($\chi^2 = 18{\cdot}5$; $P < 0{\cdot}001$). Table II shows the incidences of ASB in primiparous patients whose immediately previous conceptions ended in abortion. Again there was a highly significant increase in the incidence of spina bifida ($\chi^2 = 10{\cdot}2$; $P < 0{\cdot}001$) after two or more abortions. There was no association between a history of abortion and the incidence of anencephaly.

The incidence of ASB was highest in social classes IV and V (table III). The prevalence of a history of abortion, however, was similar in all classes. Table IV shows that ASB occurred most often

in mothers under 20; 6·5% of these women had had one or more abortions compared with 17·7% of mothers aged 20-29 years and 28·2% of mothers aged over 30. The incidence of ASB was high in primiparous patients, lowest in mothers with one existing child, and increased thereafter with increasing parity (table V). The incidences of ASB in primiparous mothers and those with three or more pregnancies were similar (5·1 and 5·3/1000 births respectively), although a history of abortion was present in only 11·0% of primiparous mothers compared with 40·2% of mothers with three or more previous children.

TABLE V—*Percentage incidences of neural-tube defects and prevalences of previous abortion according to parity. (Numbers of infants with ASB given in parentheses)*

Parity	Neural-tube defect		Prevalence of previous abortion	
	Anencephaly	Spina bifida	1	⩾2
0	0·22 (61)	0·29 (80)	9·4	1·6
1	0·15 (38)	0·22 (48)	14·6	4·0
2	0·23 (25)	0·23 (25)	18·8	2·9
⩾3	0·23 (22)	0·30 (28)	27·5	12·7

Discussion

The hypothesis that residual trophoblastic material from an abortion might interact unfavourably with the fetus in an immediately succeeding pregnancy and cause anencephaly or spina bifida was supported by studies[1 4 5] showing that the incidence of miscarriage in pregnancies immediately before ones resulting in a child with ASB was higher than in pregnancies occurring immediately after. The study by Gardiner *et al* was limited by small numbers and was biased by the fact that the index group with a past miscarriage contained several abortions which themselves were associated with neural-tube defects. The residual trophoblastic material hypothesis would be tested only if all spontaneous abortions had been free of CNS defects.[6]

I analysed a single population over 12 years in a high-risk area for anencephaly and spina bifida. The number of cases occurring in women with no history of abortion was similar to what might be expected by chance. If residual trophoblastic material was responsible for affecting normal CNS organisation in early fetal life, a commensurately higher incidence of ASB would be expected irrespective of whether there had been one, or more than one, previous abortion. In particular, the most noticeably raised incidence would be expected immediately after a previous abortion. Neither factor was found in this study. The higher incidence of spina bifida in the offspring of mothers who had had two or more abortions may be explained on the grounds that they represented a population containing a proportion of mothers who had previously conceived fetuses with ASB[7] and who therefore had an increased susceptibility of producing future affected offspring.[8]

It is remarkable that the increased incidence of ASB after abortion was confined to spina bifida and never shown for anencephaly. It could be argued that there are factors responsible for interfering with normal closure of the spinal neural tube that differ from those affecting normal development of the cranial vault. No study, however, has disclosed any aetiological factor that influences the incidences of anencephaly and spina bifida in a different manner. Furthermore, the risk of producing a future offspring with ASB is increased irrespective of whether the previous child had spina bifida or anencephaly.[8 9] In pursuing causal factors relating to ASB, I found that despite the well-known relation between ASB and social class there was no difference in the incidence of abortion between the respective social classes. The finding of a higher incidence of ASB in younger and primiparous patients, who had the lowest rate of previous abortions, was also converse to what might be expected.

My results provide strong circumstantial evidence against residual trophoblastic tissue being the cause of ASB in subsequent offspring and support the view that previous abortions are sometimes the manifestation of a tendency towards ASB rather than a primary cause.

TABLE I—*Incidence of neural-tube defects/1000 births according to past history of abortion. (Numbers of infants with ASB given in parentheses)*

	No of abortions		
	0	1	⩾2
Anencephaly	2·1 (116)	1·8 (18)	2·0 (6)
Spina bifida	2·3 (132)	3·1 (30)	6·4 (19)
All neural-tube defects ..	4·4 (248)	4·9 (48)	8·4 (25)
Total births	56 235	9745	2972

TABLE II—*Incidence of neural-tube defects in primiparous women/1000 births according to immediate history of abortion. (Numbers of infants with ASB given in parentheses)*

	No of abortions		
	0	1	⩾2
Anencephaly	2·2 (53)	2·7 (7)	2·2 (1)
Spina bifida	2·7 (67)	3·1 (8)	10·8 (5)
All neural-tube defects ..	4·9 (120)	5·8 (15)	13·0 (6)
Total births	24 402	2577	461

TABLE III—*Percentage incidences of neural-tube defects and prevalences of previous abortion according to social class. (Numbers of infants with ASB given in parentheses)*

Social class	Neural-tube defect		Prevalence of previous abortion	
	Anencephaly	Spina bifida	1	⩾2
I+II	0·13 (17)	0·16 (21)	14·5	4·2
III	0·16 (62)	0·29 (99)	14·3	4·4
IV+V	0·30 (44)	0·32 (47)	14·6	4·7

TABLE IV—*Percentage incidences of neural-tube defects and prevalences of previous abortion according to maternal age. (Numbers of infants with ASB given in parentheses)*

Maternal age	Neural-tube defect		Prevalence of previous abortion	
	Anencephaly	Spina bifida	1	⩾2
<20	0·26 (24)	0·30 (28)	5·9	0·6
20-24	0·25 (59)	0·26 (61)	14·0	3·7
25-29	0·17 (35)	0·27 (56)		
30-34	0·17 (17)	0·23 (23)	19·8	8·4
⩾35	0·12 (6)	0·28 (14)		

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